LIFE HISTORY COST OF TREMATODE INFECTION IN HELISOMA ANCEPS USING MARK–RECAPTURE IN CHARLIE’S POND

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ABSTRACT: Parasitism has the potential to affect key life history traits of an infected host. Perhaps the most studied interactions are in snail–trematode systems, where infection can result in altered growth rates, survival, and/or fecundity of the individual. Positive correlations between host size and parasite prevalence are often attributed to changes in growth rates or mortality, which have been observed in the laboratory. Extending lab-based conclusions to the natural setting is problematic, especially when environmental conditions differ between the laboratory and the field. The present study uses reproduction experiments and mark–recapture methods to directly measure key life history traits of the pulmonate snail Helisoma anceps in Charlie’s Pond. Based on previous laboratory and field experiments on H. anceps, we predict a significant reduction in fecundity, but not growth rate or survival, of infected snails. Individual capture histories were analyzed with multistate models to obtain estimates of survival and infection probabilities throughout the year. Recaptured individuals were used to calculate specific growth rates. Trematode infection resulted in complete castration of the host. However, neither survival nor growth rates were found to differ between infected and uninfected individuals. The probability of infection exhibited seasonal variation, but it did not vary with size of the snail. These results suggest that the correlation between host size and trematode prevalence is not due to differential mortality or changes in growth rates. Instead, the infection accumulates in large snails via the growth of smaller, infected individuals.

Trematode larvae commonly castrate their molluscan hosts. The mechanism of castration depends on the specific larval stage infecting the host. For example, rediae mechanically castrate the host by consuming host tissue, including the high-energy fat of the gonads. Sporocysts, lacking a mouth and pharynx, likely castrate the host via chemical inhibition. In particular, Trichobilharzia ocellata stimulates the release of schistosomin, which inhibits oviposition by Lymnaea stagnalis (De Jong-Brink, 1995). Castration could be complete, as in the Helisoma anceps–Halipegus occidualis system (Crews and Esch, 1986), or partial, as in the mudsnail Hydrobia ventrosa (Kube et al., 2006). The individual cost of castration includes the loss of future reproduction. Fecundity compensation has been proposed as a mechanism by which the host could potentially offset this negative effect (Minchella, 1985; Sorensen and Minchella, 1998). Compensation has been recorded in the T. ocel- lata–L. stagnalis, Schistosoma mansoni–Biomphalaria glabrata, and S. mansoni–Biomphalaria pfeifferi systems (Sorensen and Minchella, 2001), and it occurs by increasing reproductive effort, i.e., oviposition or maturation, before castration.

Many researchers have noted a positive correlation between prevalence of infection and size of the snail host. This has led some to question whether growth schedules vary between infected and uninfected hosts (Baudoin, 1975; Fernandez and Esch, 1991a; Sorensen and Minchella, 2001). Common responses to infection include gigantism (the rapid increase in size after infection) and stunting. The growth response by a snail may depend on when it was infected. For example, if infected before reproductive maturity, B. glabrata experiences rapid growth until maturity, and then it grows less rapidly than uninfected individuals (Gerard and Theron, 1997).

Recently, trematode infections have been shown to increase mortality of the snail host. Negative effects on survival have been observed from infections by both sporocysts (Makanga, 1981; Minchella and Loverde, 1981; Zakikhani and Rau, 1999) and rediae (Dreyfuss et al., 1999; Sandland and Minchella, 2003). Physical damage caused by the parasite during feeding and cercariae release, and the energetic cost of an immune response may contribute to, if not cause, the increase in mortality. Reinfection by cercariae has also been implicated in decreasing survival of the snail host (Kuris and Warren, 1980; Sandland and Minchella, 2003).

The difficulties in estimating the true cost of parasitism arise when extending the results derived from laboratory experiments to observations in the field. For example, lab-reared Lymnaea elo- des grew faster and laid more eggs than snails raised in the field (Eisenberg, 1966, 1970). If food was supplied in the field, then both growth and fecundity increased. Likewise, H. anceps in Charlie’s Pond are protein deprived, and they exhibit lower fecundity and growth rates than snails raised in the laboratory (Keas and Esch, 1997). To accurately assess the impact of parasitism, studies should be performed under natural field conditions.

