Parasites influence on host life history: Echinostoma revolutum parasitism of Lymnaea elodes snails

Abstract Using field surveys and experimental infections, we investigated the influence of a trematode parasite on life history traits of adult Lymnaea elodes snails. We found that parasitism significantly affected the growth, fecundity, and survival of host snails. Within five of the six natural L. elodes populations we sampled, shell length of echinostome-infected hosts was significantly greater than for uninfected conspecifics. Furthermore, we show that gigantism occurs among experimentally infected snails due to an accelerated growth rate and size-selective mortality following an Echinostoma revolutum infection. The fecundity of infected snails sharply decreased beginning at 3 weeks post exposure (PE) and all egg production eventually ceased for most hosts by 5–6 weeks PE. Energy constraints, imposed by parasite development, alter the host energy budget. Early in the infection, parasite depletion of host energy reserves reduces host reproduction, but sufficient resources remain to allow accelerated host growth. Mortality was increased among host snails at two distinct stages: shortly after exposure and several weeks after cercariae were first released. We did not observe tissue degradation in snails during the first 4 weeks after exposure to the parasite, but destruction of host tissues was noted among snails dying later in the infection.

Key words Host life history · Parasitism · Gigantism · Echinostoma · Lymnaea

Introduction

An organism’s differential investment into growth, reproduction, and survival defines its life history strategy. Allocation trade-offs exist between these processes such that energy channeled to any one of them is unavailable to the remaining two components (Stearns 1976, 1992). The lifetime reproductive success of an organism is a manifestation of the life history traits expressed by that organism throughout its life. Interspecific interactions, such as predation (Crowl and Covich 1990) and competition (Brown 1982), influence life history traits; likewise, parasitism shapes individual host life history strategies (Sousa 1983; Minchella 1985). Price (1980) elegantly emphasized the potential relationship between parasitism and host life history strategies, but researchers have just begun to elucidate this interaction during the last decade (Esch and Fernandez 1994; Michalakis and Hochberg 1994). By utilizing field collections and experimental trematode infections of Lymnaea elodes, a common freshwater snail, we assessed parasite influences on host growth, fecundity, and survival.

Parasites impose direct fitness costs on their hosts by stealing nutrients normally used for host life processes (Toft 1991). Trematodes are a diverse group of endoparasites requiring at least two hosts: a mollusc and a vertebrate. Intramolluscan trematode parasitism is frequently associated with the alteration of a host’s growth, fecundity, or survival; yet, few clear patterns exist (reviewed in Thompson 1997). The results vary depending upon several factors, including: (1) the specific host-parasite combination (Minchella and LoVerde 1983; Schrag and Rollinson 1994), (2) host age (Loker 1979; Kuris 1980; Keas and Esch 1997), (3) the life span and typical reproductive pattern of the host (Sousa 1983; Curtis 1995), and (4) whether the experiments were conducted in a laboratory or in the field (McClelland and Bourns 1969; Fernandez and Esch 1991). However, in the midst of these host response variations, there appears to be one common outcome among molluscs following trematode infection: an eventual elimination of host reproductive output, often termed “parasitic castration” (Wright 1971; Malek and Cheng 1974).

For uninfected freshwater snails, the production of energetically expensive egg masses occurs at the expense of body growth (Geraerts and Joosse 1984) and adult life
of reproduction (Eisenberg 1970; Brown et al. 1985); however, the life history traits of this species vary with population density, nutrient availability, and habitat permanence (Eisenberg 1966; Hunter 1975; Brown et al. 1985). Voluntism, growth rate, and size at maturity increase in permanent productive habitats, whereas, life span and reproductive rate are greatest in vernal, densely populated habitats. Because *L. elodes* is simultaneously hermaphroditic, it can reproduce through either self-insemination or outcrossing.

The trematode, *E. revolutum*, has a complex three-host life cycle and adults use avian species, primarily waterfowl, as definitive hosts (Kanev 1994). *E. revolutum* adults, occupying the posterior digestive tract of infected birds, produce eggs that give rise to a free-living stage, called a metacercid, that infects *L. elodes* snails in northern Indiana, USA (Sorensen et al. 1997). Metacercidia of *E. revolutum* that successfully infect *L. elodes* produce three distinct sexual stages of the parasite, a mother sporocyst and two subsequent redial stages. The second redial generation produces an infective, free-living cercarial stage that exits the host snail and forms metacercariae in another aquatic host, typically another snail (Beaver 1937). Metacercariae develop into adults when ingested by a suitable vertebrate host.

