Evolutionary Ecology Research, 2006, **8**: 753–763

Prevalence of parasites does not predict age at first reproduction or reproductive output in the freshwater snail, *Helisoma anceps*

Amy C. Krist*

Department of Biology, University of Wisconsin-Eau Claire, Eau Claire, WI 54701, USA

ABSTRACT

Question: Are reproductive output and age at first reproduction explained by the prevalence of castrating parasites in a population? Life-history theory predicts this relationship.

Data studied: Reproductive output over 21 days in field-caught freshwater snails from 12 lake populations in Wisconsin. I also measured age at first reproduction and reproductive output over 27 weeks in laboratory-reared snails from the same populations. I determined the prevalence of parasites in each of 2 years as the proportion of snails infected with digenean trematodes.

Search method: *Field-caught*: I regressed mean reproductive output on mean prevalence. *Laboratory-reared*: I regressed mean reproductive output and mean age at first reproduction on mean prevalence.

Conclusions: Neither measure of reproductive output, nor age at first reproduction, was predicted by the mean prevalence of trematode parasites.

Keywords: age at first reproduction, castrating parasites, freshwater snails, life-history theory, reproductive output, trematodes.

INTRODUCTION

Parasites have a much greater role in the lives of their hosts than just the immediate harm that they cause. For example, the interactions between parasites and hosts can modify the host's life-history traits through short-term physiological or evolutionary responses. Theory predicts similar adaptive modifications of host life-history traits on both physiological and evolutionary time scales. Virulent parasites should favour early reproduction (Hochberg *et al.*, 1992, Koella and Restif, 2001) and increased reproductive effort in infected individuals (Minchella, 1985; Forbes, 1993; Perrin *et al.*, 1996; Sorci *et al.*, 1996; Gandon *et al.*, 2002) under some conditions. Similarly, uninfected individuals from populations with a high incidence of castrating parasites should mature early (Kozlowski and Uchmanski, 1987; Kozlowski and Weigert, 1987) and increase reproductive effort under most conditions (Kozlowski and Uchmanski, 1987; Kozlowski and Weigert, 1987;

* Address all correspondence to Amy Krist, Department of Zoology and Physiology, Dept. 3166, University of Wyoming, 1000 E. University Avenue, Laramie, WY 82071, USA. e-mail: krist@uwyo.edu

Gandon *et al.*, 2002). Although the first two models address populations with high extrinsic mortality, parasitic castration and mortality have equivalent effects as agents of natural selection.

Empirical studies confirm these predictions. Parasitized hosts reproduce early (Thornhill *et al.*, 1986; Schallig *et al.*, 1991; Agnew *et al.*, 1999; Ebert *et al.*, 2004) and increase reproductive effort (Minchella and Loverde, 1981; Thornhill *et al.*, 1986; Schallig *et al.*, 1991; Christe *et al.*, 1996; Sorci *et al.*, 1996). This type of adaptive phenotypic plasticity should increase the host's fitness in the short term to partially compensate for the expected loss of future fitness from the parasite infection (Minchella, 1985; Forbes, 1993). Similarly, uninfected individuals mature earlier (Lafferty, 1993; Jokela and Lively, 1995) and have higher reproductive output (Krist, 2001) in populations where parasite prevalence is high. These traits are the products of long-term interactions between hosts and castrating parasites favouring hosts that mature early and exhibit higher investment in reproduction. Reciprocal transplant experiments by Lafferty (1993) revealed a genetic basis to early maturity, but the mechanism of response in the other two studies was not determined. In this study, I examined life-history traits in both field-caught and laboratory-reared freshwater snails to determine whether the mechanism of response is plastic or genetically canalized.

Specifically, I examined the predictions from life-history theory by comparing reproductive output and age at first reproduction among populations of the freshwater snail, *Helisoma anceps*. Populations differed in parasite prevalence, the proportion of hosts infected by one or more castrating trematodes (Bush *et al.*, 1997). Prevalence of these parasites is an estimate of the selection pressure applied by trematodes within populations because castration causes reproductive 'mortality' (Lafferty, 1993). Assuming parasite prevalence reflects long-term levels of interaction between snails and parasites, selection pressure by parasites will favour snails with a high investment in reproduction and early maturity. This selection should lead to uninfected individuals from populations with a high prevalence of parasites exhibiting higher reproductive output and earlier maturity than uninfected individuals from populations with a low prevalence of parasites.