Mark–recapture sampling protocols provide the data required to estimate survival in the field. Moreover, recent advances have expanded the scope of analysis, such that simple estimates of survival and population size are becoming secondary to more complex questions. One advance is the development of multistate models, which permit the estimation of transition probabilities between states (Hestbeck et al., 1991; Lebreton et al., 1992; Brownie et al., 1993; Nichols et al., 1994; Nichols and Kaiser, 1999; Lebreton and Pradel, 2002; White et al., 2006). For example, many researchers have used multistate models to estimate the cost of breeding on survival rates (White et al., 2006), whereas others have calculated dispersal rates between various habitats (Hestbeck et al., 1991). Application of mark–recapture protocols to snail populations has been limited to a few studies (O’Keeffe, 1985; Woolhouse, 1988; Goater et al., 1989; Woolhouse and Chandiwana, 1990; Fernandez and Esch, 1991a; Chlyeh et al., 2002, 2003). One reason for the lack of interest may be the low recapture rate of aquatic snails, as a consequence of their vagility. In Charlie’s Pond, 97% of H.
Helisoma anceps were collected from 2 sites in Charlie’s Pond, Stokes County, North Carolina. Charlie’s Pond is approximately 1 ha, and it has been described previously (Crews and Esch, 1986; Esch et al., 1997). Beginning in May 2005, snails were collected by random sampling with a dip net for 1 hr each week. Weekly collecting sites alternated between the northwest cove (NW) and east side (ES) of Charlie’s Pond. No collections were made from mid-November 2005 through February 2006. The collection regime resumed in March 2006 and concluded in October 2006.

Snails were transported to the laboratory where they were individually isolated in 55-mm plastic jars. The shell length of each snail was measured to the nearest 0.05 mm, and the jars were examined for presence of cercariae. Each snail was marked with colored enamel paint to signify month, with a letter indicating year and site, and a number. The combination of color, letter, and number represented a unique identifier that span the entire collecting period. The capture histories consisted of a series of 0, A, and B. Each value holds unique information. Differences in mean size of uninfected and infected individuals were tested with anova. A new set of capture histories was created for each iteration. One thousand iterations were performed for each size class, and the model deviances were saved. The deviance from the general model was divided by the mean deviance from the simulated models to obtain \( \hat{\epsilon} \) (Cooch and White, 2006). AIC was divided by \( \hat{\epsilon} \) to obtain a quasi-likelihood adjustment AIC (QAIC), which corrects for excess variation in the models (Cooch and White, 2006). Models with small QAIC are preferred to those with large QAIC. More specifically, differences in QAIC of >7 strongly suggest a real difference between the models being compared (Anderson and Burnham, 1999a, 1999b).

The POPAN algorithm of Program MARK was used to estimate recruitment (births + immigration) and population size. This method models a super population \( N \) that enters the local population with a probability of \( b \). The product of the probability \( b \) and the super population \( N \) equals the number of recruits. Recruitment or births during monthly transition \( i \) (\( B = N \cdot b \)). Population size \( \hat{N} \) during month \( i \) is derived from estimates of survival, population size, and recruitment in the previous month (Cooch and White, 2006).

RESULTS

In total, 910 \( H. \) anceps were placed in the reproduction platforms. Because fecundity varies from year to year (Negojetich and Esch, 2007), the effect of infection status was assessed by comparing fecundity of only those snails that were isolated in 2006. The analysis included 674 snails, 34 were infected with \( H. \) occidualis (rediae), and 27 with miscellaneous trematode species (mostly sporocysts). Infection resulted in a significant reduction in egg output (KW: \( \chi^2 = 48.3, 2 \text{ df}, P < 0.0001 \)). Uninfected snails laid an average of 52.6 ± 0.57 eggs/wk compared with 56.0 ± 0.32 and 74.0 ± 0.44 eggs/wk for \( H. \) occidualis-infected and other infected snails, respectively.

Restricting analysis to uninfected snails reveals that monthly mean fecundity decreased throughout the year for both locations within the pond (Fig. 1). Randomization tests detected a significant difference in March \( (P < 0.0001) \), May \( (P < 0.0001) \), June \( (P < 0.0003) \), and August \( (P < 0.0001) \). Analysis with POPAN strongly suggests variation in both site and time for the probability to enter the population (AIC = 7,175.8, model likelihood = 1.00, deviance = 7,036.1). For monthly transitions in 2006, recruitment was higher at NW than ES; yet, the

\[ \text{MATERIALS AND METHODS} \]

\[ \text{RESULTS} \]
FIGURE 1. Mean fecundity (±SE) of uninfected snails from ES and NW of Charlie’s Pond in 2006. Data points with an asterisk indicate site differences in fecundity, as determined by randomization tests.