Field survey studies

Between 12 July and 9 August 1995, we collected *L. elodes* from six wetlands in northern Indiana to assess the prevalence of echinostome parasitism within various snail populations. Five of these sites – pond A, pond B, pond 39, Merry Lea Pond, and Garwood Pond – are located within Whitley County, Ind., whereas Shock Lake is in Kosciusko County, Ind. These six wetlands present a range of size, permanence, predominant vegetation, and nutrient productivity characteristics (Table 1). Two of these sites (pond A and pond B) have been previously studied (Brown et al. 1985; Minchella et al. 1985).

Snail life history traits following experimental infections

*E. revolutum* cercariae were obtained from the infected snails collected during the survey portion of this study in order to experimentally infect snails in the laboratory. Parasites were maintained in the laboratory by passage through chickens (*Gallus gallus* dom.) and their natural host snails (Sorensen et al. 1997).

We collected 500 *L. elodes* snails, ranging in size from 12 to 23 mm, on 17 June 1996 from Shock Lake. These snails were individually isolated under illumination twice (18–20 June and 2–3

### Materials and methods

#### Snail and trematode descriptions

The snail *L. elodes* (= *L. palustris* = *Stagnicola palustris* = *S. elodes*) is a common, freshwater pulmonate snail that inhabits a variety of ephemeral and permanent lentic systems. *L. elodes* is typically described as an annual species with a semelparous pattern of reproduction (Eisenberg 1970; Brown et al. 1985); however, the life history traits of this species vary with population density, nutrient availability, and habitat permanence (Eisenberg 1966; Hunter 1975; Brown et al. 1985). Voluntism, growth rate, and size at maturity increase in permanent productive habitats, whereas, life span and reproductive rate are greatest in vernal, densely populated habitats. Because *L. elodes* is simultaneously hermaphroditic, it can reproduce through either self-insemination or outcrossing.

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### Table 1 Environmental characteristics of the six sites where echinostome-infected and uninfected *Lymnaea elodes* snails were collected during 1995 in northern Indiana, USA

<table>
<thead>
<tr>
<th>Pond</th>
<th>Hectares</th>
<th>Permanence</th>
<th>Vegetation type</th>
<th>Productivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pond B</td>
<td>&lt;0.1</td>
<td>Temporary</td>
<td>Grasses</td>
<td>Low</td>
</tr>
<tr>
<td>Merry Lea</td>
<td>&lt;0.1</td>
<td>Semipermannt</td>
<td>Deciduous</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Pond 39</td>
<td>0.4</td>
<td>Temporary</td>
<td>Deciduous</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Garwood</td>
<td>0.4</td>
<td>Semipermannt</td>
<td>Deciduous</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Pond A</td>
<td>1.6</td>
<td>Temporary</td>
<td>Deciduous</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Shock Lake</td>
<td>2.0</td>
<td>Permanent</td>
<td>Grasses</td>
<td>High</td>
</tr>
</tbody>
</table>
July) to identify snails that were releasing trematode cercariae. Infected snails were removed from the study population and uninfected snails were housed in several aquariums until 3 July 1996. The overall prevalence of patent trematode infections among these snails was 5%; three trematode species were detected: *E. revolutum* (48%), an unidentified trematode possessing amphistome cercariae (32%), and an unidentified sporoclad species (20%). No multiple infections were observed among these snails.