To address these predictions, I measured the prevalence of castrating trematodes and reproductive output in 12 lake populations of *H. anceps* to determine whether prevalence explained variation in fecundity. Then, because genetic and phenotypic differentiation among populations can be masked by differences in the native environment, I also measured reproductive output and age at first reproduction in the laboratory-reared offspring of the snails.

MATERIALS AND METHODS

Helisoma anceps (Pulmonata: Planorbidae) is a freshwater snail that is widely distributed across North America (Burch, 1982). The snail has an approximately one-year life cycle in natural populations (Fernandez and Esch, 1991; personal observation). In north-central Wisconsin, egg production begins in the spring and all individuals of the adult cohort have senesced by mid-July. Consequently during May, June, and part of July, the juvenile and adult cohorts overlap. This species is hermaphroditic (Brown, 1991) and can self-fertilize. Hence, the age at first reproduction is simply the age at which the snails begin to lay fertilized eggs.

This species is commonly infected by larval digenean trematodes. Digenean trematodes have complex life cycles containing at least two hosts. The adult worms live in vertebrates and the asexually reproducing larval stages live primarily in snails.

Field-collected snails

In late May 2002, I used a dip net to collect random samples of 75–100 adult *H. anceps* from the littoral zone of 12 lakes in north-central Wisconsin. In the laboratory, I measured each individual's shell length, and placed it in a 300 ml clear plastic cup filled with de-chlorinated water. The water in the cups was changed every 4 days and the snails were fed lettuce *ad libitum*. To measure reproductive output, I counted the total number of eggs produced by each individual in a 21 day period. To omit all infected snails from the study, I determined the infection status of all individuals at the end of the 21 day period using cercarial shedding and dissection. First, all of the snails were placed in 20 ml cups by a west-facing window without artificial light for 18 h. In this setting, the snails experience light conditions that vary from direct sunlight in the late afternoon to decreasing light at dusk, complete darkness at night, and increasing light at dawn. Because each species has a different cue for emergence, one of these light conditions will cause cercariae, a transmission stage of the larval trematode, to emerge from infected snails. Second, I dissected all snails that did not lay eggs and did not shed cercariae to determine whether pre-patent (immature) infections were present. Since reproduction in *H. anceps* commenced before the sampling period, infections could explain why some of the adult snails did not reproduce.

Laboratory-reared snails

Because differences in reproductive output among field-collected snails could be either phenotypic or genotypic, I also raised snails in the laboratory to determine whether genotypic differences existed among populations. Additionally, raising snails from eggs allowed me to determine the age at first reproduction. The snails raised in the laboratory hatched from eggs laid by the field-caught snails. To isolate these eggs, I transferred the field-caught snails to separate cups for 7 of the 21 days that reproductive output was measured. To balance the number of hatchlings per cup and to reduce density-dependent effects on growth, I reduced the density of juvenile snails in each cup, by choosing the survivors at random, twice in the first 8 weeks. Beginning at 8 weeks after hatching, each snail was maintained individually. I raised two offspring per parent such that at the beginning of data collection, samples sizes varied from 38 to 56 individuals per population.

For the duration of the study, the laboratory-reared snails were housed individually in 300 ml clear plastic cups filled with de-chlorinated treated water that was changed every fourth day. The rearing conditions were maintained at 19°C with a 15.5 : 8.5 h light/dark cycle (corresponding to the daylength on 21 June 2002 in Eau Claire, WI). The diet consisted of lettuce that was supplemented with fish flakes (Tetramin® Tropical Flakes) after 9 April 2003. Because algae eventually grew inside the cups, the snails received new cups approximately every 8 weeks, and the position of the cups relative to the light source was rotated every 4 weeks.

For each laboratory-reared snail, I measured age at first reproduction and reproductive output. Age at first reproduction is the number of days between the first appearance of eggs, determined by monitoring the cups every other day, and the snail's 'birthday'. Because the snails hatched from eggs laid over a 7 day period, I estimated the 'birthday' as day 4 of that week. To determine reproductive output, I counted egg production every other week over 27 weeks. Although I originally intended to measure life-time reproduction, I stopped measuring reproductive output at 27 weeks because most of the snails survived well beyond

their natural lifespan (based on the longevity of snails in lakes). Although laboratory conditions are unlikely to accurately reflect natural conditions, I did not want to introduce further differences by measuring reproductive output in the older snails. For analyses of reproductive output, I only included snails that survived the 27 weeks.