The mark–recapture experiment resulted in a total of 4,850 individual capture histories. Of these, 1,114 capture histories represented individuals that were recaptured at least once (Table I). Only a single snail migrated across the pond, and this individual was excluded from analysis. Seasonally corrected mean sizes were significantly different between uninfected, *H. occidualis*-infected, and miscellaneous infected snails (KW: $\chi^2 = 153.1$, 2 df, $P < 0.0001$). Snails infected with *H. occidualis* ($\bar{x} = 9.72 \pm 0.05$ mm [±SE]) were larger than snails infected with other trematode species ($\bar{x} = 9.61 \pm 0.10$ mm), and these were larger than uninfected snails ($\bar{x} = 9.07 \pm 0.02$ mm; Steel-type MC: $P < 0.01$ for all comparisons).

FIGURE 2. Number of new recruits (±SE) estimated with POPAN in 2006 for ES and NW of Charlie’s Pond. Recruitment is measured as the increase in population size from 1 mo to the next after accounting for mortality, and entrance via birth and immigration.
Table I. Percentage of capture histories representing the number of times an individual was captured for ES, NW, and the entire Charlie’s pond. Snails captured once were marked and never recaptured. The total number of capture histories for each site and for the Charlie’s Pond is also included.

<table>
<thead>
<tr>
<th>Times captured</th>
<th>ES</th>
<th>NW</th>
<th>Pond</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>77.54</td>
<td>76.60</td>
<td>77.03</td>
</tr>
<tr>
<td>2</td>
<td>20.49</td>
<td>20.08</td>
<td>20.27</td>
</tr>
<tr>
<td>3</td>
<td>1.84</td>
<td>3.13</td>
<td>2.54</td>
</tr>
<tr>
<td>4</td>
<td>0.09</td>
<td>0.11</td>
<td>0.10</td>
</tr>
<tr>
<td>5</td>
<td>0.04</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>Total no. of capture histories</td>
<td>2,226</td>
<td>2,624</td>
<td>4,850</td>
</tr>
</tbody>
</table>

SGR did not differ by site or infection status for almost all size classes examined (Fig. 3). The single exception when comparing the sites was for the 7-mm size class. SGR for NW was significantly higher than ES (analysis of variance [ANOVA]: \( F_{1,10} = 6.9, P < 0.01 \)). Upon closer inspection, the difference in SGR persisted for uninfected (ANOVA: \( F_{1,10} = 7.9, P < 0.006 \)), but not infected (ANOVA: \( F_{1,10} = 0.0003, P > 0.05 \)), individuals. Seasonally, SGR fluctuated little from March to May, increased to a peak in August, and then decreased until winter (Fig. 4). Uninfected individuals exhibited a higher SGR in August (KW: \( \chi^2 = 20.0, 1 \text{ df}, P < 0.0001 \)). Comparing the sites, significant differences in SGR were observed in August (ANOVA: \( F_{1,134} = 6.1, P < 0.015 \)) and September (KW: \( \chi^2 = 19.2, 1 \text{ df}, P < 0.0001 \)). When examining infection status within these months, SGR was not different in August for both uninfected (ANOVA: \( F_{1,98} = 3.5, P > 0.05 \)) and infected individuals (ANOVA: \( F_{1,34} = 0.74, P > 0.05 \)). However, in September, NW growth rates were significantly higher only for the uninfected snails (KW: \( \chi^2 = 13.3, 1 \text{ df}, P < 0.0003 \)).

The effect of infection on survival was assessed by fitting various models to the set of capture histories from the mark-recapture experiment. To avoid complications of initial size and growth of the snails, analyses were performed by size class. For example, the capture histories of all snails that were 6–6.95 mm at initial capture were analyzed independently from the remaining size classes. After fitting the general model and correcting for overdispersion of the errors, the most likely model that fits the data was \( \Phi_i \rho_i \Psi_i \), (Table II). For the 10-mm size class, there is some support for a model that allows survival to vary with site and time; yet, the best model was 2.5 times more likely than this second best model. Survival from 1 mo to the next varied seasonally (Fig. 5). Small snails suffered from higher mortality (=low survival probabilities) than larger snails, except in the period during cohort turnover.