On 3 July 1996, 118 of the uninfected Shock Lake *L. elodes*, ranging in size from 17.52 to 22.63 mm, were each isolated in 400-ml glass jars to determine their weekly egg production. All snails were fed green leaf lettuce ad libitum throughout the experiment and their water was replaced every 7 days with aged well water. On 11 July 1996, snails were separated into two treatment groups: control snails (n = 26, $\bar{x}_{\text{size}} \pm \text{SE} = 19.47 \pm 0.87$, $\bar{x}_{\text{fecundity}} \pm \text{SE} = 92.38 \pm 59.74$), and *E. revolutum*-exposed snails (n = 92, $\bar{x}_{\text{size}} \pm \text{SE} = 19.47 \pm 0.07$, $\bar{x}_{\text{fecundity}} \pm \text{SE} = 96.43 \pm 40.70$). All snails were placed individually into 10-ml vials containing 9 ml of aged well water. For the snails in the exposed treatments, we added ten miracidia to the vial. Control snails received no additional treatment beyond the isolation into vials. All snails were left in vials for 4 h and then returned to their 400-ml jar.

We recorded the shell length, fecundity, and mortality of all snails for 15 weeks. Snail size was measured once each week. Egg masses were removed from the snail jars twice a week throughout the study period and the number of eggs in each mass was recorded.

We isolated all snails beginning at 3 weeks post exposure (PE) and twice each week thereafter to ensure that no prepatent infections were included in the study populations. The 49 days between the collection date of these snails and the 3-week PE shedding date provided ample time to ensure that the remaining snails contained no prepatent infections, because the trematodes shed on the ovotestis/digestive gland (ODG) region, the site where *E. revolutum* larvae develop.

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We used parametric tests provided the assumptions of normality and homoscedasticity were met, otherwise we employed non-parametric procedures. An $\alpha$ of 0.05 was used to evaluate statistical significance, except when sequential tests were performed, in which case a “table-wide” a-value was calculated (Rice 1989). Because previous studies have shown that life history characteristics vary from pond to pond for *L. elodes* and because size heteroscedasticity existed at several of the sites across the infected and uninfected snails, we utilized the appropriate t-tests (either assuming equal variances or unequal variances) to assess differences in mean size for field-collected snails in the two infection classes for each of the six collection sites rather than a two-way ANOVA. However, factorial ANOVA tests were used to test hypotheses involving differences at a single point in time among the experimentally infected snails. When multiple pairwise comparisons were involved in the ANOVA, the Bonferroni/Dunn post hoc procedure was used to evaluate differences between means.

We employed repeated-measures profile analysis, MANOVAR (Potvin et al. 1990) when sequential data were collected on the same individuals over time (von Ende 1993).

We employed non-parametric tests (Mann-Whitney *U* and Kruskal-Wallis) on snail size data after 3 weeks PE and on fecundity data after 5 weeks PE because those data violated parametric assumptions after that time. When Kruskal-Wallis tests showed significant differences, we conducted multiple pairwise non-parametric comparisons (Zar 1984). We tested for associations between exposure group and infection status or mortality status with G-tests.

### Results

#### Field survey studies

Based on the release of echinostome cercariae during isolation, 267 (32.4%) of the snails collected from the six populations possessed patent echinostome infections. The prevalence of echinostome infections ranged from 57.6% at Merry Lea Pond to 17.1% at Shock Lake (Table 2). The prevalence of echinostome infections was related to the predominant vegetation type with a greater proportion of infected snails being found in ponds with deciduous vegetation dispersed throughout the littoral zones when compared to sites inhabited primarily by grass species, such as *Typhus* ($G = 12.86, P < 0.001$).

The mean size of echinostome-infected snails was significantly larger than that of uninfected *L. elodes* at five of the six wetlands (Table 2). Infected and uninfected groups of snails from Shock Lake had equal mean sizes, and these snails were more than 5 mm smaller than the average size of snails at the other study sites (Table 2). When we compared the size distributions for infected and uninfected snails from Garwood Pond and Shock Lake, we found that all the infected snails at Garwood Pond were larger than the mean size for uninfected snails at that site, whereas infected snails from Shock Lake were distributed across the range of sizes for uninfected snails (Fig. 1).

