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Decreased reproductive investment of female threespine stickleback *Gasterosteus aculeatus* infected with the cestode *Schistocephalus solidus:* parasite adaptation, host adaptation, or side effect?

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Parasitic infections may cause alterations in host life history, including changes in reproductive investment (absolute amount of energy allocated to reproduction) and reproductive effort (proportion of available energy allocated to reproduction). Such changes in host life history may reflect: 1) a parasite tactic: the parasite adaptively manipulates energy flow within the host so that the host is induced to make a reduction in reproductive effort and reproductive investment, making more energy available to the parasite; 2) no tactic: there is no change in host reproductive effort and reproductive investment simply decreases as a side effect of the parasite depleting host energy stores; 3) a host tactic: the host adaptively increases reproductive effort in the face of infection and loss of body condition, reproductive investment possibly being reduced despite the increased reproductive effort. Females in Alaskan lake populations of threespine sticklebacks (*Gasterosteus aculeatus*) are capable of clutch production when parasitized by the cestode *Schistocephalus solidus* despite large relative parasite masses. We analyzed the somatic energy reserves, maturation stage and ovarian mass of female sticklebacks collected from an Alaska lake during a single reproductive season. We found that parasitized females were less likely to carry fully-matured gametes, had smaller ovarian masses, and had lower somatic energy stores than unparasitized females. The relationship between reproductive investment and energy storage did not differ between parasitized and unparasitized females. Thus, reproductive effort did not change in response to parasitic infection. We conclude there was no indication of either a parasite tactic or a host tactic. Simple nutrient theft is involved in the parasite's influence on host reproduction, consistent with an earlier hypothesis that reproductive curtailment in threespine sticklebacks is a side effect.

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Parasitic infection is often associated with changes in host life-history phenotype, such as an alteration in host reproductive performance. What is the ultimate significance of such changes? Parasites may evolve means to inhibit host reproduction in order to keep the host in relatively good somatic condition (Hurd 2001, Heins et al. 2004). Hosts may evolve induced responses to infection that involve changes in reproductive effort (RE, the proportion of available energy that is allocated to reproduction–Minchella 1985, Forbes 1993, Hurd 2001). The reproductive investment (absolute amount of energy devoted to reproduction) of parasitized indivi-

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duals frequently is less than that of unparasitized individuals (Hurd 2001). Whether change in reproductive investment associated with infection represents a change in RE cannot be fully understood unless the parasitic effect on host energy reserves is known as well. In the present study we compared unparasitized threespine sticklebacks (*Gasterosteus aculeatus* L.) with sticklebacks that were parasitized by the cestode (*Schistocephalus solidus* [Müller]), to assess reproductive investment of hosts relative to somatic energy reserves.

Infection by *S. solidus* in freshwater populations of stickleback is associated with multiple changes in host phenotype (Wootton 1984, LoBue and Bell 1993, Ness and Foster 1999) that derive from the life cycle of the parasite. The cestode has a complex life cycle, involving a free-swimming larva called a coracidium, a larval stage within copepods called a procercoid, a second larval stage in fish called a plerocercoid, and an adult worm in a piscivorous bird. Almost all of the parasite's growth occurs during the plerocercoid stage. Parasitized fish may have a distended abdomen and may carry a parasite mass exceeding the host's own mass. Parasitized fish show alterations in morphology (Barber and Svensson 2003), changes in behavior and color (LoBue and Bell 1993, Tierney et al. 1993, Ness and Foster 1999), increased or decreased growth rate (Arnott et al. 2000, Barber and Svensson 2003), reduced energy stores and somatic condition (Tierney et al. 1996, Barber and Svensson 2003, Bagamian et al. 2004), reduced probability of reproduction (McPhail and Peacock 1983, Tierney et al. 1996, Heins et al. 1999), and reductions in reproductive investment shown by decreased egg size (Heins and Baker 2003) and gonad size (Tierney et al. 1996, Huntingford et al. 2001, but see Barber and Svensson 2003). In some locales, most parasitized sticklebacks are incapable of reproducing (Tierney et al. 1996), but in the Cook Inlet region of Alaska most parasitized stickleback females are capable of producing clutches albeit at reduced frequency (Heins et al. 1999, 2002, Heins and Baker 2003).