Recruitment of trematode infections is reflected in the probability of being infected (Fig. 6). Early in the year, infection probabilities remained under 0.2 for all size classes. Peak infection probabilities occurred from June to August, which corresponds to a large recruitment of trematode infections and new snails at this time. The risk of infection then returned to levels previously observed during spring.

**DISCUSSION**

When comparing fecundity, prominent differences exist between ES and NW. In both locations, fecundity decreases throughout the year, but NW snails lay more eggs/week until cohort turnover in July (Fig. 1). In March, the difference in reproductive output is most likely related to emergence of snails from the substratum as the water warms after winter. Specifically, more snails per hour were collected in NW than ES during March, which suggests that snails become active earlier in the year at NW than in ES. May was the second month that produced a difference in fecundity. In this month, snails >9 mm were more fecund, and closer inspection reveals that reproductive activity is responsible. The percentage of reproducitively active snails differed for the 9-mm (93% in NW vs. 0% in ES) and >10-mm size classes (96% in NW vs. 43% in ES). When accounting for reproductive activity, snails >10 mm remain more fecund at NW compared with ES. Reproduction is depressed when a snail approaches death (Herrmann and Harman, 1975). Based on size frequency histograms (Fig. 7), cohort turnover at ES is apparent by June, which suggests that those snails were near death in May. Moreover, the NW decrease of fecundity in July corresponded to turnover at that location, which occurred nearly a month after ES.

Alternative explanations for a difference in fecundity between the sites include local productivity and snail size. First, large snails have the capacity to lay more eggs than smaller individuals (Brown, 1985; Brown et al., 1985; Lazaridou-Dimitriadou et al., 1998). The NW snails placed in the reproduction platforms were larger than ES snails in May–July. However, fecundity at the 2 sites was similar in July, which diminishes the likelihood that snail size is the primary cause of the fecundity differences. Second, reproduction is depressed when food supplies are limiting (Brown et al., 1985; Byrne et al., 1989; Lam and Calow, 1989; Wayne, 2001). NW contains more emergent vegetation and leaf litter, surfaces on which periphyton can grow. If productivity is contributing to the differences in fecundity, then NW snails of a specific size class should be consistently more fecund than ES snails of the same size class during a particular month. This was only observed in March for the 8- and 10-mm size classes, and in May for the 9- and 10-mm size classes. The lack of consistency from 1 mo to the next implies that productivity is not the only factor affecting egg production in the Pond. Instead, it may be contributing to the observed differences between the 2 sites.

Infection severely decreased egg production in *H. aniceps*. For snails infected with rediae of *H. occidualis*, the mechanism is mechanical castration by direct consumption of the gonads (Crews and Esch, 1986). Of the 27 snails infected with a trematode species other than *H. occidualis*, 22 individuals were shedding 1 of 2 unknown spirorchiid cercariae. Within the snail host, the 2 spirorchiids have sporocyst larval stages. Unlike *H. occidualis*, sporocysts have been known to chemically castrate the host (Cheng et al., 1973; De Jong-Brink et al., 1991; Wayne, 2001). The size at first reproduction was 6 mm, and given that all but 1 snail were larger than 7 mm, it is reasonable to assume that the unknown spirorchiids also castrate *H. aniceps* in Charlie’s Pond. In fact, 19 of 22 spirorchiid-infected snails did not lay eggs, and the 3 snails that were reproductively active produced no more than 10 eggs within a week. In some snail–trematode systems, snails often exhibit fecundity compensation (Sorensen and Minchella, 2001). Specifically, some species increase their reproductive activity between the time of initial exposure to the egg or miracidium, and patency. Fecundity
compensation may be occurring in the Pond, but we feel that it is unlikely given that increases in egg production were not observed in lab studies on *H. anceps* (Keas and Esch, 1997).