<table>
<thead>
<tr>
<th>Pond</th>
<th>Date</th>
<th>Mean size snail (mm)</th>
<th>t</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Infected</td>
<td>Uninfected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pond A</td>
<td>July 12</td>
<td>24.27 (170)</td>
<td>20.87 (312)</td>
<td>9.72</td>
<td>169</td>
</tr>
<tr>
<td>Pond B</td>
<td>July 12</td>
<td>28.23 (8)</td>
<td>24.03 (30)</td>
<td>3.58</td>
<td>36</td>
</tr>
<tr>
<td>Merry Lea</td>
<td>July 13</td>
<td>29.15 (19)</td>
<td>25.81 (14)</td>
<td>3.27</td>
<td>13</td>
</tr>
<tr>
<td>Pond 39</td>
<td>July 14</td>
<td>21.23 (50)</td>
<td>18.67 (128)</td>
<td>4.25</td>
<td>49</td>
</tr>
<tr>
<td>Garwood</td>
<td>August 9</td>
<td>22.21 (8)</td>
<td>19.17 (16)</td>
<td>2.49</td>
<td>21</td>
</tr>
<tr>
<td>Shock Lake</td>
<td>August 9</td>
<td>14.37 (12)</td>
<td>14.38 (58)</td>
<td>0.01</td>
<td>68</td>
</tr>
</tbody>
</table>
Snail life history traits following experimental infections

Snail infection and mortality

We first detected *E. revolutum* larvae at 29 days PE when two necropsied *L. elodes* snails contained rediae. Echinostome cercariae were first observed during snail isolations at 32 days PE. Based on the eventual release of cercariae or the presence of rediae, it was determined that 56.5% (52) of the EXP snails became infected with *E. revolutum*. There was no relationship between initial snail size and infection status.

*E. revolutum* miracidia increased mortality of EXP *L. elodes* relative to control snails during the early stages of infection ($G = 6.2, P = 0.013$). Between weeks 1 and 4 PE, 14.1% of the EXP and 7.7% of the CTL snails died. All of the CTL snails that died did so during the first week PE, whereas the EXP snails died throughout the 4-week period PE.

*E. revolutum* infection also decreased host survival after 4 weeks PE (Fig. 2). INF snails showed significantly higher mortality than uninfected snails (both EBU and CTL combined) at 6, 7, and 8 weeks PE ($G = 5.3, P = 0.02$; $G = 14.3, P < 0.001$; $G = 18.4, P < 0.001$, respectively). The EBU *L. elodes* survived at rates comparable to CTL snails (Fig. 2). No INF snails survived beyond 10 weeks (Fig. 2); furthermore, the proportion of INF snails that survived through the 8th week PE was significantly smaller than the proportion of surviving EBU and CTL snails ($G = 16.7, P < 0.001$).

Necropsies of infected snails and cercariae release patterns showed common trends over time. After 29 days, we found few rediae (<30) in the ODG and few cercariae were released over a 2-h period (<50). During later weeks, the number of rediae and cercariae that were observed increased by one to two orders of magnitude. The growth of the rediae population had a strong negative influence on the ODG as rediae apparently consumed ODG tissue, causing its size and structural integrity to decrease over time. The ODG of most hosts that died after 7 weeks PE was virtually destroyed. With the increasing number of cercariae being produced by these rediae, there was a corresponding increase in the number of metacercariae present in their hosts. Because snails were individually isolated, cercariae released from the host reentered the snail’s heart and kidney tissues producing a superinfection.

Snail fecundity

An *E. revolutum* infection had a strong effect on *L. elodes* fecundity; egg production decreased sharply beginning at 3 weeks PE (Fig. 3). INF snails had significantly lower weekly egg output compared to the two uninfected snail classes (EBU and CTL) at 5 weeks PE ($F_{2,92} = 23.71, P < 0.001$; infected $\bar{x} = 31.97$, uninfected $\bar{x} = 234.25$, control $\bar{x} = 146.59$). Mean fecundity values for EBU and CTL snails did not differ during that time period. The reduction in egg production among parasitized snails continued, and fewer hosts laid eggs over time until eventually all egg production ceased after 7 weeks PE (Fig. 3).

Among snails in the two uninfected classes (EBU and CTL), fecundity was variable beyond 3 weeks PE, but there was a general negative trend as time increased.
Most of these snails continued to lay eggs throughout the experiment. For instance, 13 of the 18 EBU and CTL snails that survived until week 15 PE were still producing eggs at that time.