There are three alternative interpretations for the reduced reproductive investment of parasitized sticklebacks (Minchella 1985, Dawkins 1990, Hurd 2001). The first possibility is that it is a parasite tactic: the parasite inhibits host reproductive function (e.g. through endocrine disruption), thereby conserving host energy and maximizing plerocercoid production. Parasitized fish in this case would have reduced RE and diminished reproductive investment relative to unparasitized fish. A second possibility is that it is neither a parasite tactic nor a host tactic, but is a by-product of the parasitic theft of host energy reserves. Reproductive investment is diminished but the RE of parasitized fish is comparable to unparasitized fish. A third possibility is that it is a host tactic: any reduction in reproductive investment belies an actual increase in the proportional allocation of available energy to reproduction, thereby offsetting in part the potential loss of fitness due to infection (Minchella 1985, Forbes 1993). Parasitized fish in this case would have higher RE than unparasitized fish, despite the reduced reproductive investment.

In this paper, we examine the effect of *S. solidus* on the life history of female *G. aculeatus* in a lacustrine population in the Cook Inlet area of Alaska. We compare the reproductive investment and somatic energy reserves of parasitized and unparasitized females to test whether parasitic infection alters the RE of the host. The metric of RE we use is the level of reproductive investment in ovarian mass relative to somatic energy reserves. A respective increase or decrease in RE of parasitized fish relative to unparasitized fish would be interpreted as either a host tactic or a parasite tactic.

Methods

We collected samples of threespine stickleback from Walby Lake (61.619° N, 149.211° W), which lies within the Matanuska-Susitna Valley of south-central Alaska (Heins et al. 1999, 2002). Collections were made on May 31, 2001 (87 females) and June 8, 2001 (253 females) using unbaited six-mm wire-mesh minnow traps. All fish were euthanized with an overdose of tricaine methanesulfonate (MS 222) and then fixed and stored in 10% buffered formalin.

We examined female stickleback following a 24-h soak in deionized water. For each specimen, we measured standard length (SL, to 0.1 mm) and blotted wet mass (WM, to 0.001 g). Upon dissection, we cut open the perivisceral cavity, and removed parasites, stomach contents, and ovaries. The parasites were counted and weighed (PM, to 0.001 g) and the ovaries were blotted and weighed (OM, to 0.001 g). Measurement of gonad mass was inadvertently omitted in 23 females; these females are excluded from analyses that include reproductive investment variables. Parasite index was calculated as: $PI = PM/WM$.

We classified females by reproductive stage based on the macroscopic appearance of the ovaries. We based our classification on the seven-stage system outlined by Baker et al. (1998) and Heins et al. (1999), consolidating these to three stages that signify major steps in investment of reproductive energy: females who contained only pre-vitellogenic oocytes lacking yolk reserves (PreV; including latent and early maturing), those who contained oocytes that were growing because of yolk loading or vitellogenesis (V; including late maturing, mature), and those who contained post-vitellogenic oocytes (PostV; including late mature, ripening, and ripe). Females sequentially cycle multiple times in a single reproductive season through the V and PostV

stages (the clutch-production cycle, Heins and Baker 1993).

We estimated somatic energy (i.e. energy in the nonreproductive portion of the body) from the mass of lipid and lean tissue, modifying slightly the methods used in previous analyses (Schultz and Conover 1997, 1999, Schultz et al. 1998). Dissected fish were frozen for a minimum of 24 h at −25°C, dehydrated in a lyophilizer for 24–48 h, then held at 25°C in a convection oven for 48 hours to stabilize and standardize weighing temperature and sample moisture. Specimens were weighed (dry mass 1: DM1) to the nearest 0.00001 g and placed in porous thimbles (Fisher Scientific, 22 mm diameter × 80 mm height Alundum extraction thimbles). We extracted lipid in a custom Soxhlet apparatus modified for processing about 20 specimens at a time, using petroleum ether to remove nonpolar storage lipids. After a five hour extraction (ca 15 cycles of solvent extraction), thimbles were replaced in the convection oven and held at 25°C again for 24 h. Specimens were weighed (DM2, to 0.00001 g) and were ashed in the thimbles for 8 h at 550°C. The specimens were weighed again (DM3, to 0.00001 g) after their temperature returned to 25°C. The following estimates are based on these masses:

lipid mass: (LiM, in g) = DM1 − DM2,

lean mass (LeM, in g) = DM2 − DM3, and

total somatic energy (TSE, in kcal) = 9.45 × LiM + 4.8 × LeM.

Compositional analysis was completed on 218 females.

We described and tested the effect of infection on host composition and energy. Except where noted, statistical analyses were conducted after SL, OM, TSE, and PM were log10 transformed. PI and GSI were arcsine-root transformed. Mean values of somatic energy statistics were calculated for each reproductive stage of parasitized and unparasitized fish. For this description, lipid and lean mass were normalized (LiM/DM1 × 100%, LeM/DM1 × 100%) and TSE was expressed as an energy condition index (EC, as in Bagamian et al. 2004). EC is the amount that the individual's energy departs from the expected value based on her size. It is calculated by modeling TSE against SL in a bivariate regression for all females; each individual's EC is her residual from the expected value in that regression. We tested the significance of parasite effects on TSE in two analyses. The first was an analysis of covariance, with TSE as the response variable, SL as a covariate, and reproductive stage and parasitic infection (presence or absence) as categorical predictors. The second analysis of parasite effects on somatic energy was conducted via multiple regression with TSE as the response variable and SL and PM as predictor variables, and was conducted only on parasitized PostV females.

To describe and test the effect of parasitism on host reproduction, we analyzed the frequency of parasitic infections in females by reproductive stage to test whether infection affected the ability to produce clutches. Patterns of prevalence were analyzed via logistic regression, coding infection as a binary variable (parasite present or absent) against two predictor variables, SL (continuous variable, not log10 transformed) and reproductive stage (categorical variable). We tested whether infection affected reproductive investment in PostV females via analysis of covariance: log-transformed OM was the response variable, parasite presence/absence was a categorical predictor, and log-transformed SL was a continuous covariate. To compare the reproductive investment of parasitized and unparasitized PostV females we estimated log10OM of each group at the overall mean SL, estimating the least-squares means (LSmeans: Searle et al. 1980, SAS Institute 1999). Back-transformed LSmeans were corrected for bias according to Sprugel (1983) and Newman (1993).

To test whether parasitic infection involves a tactical alteration of reproductive effort, we examined the independent effects of infection, SL, and energy reserves on reproductive investment. The response variable was OM. Energy reserves were expressed as EC rather than TSE to avoid collinearity with female size. The effect of infection was tested in analysis of covariance as a categorical variable (parasite present or absent) and was tested in multiple regression as a continuous variable (PM) among parasitized females only. In addition to evaluating the significance of coefficients in the multiple regression, we examined the structure coefficient or loading of each regressor. Loadings represent how well each regressor correlates with the linear combination of all regressors that best predicts the response variable; they can help to assess the predictive value of regressors when there is some collinearity among them (Dunlap and Landis 1988).

Results

Infection and host composition/energy

Females in later reproductive stages and females with parasites had less lipid mass and less somatic energy than early-stage or unparasitized females, respectively. Mean values for lipid mass (normalized LiM) and for energy (EC) were consistently lower for parasitized females in each reproductive stage and decreased for each reproductive stage (Table 1). In contrast, lean mass (normalized LeM) was higher in parasitized females and females in later reproductive stages. We confirmed the significance of the stage and parasite effects on TSE via analysis of covariance. The three-way interaction of main effects (parasite × stage × SL) and both two-way interactions involving parasite presence were not significant. When these nonsignificant interactions were

Table 1. Composition and energy reserves by reproductive stage and presence of parasites. Entries of the table are the sample size (N) and means (SE in parentheses) of normalized lipid and lean mass (as % of dry mass) and energy condition (in kcal), for parasitized and unparasitized females in three reproductive stages.