Infected snails are larger than uninfected snails, but this does not imply a difference in growth rate. Much work has been done on the influence of trematode infections on the growth rate of snails. In all of the field studies at Charlie’s Pond, *H. occidualis*-infected *H. anceps* were larger than uninfected in-

**Figure 3.** Mean specific growth rate (±SE) for the different size classes of snails, examined by (A) infection status and (B) site (ES and NW). Pairs of bars marked with an asterisk were significantly different at the $P \leq 0.05$ level. Only 1 infected snail from the 5-mm size class was recaptured, and that value is excluded from the graph.
Figure 4. Mean specific growth rate (±SE) throughout the year, examined by (A) infection status and (B) site (ES and NW) of the uninfected snails. Pairs of bars marked with an asterisk indicate a significant difference at the $P \leq 0.05$ level.

Individuals (Crews and Esch, 1986; Fernandez and Esch, 1991c; Williams and Esch, 1991; Negovetich, 2003). Similar associations between prevalence and snail size occur in 68% of marine and 73% of freshwater gastropods (Sorensen and Minchella, 2001). Four possibilities can explain the positive correlation between host size and parasite prevalence, i.e., unequal catchability, differential mortality, variation in susceptibility, and changes in growth rates (Baudoin, 1975).

Catchability describes the likelihood of collecting snails given their infection status. In some systems, the parasite alters the
behavior of the host so that infected individuals are more likely to be consumed by the next host in the parasite’s life cycle (Poulin, 2007). In the context of Baudoin (1975), the parasite may have a size-dependent effect on behavior, such that large, infected individuals will be easier to catch than small, infected individuals. Thus, prevalence would be positively correlated with size of the snail. In Charlie’s Pond, no evidence exists that would suggest that infected snails are easier to catch than uninfected snails. Furthermore, analysis of capture histories indicates that models with capture probabilities that vary with infection status do not fit the data better than the general model (data not shown). Thus, catchability of H. anceps is not influenced by infection status.

There are 2 situations where differential mortality will produce a size difference between uninfected and infected individuals. The first is an increase in mortality of young snails after infection, which seems more common with rediae stages than with sporocysts (Sorensen and Minchella, 2001). If younger snails are more likely to die after infection, then the size distribution of infected snails will be skewed, with a greater quantity of infected snails tending to larger sizes. The second situation is a reduction of mortality of infected individuals. Specifically, if infected snails live longer than uninfected snails, and size is positively correlated with age, then the infected population will be larger, on average, than the uninfected population. In the current study, differential mortality is not apparent. None of the most parsimonious models included infection status as a determinant of survival (Table II). Prepatent infections could have increased mortality, especially in small snails. This increase in mortality would not be detected because the sampling methods only permitted the identification of patent infections. However, laboratory studies have also failed to reveal a
significant difference in survival (Keas and Esch, 1997); so, it seems that mortality of *H. anceps* in the field is not influenced by infection status.

A positive correlation between prevalence and snail size may have arisen from differences in susceptibility. Two situations would produce the same observation, i.e., equal probability of infection across size classes, or increases of infection probabilities with size (Baudoin, 1975). If the probability of infection is constant across size classes, then infections will accumulate in larger snails. For example, the 9-mm size class includes newly infected 9-mm snails, in addition to individuals that were infected at a smaller size and grew into the size class. In the absence of increased mortality, a plot of prevalence versus size class will produce a straight line with positive slope, where the slope is equal to the probability of infection. Alternatively, the probability of infection could increase with size, in which case prevalence will exhibit a curvilinear increase as a function of size class. The highest prevalence, regardless of trematode species, was observed in June (20%), and it remained above 16% in July and August. To maintain the level of prevalence during cohort turnover, the probability of infection must increase so that young, newly infected individuals replace the dying, in-

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**Figure 5.** Probability to survive ($\Phi$) during the monthly transitions for the 6 size classes of snails.
fected snails (Fig. 6). This corresponds with the timing of peak recruitment and maturation of *H. occidualis* in the green frogs (Goater, 1989; Wetzel and Esch, 1996). There is little evidence to suggest that infection probability varies with size class. With the exception of the July–August and August–September transitions, the 3 largest size classes differ by no more than 12%, and the SE of the estimates overlap, suggesting that infection probabilities are not significantly different. The SE of the 3 smallest size classes also overlap. Thus, the correlation between shell length and prevalence is probably caused by accumulation of infected individuals in the larger size classes.