Snail growth

The average shell length of snails in the three groups (INF, EBU, and CTL) increased over time through 3 weeks PE as shown by the Wilks' lambda test associated with a MANOVAR phase analysis of a time effect ($F_{3,99} = 86.70$, $P < 0.0001$). Furthermore, differences in growth rate were not observed between the snails in the three experimental groups because no time * experimental group effect was noted during this 3-week period ($F_{6,198} = 0.31$, $P = 0.93$). After 3 weeks PE, however, the mean size for INF snails increased rapidly compared to either EBU and CTL snails (Fig. 4). At 6 weeks PE, a significant size difference between INF, EBU, and CTL snails was noted ($H = 8.9$, $P < 0.001$). Multiple pairwise comparisons showed that INF snails were significantly larger than either EBU ($Q = 2.7$, $P < 0.05$) and CTL snails ($Q = 3.2$, $P < 0.005$). This difference in mean shell length among the three groups remained throughout the 10 weeks PE that INF snails survived (Fig. 4). INF snails were nearly 1 mm larger, on average, than snails in the EBU and CTL classes at 5 weeks, and >2 mm larger at 10 weeks PE.

Altered growth patterns were also observed among EBU snails beyond 5 weeks PE. Mean shell length of EBU snails was always greater than that of CTL snails (Fig. 4). At week 9 PE, EBU snails were significantly larger than CTL snails (time * treatment, $F_{1,9} = 2.674$, $P = 0.005$). After 9 weeks PE, the EBU snails were >1 mm larger than CTL snails, on average, and this difference increased slightly by week 15 (Fig. 4).

Either divergent shell growth rates or size-specific mortality could have yielded mean size differences among snails in the treatment groups. To test for growth rate differences across treatments, the size-dependent weekly growth increment was calculated for weeks 3 through 8 PE. We found significant differences in weekly growth rate for all weeks except week 5 PE (for weeks 3, 4, 6–8, $H > 9.0$, $F < 0.04$; for week 5, $H = 4.7$, $P = 0.09$). Subsequent multiple comparisons showed that INF snails increased their shell length to a greater extent than either the EBU or CTL snails during weeks 3 and 4 PE ($Q = 3.3$, $P < 0.005$ and $Q = 3.3$, $P < 0.05$, respectively). Beyond this time, EBU snails added shell material more quickly than the INF snails ($Q = 3.3$, $P < 0.005$; $Q = 2.9$, $P < 0.05$; $Q = 3.5$, $P < 0.005$, for weeks 6, 7, and 8, respectively); thus, the increase in mean size of INF snails relative to EBU or CTL snails beyond week 5 PE was caused by size-specific mortality, not by an increased growth rate. Larger INF snails survived longer than smaller INF snails in weeks 5 and 7 PE ($U = 25.0$, $P = 0.014$ and $U = 132.0$, $P = 0.015$, respectively). This effect was not found in the EBU and CTL snails.

Discussion

Parasitism by *E. revolutum* has a significant influence on *L. elodes* growth, fecundity, and survival. We found that snails exposed to *E. revolutum* suffered increased mortality compared to control snails. Furthermore, infected
snails showed size-specific mortality favoring larger individuals, grew faster early in the infection, and displayed reduced fecundity relative to uninfected snails.

Snail infection and mortality

Both exposure to and infection by *E. revolutum* markedly reduced *L. elodes* survival as also demonstrated in several other freshwater trematode-snail systems (Anderson and May 1979; Loker 1979; Kuris 1980; Minchella and LoVerde, 1981). We detected two phases of host mortality: during the prepatent stage (weeks 0–4), and several weeks postpatency (Fig. 2). Host mortality during the prepatent stage may have resulted from increased energetic demands of host defense, or from starvation, because tissue degradation was not obvious. In contrast, mortality of older snails involved tissue degradation. Superinfection with metacercariae is another potential cause of reduced survival in host snails during the late stages of infection; however, little is known about the relationship between metacercariae burden and mortality (Beaver 1937). The function of host immune responses was not investigated here, but initiation of these responses must be energetically demanding and the cost may depend upon the number of miracidia or metacercariae that enter the snail (Bayne and Loker 1987).