		No parasites		Parasites	
PreV					
	N	13		43	
	Lipid	8.3%	(2.1%)	2.5%	(0.56%)
	Lean	75%	(1.5%)	77%	(1.3%)
	Energy	0.085	(0.024)	−0.024	(0.025)
V					
	N	48		55	
	Lipid	3.1%	(0.58%)	2.9%	(0.67%)
	Lean	79%	(0.52%)	78%	(0.53%)
	Energy	0.026	(0.010)	−0.0057	(0.016)
PostV					
	N	36		27	
	Lipid	2.8%	(0.70%)	1.7%	(0.77%)
	Lean	78%	(0.47%)	79%	(0.56%)
	Energy	−0.0079	(0.023)	−0.023	(0.017)

eliminated, the stage × SL interaction was significant ($F_{2,211}=4.0$, $P=0.02$), as were the main effects (SL: $F_{1,211}=400$, $P<0.0001$; parasite: $F_{1,211}=7.1$, $P=0.0084$; stage: $F_{2,211}=4.0$, $P=0.019$). The slope of the TSE-SL relationship was close to the expected isometric value of 3 (slope = 3.4, SE = 0.27). The significant effect of parasitic infection on TSE was also confirmed via multiple regression conducted on parasitized PostV females. TSE was lower in females carrying a greater mass of parasites (slope of PM effect = −0.071, $t_{25}=-2.6$, $P=0.016$).

The effect of infection on host reproduction

Parasitized females were more likely to be in early reproductive stages than unparasitized females, but some parasitized females had nonetheless completed vitellogenesis. The proportion of individuals with parasites was highest among PreV females, intermediate among V females, and lowest among PostV females (Table 2). Parasite prevalence was higher among PreV females of all sizes, and was higher among V females than PostV females in all size classes but one (Fig. 1). Analysis by logistic regression revealed a significant change in prevalence with stage ($\chi^2_2=26$, $P<0.0001$) and no change in prevalence with SL ($\chi^2_1=3.4$, $P=0.067$).

Table 2. Prevalence of parasites by reproductive stage. Entries in the table are the proportion of females with parasites (P(parasite)) and the number of individuals (N) in three reproductive stages (PreV: previtellogenic; V: vitellogenic; PostV: postvitellogenic).

	P(parasite)	N
PreV	0.83	77
V	0.62	138
PostV	0.46	122

Parasitized PostV females exhibited lower levels of reproductive investment than unparasitized females. We evaluated the effects of length and parasite presence on reproductive investment in an analysis of covariance. The log10 SL × parasite interaction was not significant ($F_{1,99}=1.68$, $P=0.2$) and we analyzed a reduced model with only the main effects. Both length and parasite effects were significant (length: $F_{1,100}=290$, $P<0.0001$; parasite: $F_{1,100}=21$, $P<0.0001$). The OM of PostV females with parasites was 74% of unparasitized female OM (back-transformed LSmeans of 45 mm fish: 0.24 g vs 0.33 g).

Reproductive effort of PostV females was not affected by the presence of parasites. Ovarian mass increased with TSE and was similar in parasitized and unparasitized females (Fig. 2). We tested the effect of parasitic

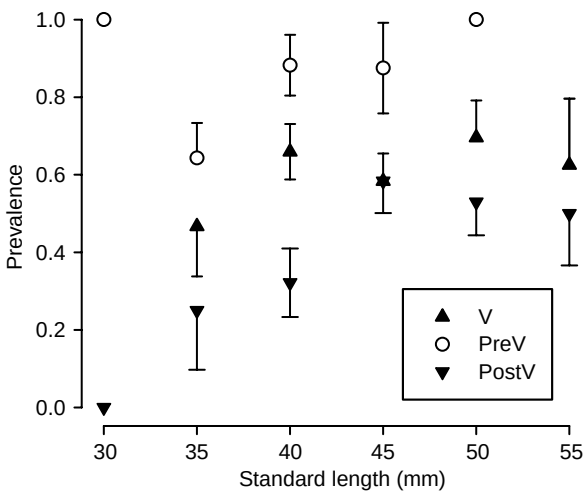

Fig. 1. Parasite prevalence and mass by size and reproductive stage. Mean prevalence (error bars are binomial standard deviations) is plotted against size (standard length, divided into 5-mm classes). Points without error bars represent multiple individuals; standard error is 0. Three female reproductive stages are plotted using different symbols.