Prevalence of castrating trematodes

In addition to the parental generation of snails that I collected in May 2002, I also collected 50–120 snails from the littoral zone of the same 12 lakes in late June 2002 and 2003 to estimate prevalence of castrating trematodes. For each lake, prevalence was determined by counting the number of snails infected with one or more type of castrating trematode (Bush *et al.*, 1997). I used cercarial shedding to determine infection status; infected snails released cercariae after 18 h of varied light conditions. Although this method provides a conservative estimate of prevalence because it fails to detect snails with prepatent infections, the method is common practice in snail–trematode studies (e.g. Thornhill *et al.*, 1986; Schallig *et al.*, 1991; Schrag and Rollinson, 1994; Sorensen and Minchella, 1998). I calculated the mean prevalence as the arithmetic mean of the prevalence in 2002 and 2003.

For this study, I assumed that all of the trematodes that infected *H. anceps* were castrators. Two observations justify this assumption. First, asexual reproduction of trematodes inside their molluscan intermediate hosts nearly always causes complete castration of the host. In a recent review of 41 papers on the life-history characteristics of trematode-infected snails, trematodes ceased or reduced reproduction of their snail hosts in all of the studies (Sorenson and Minchella, 2001). Second, in my observations of the 67 infected field-caught snails, which I determined to be infected by both cercarial shedding and dissection, only one infected snail laid eggs during the entire sampling period (the infected snail laid 16 eggs, far below the mean of 59 eggs laid by the 507 uninfected snails).

Data analysis

Field-collected snails

For all analyses, I used a square-root transformation of the egg counts so that the values more closely approximated the normal distribution. I used analysis of variance to determine whether reproductive output differed among populations. To determine whether reproductive output of the field-caught snails was explained by mean prevalence of trematode parasites, I corrected the transformed egg counts for body size, because size is significantly related to fecundity in half of the individual populations and in all populations combined ($F = 17.8$, d.f. = 505, $P < 0.001$). To correct for size, I extracted the residuals from a linear regression of the transformed values of egg counts as a function of length. Then, I calculated the mean of the residuals for each population and used linear regression to determine whether the mean residuals were explained by mean prevalence.

Laboratory-reared snails

As with the egg counts of the field-collected animals, I conducted a square-root transformation on the total egg counts of the laboratory-reared snails to normalize the data. However, unlike the fecundity measurements for animals from the field, I did not correct for size in the egg totals because there was no positive relationship between size and fecundity

in any of the 12 populations of laboratory-reared animals [linear regressions for each population revealed a range of results from significantly negative relationships (e.g. $P = 0.001$ for Lake 3) to no relationship between reproductive output and size (e.g. $P = 0.603$ for South Shattuck)]. I used analysis of variance to determine whether age at first reproduction or reproductive output differed among populations. For each population, I calculated the mean age at first reproduction and mean reproductive effort and used linear regression to determine whether mean prevalence of trematodes explained the variation in either of these means. For the analyses of reproductive effort, I excluded all animals that died before 27 weeks ($n = 145$) and 23 snails that never reproduced during the study. These animals were distributed among nine populations. All statistics were conducted using the R statistical package (R Development Core Team, 2005).

RESULTS

Field-collected snails

A one-way analysis of variance (ANOVA) revealed significant differences in reproductive output among populations (Tables 1 and 2). However, these differences in reproductive output were not predicted by mean parasite prevalence ($r^2 = 0.011$, slope = 0.006, standard error of slope = 0.017, $P = 0.741$, d.f. = 10) (Fig. 1).