Snail fecundity

Trematode parasitism typically decreases the fecundity of hosts during the patent stages of an infection. Our results demonstrate that “parasitic castration” occurs in this parasite-host system (Fig. 3). The precise cause of this reduction is unknown, but it seems unlikely that physical destruction of host tissues is the sole mechanism (Wilson and Denison 1980), since few (<30) rediae were found in necropsied snails that died early in patency. Furthermore, the digestive gland and ovotestis of snails displayed no evidence of damage until later in the patency (5+ weeks). These results parallel those of Cheng et al. (1973), who concluded that chemical factors rather than physical damage were the cause of “parasitic castration” in *Ilyanassa obsoleta* snails infected with the sporocyst trematode, *Zoogonus rubellus*.

During the early stages of an infection (0–4 weeks), before reproductive tissues become damaged, it is possible that snail fecundity is reduced in this host-parasite system simply because of a reduction in the amount of energy accessible to the snail given the energetic demands associated with parasitism. Rollo and Hawryluk (1988) showed that uninfected *L. elodes* snails fed a low-quality diet spent more time foraging and reduced the amount of energy allocated to reproduction relative to snails on a high-quality control diet. It has been shown that schistosome infections reduce the glucose and amino acid levels of host snails in a manner similar to starvation (Christie et al. 1974; Stanislawski et al. 1979; Trede and Becker 1982); therefore, if echinostome infections deplete host resources, the reduction in nutrient availability may provide the impetus for altered fecundity patterns among infected *L. elodes*.

Minchella and LoVerde (1981) documented a process whereby schistosome-infected snails compensate for future reproductive losses due to parasitism by increasing their fecundity during the prepatent period of an infection. More recently, this “fecundity compensation” mechanism has been reported in two other schistosome systems involving distantly related planorbid and lymnaeid snail hosts (Thorin et al. 1986; Schallig et al. 1991). We found no evidence of “fecundity compensation” among *E. revolutum*-infected *L. elodes* snails; the mean number of eggs laid per week by infected snails did not exceed that of uninfected or control snails during the prepatent period. Although few detailed studies of other trematode systems are available for comparison, it appears that “fecundity compensation” may be limited to trematode systems that utilize sporocysts as the pre-dominant intramolluscan stage. Thus, schistosomes may mimic the conditions of Rollo and Hawryluk’s (1988) intermediate-quality diet which yielded a temporary increase in snail reproduction relative to well-fed individuals. Energy usage by *E. revolutum* rediae may not leave sufficient host resources to elicit a “fecundity compensation” response. Therefore, energy depletion/allocation processes could explain the apparent incongruity between pre-castration fecundity among schistosome- and echinostome-infected snails.

Snail growth

Brown et al. (1988) demonstrated a positive relationship between the prevalence of trematode infections and snail size among three populations of *L. elodes* in Indiana. The same relationship holds at five of the six sites we surveyed, as the shell length of echinostome-infected *L. elodes* was significantly greater than that of uninfected *L. elodes* (Table 2, Fig. 1). Baudoir (1975) presented six alternative hypotheses to explain the positive correlation between host size and the prevalence of infection. These hypotheses involve three basic mechanisms: increased host growth rates, differential host mortality rates, and size-specific infection preferences of parasites. Our data suggest that no single hypothesis accounts for gigantism in this system. The significant increase in adult host size that occurred as a result of the experimental infections (Fig. 4) supports the growth rate hypothesis during early stages of a trematode infection and the size-specific mortality hypothesis later in the infection. It remains to be determined whether or not age-specific infection rates are involved in this system, as shown by Curtis (1995) with trematode-infected *I. obsoleta*.

Sousa (1983) predicted that among short-lived, semelparous mollusc species, gigantism will occur following trematode parasitism because excess host energy
reserves are made available by "parasitic castration." This prediction requires that the energetic demands of parasitism are less than the costs of reproduction in the absence of parasitism (Bourns 1974). Data presented here support Sousa (1983) and Bourns (1974) by showing that L. elodes growth rates increase concurrently with depressed fecundity (Figs. 3, 4). This outcome suggests that host growth increases early in an infection because larval parasite development extracts sufficient energy to reduce host fecundity, but not so much that growth is prevented. In that sense, increased growth following an E. revolutum infection can be considered a manifestation of the trade-off between L. elodes fecundity and growth.