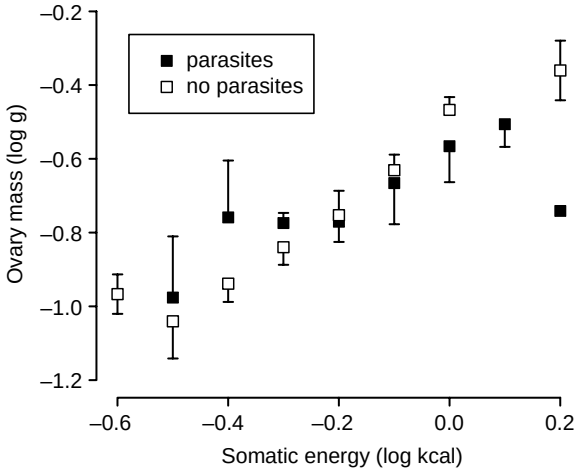

Fig. 2. Reproductive investment, energy reserves, and parasitism. Mean ovary mass (log-transformed, error bars are standard errors) is plotted against total somatic energy (in 0.1 log-kcal classes). Parasitized and unparasitized females are plotted using different symbols.

infection, SL, and energy condition on OM in an analysis of covariance. No interactions in the full model were significant. In the reduced model, the effect of SL on OM was significant and positive (slope = 3.1, SE = 0.32, $F_{1,54} = 95$, $P < 0.0001$). The effect of EC was also positive but was not significant (slope = 0.26, SE = 0.13, $F_{1,54} = 3.7$, $P = 0.061$), and the effect of parasitic infection was not significant ($F_{1,54} = 2.7$, $P = 0.1$).

Parasitized PostV females with a higher mass of worms appeared to exhibit reduced RE, but this is probably a spurious result. In multiple regression, the effect of SL on OM was significant and positive, the effect of EC was not significant, and the effect of PM was significant and negative (Table 3). The loading of PM, however, was small compared to that for EC. The significant regression coefficient and low loading for PM arise because PM is collinear with SL ($R = 0.52$, $p = 0.006$) but is not correlated with OM ($R = 0.034$, $p = 0.87$).

Discussion

Female sticklebacks in the Walby Lake population who are parasitized by the cestode *S. solidus* exhibit a reduction in reproductive investment but no reduction in RE, in agreement with the by-product hypothesis. Ovarian mass (OM) of parasitized females had the same relationship to somatic energy (TSE) as that in unparasitized females (Fig. 2). Among parasitized females, ovarian mass declined with parasite mass (Table 3), but the loading of the parasite mass was low suggesting an indirect effect arising from collinearity with female size (SL). The fact that parasitic infection did not alter the relationship between reproductive investment and energy reserves suggests that parasitized and unparasitized sticklebacks were allocating the same proportion of available resources to reproduction. We conclude that impaired reproduction does not represent a parasite tactic or a host tactic. Lower reproductive investment is instead a by-product of reduced somatic reserves caused by the demands of the parasite. Thus, our results support an earlier hypothesis (Heins and Baker 2003) that parasite-induced curtailment of reproduction in threespine stickleback represents a side effect.

Infection by *S. solidus* was associated with reduced reproductive investment. Parasitized females were more likely to be in earlier reproductive stages than unparasitized females (Fig. 1, Table 2), suggesting that parasitized females may progress through the clutch-production cycle at a reduced rate (Heins et al. 1999). The size-adjusted ovarian mass of parasitized females was smaller. Parasitized females produce smaller eggs (Heins and Baker 2003) and have reduced fecundity (D.C. Heins, pers. obs.).