Table 1. Results of ANOVA comparing egg production in field-caught snails from 12 lake populations

Source	d.f.	MS	*F*	*P*
Lake	11	27.3	3.46	0.0001
Error	468	7.9		

Table 2. Mean prevalence of castrating trematodes for each population, calculated as the arithmetic mean of prevalence in 2002 and 2003

Population	Mean prevalence	Mean number of eggs	CV	*n*
Axehandle	55.0	54.4	55.0	35
Bob	4.0	68.3	78.2	44
Boot	7.8	57.6	60.3	37
Bradley	43.2	65.1	76.4	41
Chetek	14.4	60.6	63.5	42
Goose	17.6	44.4	95.6	38
Howe	2.7	65.9	55.9	37
Lake 10	2.8	49.6	86.1	28
Lake 3	2.2	64.9	76.3	46
Payne	20.9	75.4	14.4	53
S. Shattuck	5.5	32.4	93.2	37
Snails	12.1	49.9	74.5	42

Note: The mean, coefficient of variation (CV), and sample size for the number of eggs produced by field-caught snails in a 21 day period in the summer of 2002 are shown.

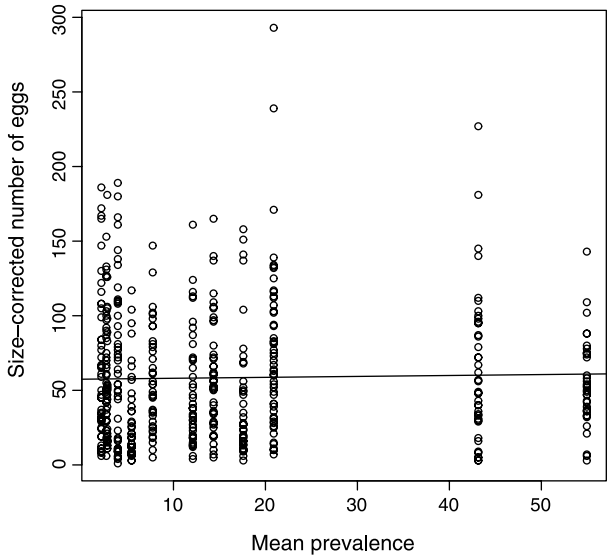

Fig. 1. Each point represents the size-corrected number of eggs produced over 21 days for one individual and the mean prevalence of castrating trematodes. The slope of the regression is not significantly different from zero ($P = 0.741$). Mean prevalence and mean egg production are given for each population in Table 2.

Laboratory-reared snails

A one-way ANOVA revealed significant differences in reproductive output and in age at first reproduction among populations (Tables 3 and 4). However, mean prevalence of castrating parasites did not predict mean reproductive output ($r^2 = 0.040$, slope = 0.05, standard error of the slope = 0.075, $P = 0.535$, d.f. = 10) (Fig. 2) or mean age at first reproduction ($r^2 = 0.003$, slope = –0.095, standard error of the slope = 0.556, $P = 0.867$, d.f. = 10) (Fig. 3) in the laboratory-reared snails.

DISCUSSION

Although reproductive output in both field-collected and laboratory-reared snails differed among populations, reproduction was not associated with prevalence of castrating trematodes. Similarly, age at first reproduction in the laboratory-reared snails was not related to parasite prevalence even though maturation age differed significantly among populations. These results are not consistent with predictions from life-history models. However, one model predicts the results found in this study under some conditions (Gandon *et al.*, 2002).

Similar to other life-history models, under most conditions Gandon and colleagues (2002) find that the evolutionarily stable reproductive effort of uninfected hosts from populations with parasites is higher than hosts living in the absence of parasites. However, similar to my results, the evolutionarily stable reproductive effort is the same between uninfected hosts from populations with and without parasites when virulence is high. In this model, virulence is measured as parasite-induced host mortality. Thus when virulence is high, direct transmission of parasites reduces prevalence. Consequently, a lower prevalence reduces

Table 3. Results of ANOVA comparing (a) reproductive output and (b) age at first reproduction among 12 lake populations of laboratory-reared snails

Analysis	Source	d.f.	MS	*F*	*P*
(a) Reproductive output	Lake	11	415.7	6.04	<0.001
	Error	259	68.8		
(b) Age at first reproduction	Lake	11	39537	20.31	<0.001
	Error	540	1947		

Note: For (a), the egg counts were square-root transformed, and only individuals that survived the entire 27 week period were included.