The final explanation for the correlation between prevalence and size involves growth rates. Sorensen and Minchella (2001) reviewed the literature on snail–trematode life history interactions. They found that enhanced growth rates of infected snails occur more frequently in freshwater systems, whereas stunting is more common in marine systems. Trematode infections can have stage-specific effects on the host, such that infected juveniles might grow faster than uninfected juveniles, whereas infected adults may grow more slowly. Most work has occurred in the laboratory, including experiments on *H. anceps* (Keas and Esch, 1997). Fernandez and Esch (1991c) suggested that
the disparity of conclusions between the field and lab experiments may be due to the abundance of food and constant environmental conditions in the laboratory. Specifically, the cost of parasitic infection on growth rates could be strengthened or diminished depending on the environmental conditions.

Growth rates vary by size class and month. Specifically, small snails grow faster than large snails (Fig. 3), and SGR peaks in August (Fig. 4). The dependence of size on growth rate is well documented for many species, including *H. anceps* (Herrmann and Harman, 1975; Fernandez and Esch, 1991a). The 7-mm size class produced a significant difference of SGR between NW and ES. However, 71% of the uninfected NW snails were initially captured in August–September, the 2 mo with the fastest growth rate, compared with 31% of the ES snails. In contrast, 75% of the infected snails for each site were collected from July to September. Thus, the observed difference in SGR for the 7-mm size class was primarily influenced by seasonal changes in growth rate, and not by variation between the 2 locations within Charlie’s Pond. Similarly, the significant difference in September between the sites is most likely due to the population structure in that month. Snails 8 mm represented 20% of the ES population and 42% of the NW population. Seasonal variation and size class effects suggest that SGR does not differ by site. Therefore, site was ignored when examining the effect of infection on SGR.

August was the only month when SGR of uninfected and infected individuals were significantly different. The distribution of size classes within this month indicates that the infection effect in this case is spurious. Specifically, 59% of the uninfected snails were <8 mm, whereas only 36% of the infected individuals were represented by the fastest growing size classes. Furthermore, no difference in SGR was detected within the size classes examined. This is surprising given the number of host–parasite associations where the growth rate of the host is affected by the trematode (Sorensen and Minchella, 2001). Competing hypotheses explain alterations in growth schedules. The first states that the available energy for the host is contingent on the needs of the parasite (Sousa, 1983). In castrated individuals, the parasite liberates energy for the host that was previously allocated to reproduction. In response, the host exhibits faster growth rates. Alternatively, gigantism could result as a response by the host to increase fitness before castration (Minchella, 1985; Sorensen and Minchella, 1998). Specifically, once infected, the host attains reproductive size and oviposits before castration. Increased growth rates may also delay castration because large hosts maintain viable gonadal tissue longer than smaller snails. In both hypotheses, life history tradeoffs minimize the effect of infection. At Charlie’s Pond, trade-offs in the life history traits of *H. anceps* were not detected. Although the trematode infections castrated the snail host, neither growth rate nor survival probability was altered in comparison with uninfected individuals.

Previous studies have examined key life history traits of *H. anceps* from Charlie’s Pond in the field (Crews and Esch, 1986; Fernandez and Esch, 1991a; Negovetich, 2003) and in the lab (Keas and Esch, 1997). The current study expands on the previous research by comparing 2 locations within Charlie’s Pond and directly estimating life history traits with a mark–recapture experiment. A decrease in fecundity was the only individual cost associated with trematode infection. Castration will negatively impact the snail host by decreasing the population growth rate and reducing the number of genotypes that individual hosts pass on to the next generation. Although individual responses to offset castration were not detected, the population may have responded with changes in the life history strategy, such as a decrease in the age at first reproduction. This could be accomplished via increased growth rates so that reproductive size is reached earlier in life, or it could arise from decreases in size at maturity (Roff, 1992). In Charlie’s Pond, snails became reproductively mature at 7.5–8.0 mm in mid-1980s (Goater, 1989), but the latest fecundity experiment demonstrated oviposition by 6-mm snails in August and September. The decrease in size at maturity could be an evolutionary response in a system where castration by *H. occidualis* has been occurring in the snail population for nearly 25 yr. The life history estimates derived from the current study are being used to construct a

![Figure 7](image-url)
model of the *H. anceps* population in Charlie’s Pond. A matrix model will quantify the cost of castration at the population level by determining the population growth rate across a range of infection probabilities. Furthermore, the effect of adjustments to the growth rates, survival, and fecundity will be assessed. Thus, mathematical modeling may provide clues as to which life history traits are most likely to offset the cost of parasitic castration.

**LITERATURE CITED**