The negative impact of *S. solidus* infection on stickleback reproduction appears to be universal. In a British Columbia population, 9% of the females parasitized with *S. solidus* were gravid, versus 62% that were not parasitized (McPhail and Peacock 1983). In Scotland (Tierney et al. 1996), 23% of parasitized females were mature, v 77% of unparasitized females and none of the mature females were "fully gravid". Our results indicate that the impact of infection on the Walby Lake population is comparatively mild; 28% of the parasitized females were in the PostV stage, compared to 50% of the unparasitized females. A previous analysis of the Walby Lake population also found high rates of clutch production among parasitized females (77%: Heins et al. 1999). The higher rates of successful clutch production among female sticklebacks from Walby Lake (Heins et al. 1999) and at least two other lakes (D.C. Heins, pers. obs.) in south-central Alaska may be partly attributable to the relatively late age of maturity among these fish (age 2+, versus age 1+ in lakes elsewhere: Wootton 1984, Heins et al. 1999). Ecological conditions may contribute to life history differences in infected females among threespine stickleback populations. Low summer

Table 3. Effect of length, somatic energy, and parasite mass on ovary mass of infected females in the PostV stage. Entries in table are results of multiple regression: estimate of regression slope (SE in parentheses), the t-statistic and P value under the null hypothesis that the slope is 0, and the loading for each predictor.

Variable	Estimate		t	P	loading
Intercept	−6.7	(1.3)	−5.2	<.0001	
SL	3.6	(0.75)	4.7	<.0001	0.88
Energy	−0.17	(0.35)	−0.48	0.64	0.23
Parasite mass	−0.13	(0.056)	−2.3	0.031	0.047

temperatures and resultant reduction in metabolic rates in the Alaska populations of sticklebacks may contribute to the lower impact of parasitism on reproductive investment in Alaska (Heins et al. 1999).

As expected, parasitism and vitellogenesis reduced somatic energy reserves (Table 1). The decreased energetic state of parasitized and post-vitellogenic females is reflected in lipid mass (normalized LiM) and in energy condition (EC). Lipid storage is expected disproportionately to affect energy condition because it has a greater caloric density and because lipid is the primary energy storage material in fish as well as many other animals (Shul'man 1974, Allen 1976, Pond 1981). In sticklebacks, lipid and glycogen stores are generally depleted from the liver and other somatic tissues when food intake does not meet the required energy amount necessary (Wootton 1973, Huntingford et al. 2001). Reduced condition and low energy content have been consistently observed among parasitized sticklebacks (Tierney et al. 1996, Barber and Svensson 2003, Bagamian et al. 2004). Nonetheless, lean mass (normalized LeM) was higher in females that were energetically depleted by parasitism or vitellogenesis (Table 1). This seemingly paradoxical result arises when two major tissue constituents are both normalized to total mass; in such cases they tend to negatively covary (Caulton and Bursell 1977).

Our analysis is the first to quantify differences in the pools of energy associated with reproduction and somatic storage between parasitized and unparasitized stickleback females. Previous studies of stickleback life history allocations and *Schistocephalus* infection differ from our findings in some cases. There is tantalizing evidence for parasite manipulation of host energy allocation. Ninespine stickleback (*Pungitius pungitius*) females parasitized with *Schistocephalus pungitii* have higher indices of body condition than unparasitized fish and yet are rarely reproductive (Heins et al. 2004). Another study (Barber and Svensson 2003) furnished suggestive evidence of an accelerated investment in reproduction among parasitized threespine stickleback. The slope of the ovarian mass–body mass relationship was significantly greater in experimentally infected fish than in unparasitized fish, and parasitized fish had greater ovarian masses than unparasitized fish of the same size. The ten-month old fish studied were not yet breeding; it would be interesting to know whether this apparently advanced investment in reproduction would have carried into greater reproductive investment and higher fecundity in this experimental setting. Our results, however, are consistent with an energetic analysis by Meakins (1974) of female sticklebacks that do not reproduce when infected. The estimated energy demands of the parasite appear to account for the energy that would have been devoted to reproduction in unparasitized fish.