Table 4. Mean egg production (number of eggs) and mean age at first reproduction (days), with coefficients of variation and sample size for laboratory-reared animals in 2002–2003

	Reproductive output			Age at first reproduction		
Population	Mean	CV	*n*	Mean	CV	*n*
Axehandle	430.4	64.0	24	272.2	12.5	46
Bob	577.3	76.9	24	285.3	14.0	42
Boot	376.7	58.9	20	248.0	7.7	46
Bradley	337.7	81.7	21	307.1	14.3	54
Chetek	150.1	128.6	27	331.8	14.6	54
Goose	512.3	100.8	22	320.4	17.2	50
Howe	200.3	139.8	21	354.4	11.0	40
Lake 10	444.0	68.5	21	270.5	18.0	38
Lake 3	308.0	79.0	21	278.3	16.0	35
Payne	635.4	76.8	28	307.9	17.7	50
S. Shattuck	249.5	97.8	19	306.0	13.6	46
Snails	274.8	92.6	23	281.3	16.2	51

Note: Sample sizes differ between the two traits because all snails that died before the end of the sampling period of 27 weeks were omitted from the analyses of reproductive output.

selection pressure on reproductive effort such that reproductive effort is equivalent in individuals from populations with and without parasites under some conditions. Although I found no relationship between level of parasitism and one measure of reproductive effort in this study, my results are not consistent with the model because several of the lakes in this study have high prevalence (Table 2). Hence, there should be no reduction in the intensity of selection in these populations and consequently no decrease in evolutionarily stable reproductive effort is predicted.

There are several possible explanations for the absence of a relationship between parasite prevalence and both age at first reproduction and reproductive output. First, the prevalence of trematodes in 2002 and 2003 may not have been indicative of long-term selection pressure by parasites. It is possible that prevalence levels fluctuate or that levels have changed in recent time. However, there was strong consistency in prevalence between the

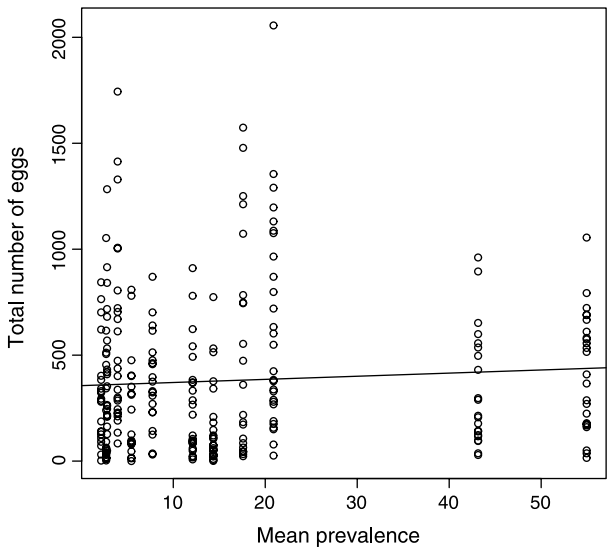

Fig. 2. Each point represents the total number of eggs produced over 27 weeks by one individual and the mean prevalence of castrating trematodes. The slope of the regression is not significantly different from zero ($P = 0.535$). For each population, mean prevalence is given in Table 2 and mean egg production is given in Table 4.

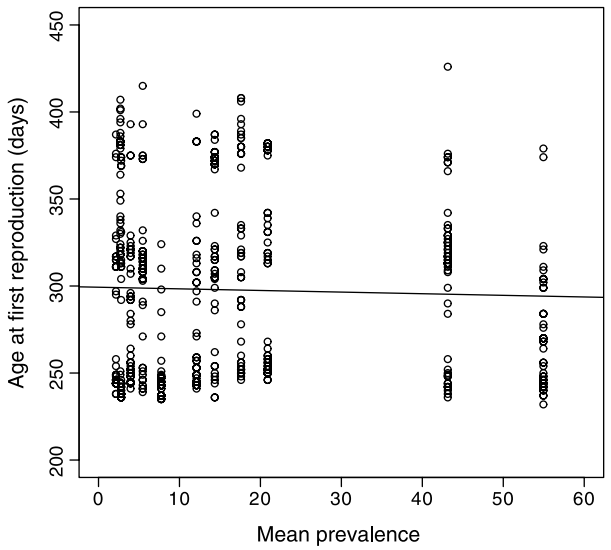

Fig. 3. Each point represents the age at first reproduction (in days) for one individual and the mean prevalence of castrating trematodes. The slope of the regression is not significantly different from zero ($P = 0.867$). For each population, mean prevalence is given in Table 2 and mean age at first reproduction is given in Table 4.