Three evolutionary hypotheses have been proposed as ultimate causes of gigantism among sterilized adult hosts: a parasite advantage, a host advantage, or a consequence of the life history trade-off between host growth and fecundity (Dawkins 1982; Minchella 1985; Minchella et al. 1985). In many cases, these alternative hypotheses cannot be clearly distinguished by considering only the proximate outcome of parasite-host interactions due to the inextricable link between host fecundity and growth. Also, it may be difficult to unambiguously assign fitness costs and benefits experienced by hosts or parasites following a change in the host's life history. We feel that the host advantage hypothesis is supported by the elevated growth of our exposed but uninfected snails relative to control snails because exposure to miracidia in the absence of an infection elicits enhanced growth rates among exposed snails.

Growth is presumably the predominant energy demand for juvenile gastropods; as a result, infected immature snails should grow more slowly than uninfected conspecifics (Sousa 1983). This prediction has been supported in several studies of infected lymnaeid snails (Zischke 1967; Loker 1979). For instance, Zischke (1967) demonstrated that uninfected, juvenile L. elodes grow more rapidly than individuals harboring echinostome infections. On the other hand, our results indicate that an increase in shell length occurs in adult snails following infection. Therefore, within natural L. elodes populations, it is likely that infected, immature L. elodes grow slower, on average, than uninfected conspecifics, while parasitized adult snails grow faster than uninfected snails.

It seems incongruous that gigantism was not observed among naturally infected Shock Lake snails, but it was observed among the experimentally infected snails which originated from Shock Lake. The age-specific parasite influence on L. elodes growth rates helps explain the lack of gigantism among infected snails collected during the 1995 survey at Shock Lake. This site is a productive, permanent wetland that provides L. elodes with the conditions to allow a cohort to reach reproductive maturity during their first year; thus, when sampled, this population contained both mature (~15 mm) and immature members of the 1995 cohort. It could be that snails infected prior to maturity suffered reduced growth rates, whereas snails infected as adults experienced increased growth rates. Thus, under these environmental conditions, a bimodal distribution of host size would be expected, and the mean values for these two size distributions would bracket the mean size of uninfected snails. The net effect of these two age-specific processes would be a similarity in mean size for infected and uninfected snails, as we found. This process requires that the infected L. elodes were parasitized during the summer we collected them. This appears likely based on the reduced survival shown by our experimentally infected snails and by prevalence surveys for an ecologically similar freshwater pulmonate, Helisoma trivolvis, that show echinostome parasitism peaking in late summer and waning throughout the winter (Schmidt and Fried 1997).

Evidence of age-specific growth rates was not found at our other collection sites because their ephemerality and limited productivity prevents new cohorts from reaching adult size by the time the survey collections were made. In other words, the snails collected at the sites where gigantism was detected were members of the previous year's cohort rather than snails produced during 1995. Presumably, these hosts became infected following maturity which led to increased growth following "parasitic castration" and the effects of size-specific mortality. This outcome demonstrates a potential bias that can result when considering the impact of trematode infections on host growth in natural populations (Fernandez and Esch 1991). In nature, the variability of host growth rates may increase markedly following parasitism if the growth rates of mature and immature individuals are affected differently.

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References


Baudoin M (1975) Host castration as a parasitic strategy. Evolution 29: 335-352


Beaver PC (1937) Experimental studies on Echinostoma revolutum (Froelich): a fluke from birds and mammals. Ill Biol Monogr 15: 1–96


Minchella DJ, LoVerde PT (1983) Laboratory comparison of the relative success of Biomphalaria glabrata stocks which are susceptible and insensitive to infection with Schistosoma mansoni. Parasitology 86: 335–344
Stanislawski E, Becker W, Muller G (1979) Alterations of the free amino acid content in the hemolymph of Biomphalaria glabrata (Pulmonata) in starvation and after infection with Schistosoma mansoni (Trematoda). Comp Biochem Physiol 63B: 477–482
Thorhill JA, Jones JT, Kusel JR (1986) Increased oviposition and growth in immature Biomphalaria glabrata after exposure to Schistosoma mansoni. Parasitology 93: 443–450