The stickleback- *S. solidus* interaction would be expected to impose selection on the host for a change in life history that is induced when infection occurs. Such inducible tactics are likely to evolve when a constitutive resistance to infection is costly, relative to the cost of infection and the probability of being infected (Minchella 1985, Forbes 1993). The cost of infection and probability of infection both seem rather high in this interaction. The cost of infection is evident in the reduced gonosomatic index and the lower probability of producing a clutch. The cost of resistance is unknown. A resistance cost may arise because of a genetic linkage between resistance and other functional traits (e.g. lateral plate development: Colosimo et al. 2005).

Selection should favor a stickleback tactic of increasing RE in response to *Schistocephalus* infection. Circumstances that diminish future reproductive prospects favor the tactic of increasing RE (Schultz and Warner 1989). For example, some gastropod hosts increase RE when infected by larval trematodes that will eventually castrate the host (Minchella and Loverde 1981, Lafferty 1993). The situation of the stickleback host in Walby Lake appears to be similar. A female parasitized by *S. solidus* faces a future involving reduced egg production, reduced egg viability, complete loss of reproductive competency, and perhaps reduced survivorship. Presumably increasing the transfer of energy into offspring is better for her because energy that is reserved in the soma will soon serve the parasite's interests.

Parasitized females may not reallocate energy to reproduce more than they do because of physiological limitation, selection for reproductive restraint, or evolutionary inertia. We suggest that the best explanation is inertia: a tactic that changes energy allocation rules in response to parasitic infection would be favored, but has not (yet) appeared in this population. The alternative explanations can be rejected. By physiological limitation we mean that females are reproducing at their maximal rate; parasitized females simply are unable to express a higher reproductive effort because the maximum amount of available energy is devoted to reproduction. We reject this explanation because there are detectable storage lipids remaining in postvitellogenic females (Table 1). By selection for reproductive restraint, we mean that parasitized females might benefit by reserving somatic energy thereby increasing the prospects for overwinter survival and reproduction in the next season. This possibility merits closer study but appears to be unlikely, given that breeding in unparasitized fish appears to be limited to a single season (Wootton 1984).

From the perspective of parasite fitness, there should be selection in favor of a strategy of interfering with the host's ability to mature and transfer nutrients from soma to developing oocytes. This argument hinges on the assumption that energy retained in the host will benefit the parasite in some way. There are two ways in which

retained energy could benefit the parasite. One is extending the survival of the host (Heins et al. 2004). Survival of sticklebacks after the end of the breeding season is low (Wootton 1984), presumably because their reserves have been exhausted. Trophic transfer of the parasite to the definitive host, aquatic birds, will be more likely if host survivorship is lengthened beyond the end of the reproductive season (Heins et al. 2004). The other benefit to the parasite is enhanced growth. Presumably energy that is not lost to reproduction but is retained in the soma is available to the parasite for growth, thereby reducing the time to and maximizing the probability of the parasite reaching competency (Heins et al. 2002, 2004) for transfer to and reproduction in the definitive host. Recent research has shown that parasites grow larger in larger, faster growing hosts that are presumably in better condition (Heins et al. 2002, Barber 2005). The absence of a selectively advantageous parasite strategy is also seemingly attributable to evolutionary inertia.

We suggest several directions for further research on parasite and host tactics in the stickleback–cestode system. Dynamic optimization modeling would be useful to help clarify the fitness payoffs for host and parasite should infection alter host RE. Studies similar to ours can readily be conducted on other lacustrine populations of *G. aculeatus,* and on closely related pairs of host and parasite (e.g. *Pungitius* and *S. pungitii*). Findings of such studies will help to determine whether our results are typical of the stickleback-cestode interaction. More precise analysis of the energetic effects of parasitic infection will require work in laboratory conditions, permitting detailed energetic measurements and experimentally-controlled infection (Barber and Svensson 2003, Barber 2005). Finally, further evidence for coevolutionary changes in sticklebacks and the cestode should be explored through cross-infection experiments, pairing host and parasite from different locations.

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