2 years in which it was examined; prevalence in 2002 strongly predicts prevalence in 2003 ($F = 52.11$, d.f. = 10, $P < 0.0001$, $r^2 = 0.84$). Although the similarity of prevalence between 2002 and 2003 suggests that prevalence does not fluctuate much in the short term, I do not have enough information to determine whether mean prevalence indicates long-term parasite prevalence.

Another possible explanation for why there was no relationship between prevalence and the two life-history traits is that other selective agents are causing evolution of life histories. I did not attempt to quantify differences among populations in predation pressure, over-winter survival, or other factors that could cause life-history evolution. These agents of selection may explain more of the variance in life-history evolution in some or all populations. For example, the laboratory-reared snails from Boot Lake matured early despite a low prevalence of parasites. This may be because some other selective agent in Boot Lake has caused selection for early maturity

Perhaps trade-offs among life-history traits have constrained the evolution of reproductive output and age at first reproduction. If the traits that are predicted to be under selection by parasites trade-off with other life-history traits, the response to selection should be hampered by the presence and strength of the trade-offs. However, in a related study of trade-offs in laboratory-reared *H. anceps*, the only costs of reproduction or early maturity were in growth, and these were not consistently found in all populations (A.C. Krist, in preparation). The study found no evidence for trade-offs between reproduction and survival or between early maturity and survival or total reproduction. The lack of consistent trade-offs with growth and the absence of other trade-offs suggest that they are unlikely to be constraining a response to selection in reproductive output and age at first reproduction in these populations.

Laboratory conditions may also explain the absence of a relationship between parasitism and life-history traits. The benefits of carrying out this study in the laboratory include the ability to measure traits of individuals and identical rearing conditions for all populations so that any effects of native environments are eliminated. However, it is these identical conditions that can be problematic when populations are not equally well adapted to the common laboratory conditions (Partridge, 1990). These genotype-by-environment interactions can create difficulties in studying life-history traits in the laboratory by obscuring the results of selection. For example, snails from Bradley Lake produced many more eggs when raised in the field than when raised in the laboratory, suggesting that this population is poorly adapted to laboratory conditions. This may also explain why laboratory-reared snails from Bradley Lake exhibited low fecundity in the laboratory despite predicted selection for high fecundity because of the high prevalence of parasites in this lake.

Assuming mean parasite prevalence is an accurate estimate of selection pressure, the results of this study suggest that if parasites are causing selection on the life-history traits of their snail hosts, they are not the primary driving force of selection in all populations. Although the selection pressure exerted by a high prevalence of castrating parasites is strong because the relative fitness of infected individuals is reduced to zero, it is possible that some populations are experiencing equally strong selection by common predators or other diseases. Hence the absence of a relationship between parasite prevalence and life-history traits can neither be interpreted as inconsistent with life-history theory, nor suggest that selection by castrating parasites is absent.

ACKNOWLEDGEMENTS

I am grateful to Alex Buerkle for statistical advice and Curt Lively and an anonymous reviewer for comments that improved the manuscript. I also thank Steve Chevalier, Tara Culligan, Lacey Eveland, Stephanie Haas, Zac Najacht, Trina Nowak, Breann Reilly, Laura VanZandt, and Abby Vogler for assistance in the field and laboratory. This study was supported by grants from the Office of University Research and Sponsored Programs at the University of Wisconsin-Eau Claire.

REFERENCES

Agnew, P., Bedhomme S., Haussy, C. and Michalakis, Y. 1999. Age and size at maturity of the mosquito *Culex pipiens* infected by the microsporidian parasite *Vavraia culicis*. *Proc. R. Soc. Lond. B*, **266**: 947–952.

Brown, K.M. 1991. Mollusca: Gastropoda. In *Ecology and Classification of North American Freshwater Invertebrates* (J.H. Thorp and A.P. Covich, eds.), pp. 285–314. San Diego, CA: Academic Press.

Burch, J.B. 1982. *Freshwater Snails (Mollusca: Gastropoda) of North America*. Cincinnatti, OH: US Environmental Protection Agency.

Bush, A.O., Lafferty, K.D., Lotz, J.M. and Shostak, A.W. 1997. Parasitology meets ecology on its own terms: Margolis *et al.* revisited. *J. Parasitol.*, **83**: 575–583.

Christe, P., Richner, H. and Oppliger, A. 1996. Begging, food provisioning, and nestling competition in great tit broods infested with ectoparasites. *Behav. Ecol.*, **7**: 127–131.

Ebert, D.E., Carius, H.J., Little, T. and Decaestecker, E. 2004. The evolution of virulence when parasites cause host castration and gigantism. *Am. Nat.*, **164**: S19–S32.

Fernandez, J. and Esch, G.W. 1991. Effect of parasitism on the growth rate of the pulmonate snail *Helisoma anceps*. *J. Parasitol.*, **77**: 937–944.

Forbes, M.R.L. 1993. Parasitism and host reproductive effort. *Oikos*, **67**: 444–450.

Gandon, S., Agnew, P. and Michalakis, Y. 2002. Coevolution between parasite virulence and host life-history traits. *Am. Nat.*, **160**: 374–388.

Hochberg, M.E., Michalakis, Y. and de Meeus T. 1992. Parasitism as a constraint on the rate of life-history evolution. *J. Evol. Biol.*, **5**: 491–504.

Jokela, J. and Lively, C.M. 1995. Parasites, sex, and early reproduction in a mixed population of freshwater snails. *Evolution*, **49**: 1268–1271.

Koella, J.C. and Restif, O. 2001. Coevolution of parasite virulence and host life history. *Ecol. Lett.*, **4**: 207–214.

Kozlowski, J. and Uchmanski, J. 1987. Optimal individual growth and reproduction in perennial species with indeterminate growth. *Evol. Ecol.*, **1**: 214–230.

Kozlowski, J. and Weigert, R.G. 1987. Optimal age and size at maturity in annuals and perennials with determinate growth. *Evol. Ecol.*, **1**: 231–244.

Krist, A.C. 2001. Variation in fecundity among populations of snails is predicted by prevalence of castrating parasites. *Evol. Ecol. Res.*, **3**: 191–197.

Lafferty, K.D. 1993. The marine snail, *Cerithidea californica*, matures at smaller sizes where parasitism is high. *Oikos*, **67**: 1–8.

Minchella, D.J. 1985. Host life-history variation in response to parasitism. *Parasitology*, **90**: 205–216.

Minchella, D.J. and Loverde, P.T. 1981. A cost of increased reproductive effort in the snail *Biomphalaria glabrata*. *Am. Nat.*, **118**: 876–881.

Partridge, L. 1990. An experimentalist's approach to the role of costs of reproduction in the evolution of life-histories. In *Towards a More Exact Ecology* (P.J. Grubb and I. Whittacker, eds.), pp. 231–246. Oxford: Blackwell Scientific.

Perrin N., Christe, P. and Richner, H. 1996. On host life-history responses to parasitism. *Oikos*, **75**: 317–321.

R Development Core Team. 2005. *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing (http://www.R-project.org).

Schallig, H.D., Sassen, M.J., Hordijk, P.L and De Jong-Brink, M. 1991. *Trichobilharzia ocellata*: influence of infection on the fecundity of its intermediate snail host *Lymnaea stagnalis* and cercarial induction of the release of schistosomin, a snail neuropeptide antagonizing female gonadotropic hormones. *Parasitology*, **102**: 85–91.

Schrag, S.J. and Rollinson, D. 1994. Effects of *Schistosoma haematobium* infection on reproductive success and male outcrossing ability in the simultaneous hermaphrodite, *Bulinus truncatus* (Gastropoda: Planorbidae). *Parasitology*, **108**: 27–34.

Sorci, G., Clobert, J. and Michalakis, Y. 1996. Cost of reproduction and cost of parasitism in the common lizard, *Lacerta vivipara*. *Oikos*, **76**: 121–130.

Sorensen, R.E. and Minchella, D.J. 1998. Parasite influences on host life history: *Echinostoma revolutum* parasitism on *Lymnaea elodes* snails. *Oecologia*, **115**: 188–195.

Sorensen, R.E. and Minchella, D.J. 2001. Snail–trematode life history interactions: past trends and future directions. *Parasitology*, **123**: S3–S18.

Thornhill, J.A., Jones, J.T. and Kusel, J.R. 1986. Increased oviposition and growth in immature *Biomphalaria glabrata* after exposure to *Schistosoma mansoni*. *Parasitology*, **93**: 443–450.