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Parasite-associated growth enhancement in a fish–cestode system

Stephen A. Arnott[1,2*], Iain Barber[1,2] and Felicity A. Huntingford[1]

[1]*Fish Biology Group, Institute of Biomedical and Life Sciences, Division of Environmental and Evolutionary Biology, Graham Kerr Building, University of Glasgow, Glasgow G12 8QQ, UK*
[2]*Institute of Cell, Animal and Population Biology, Kings Buildings, University of Edinburgh, West Mains Road, Edinburgh EH9 3JT, UK*

Parasites impose an energetic cost upon their hosts, yet, paradoxically, instances have been reported in which infection is associated with enhanced, rather than diminished, host growth rates. Field studies of these parasite effects are problematic, since the pre-infection condition of the hosts is generally unknown. Here, we describe a laboratory experiment in which the growth rate and body condition of 76 laboratory-reared three-spined stickleback fishes were examined before, during and after each fish was fed the infective stage of the parasitic cestode *Schistocephalus solidus*. Twenty-one of these fishes went on to become infected by the cestode. Fishes were individually housed and provided with an abundant food supply to eliminate the potentially masking effects of variable competitive ability. Infection occurred independently of fish gender, size, body condition or pre-exposure growth rate. After exposure to the cestode, infected fishes grew faster (excluding parasite weight) and maintained a similar or better body condition compared with uninfected fishes, despite developing enlarged spleens. The accelerated growth could not be explained by reduced gonadal development. This result, one of few demonstrations of parasite-associated growth enhancement in fishes, is discussed with respect to other such parasite systems.

Keywords: cestode; *Gasterosteus*; gigantism; growth; host–parasite manipulation; *Schistocephalus solidus*

1. INTRODUCTION

The importance of parasites in controlling host populations and community structure is being increasingly recognized (Minchella & Scott 1991; Poulin 1997, 1998). Understanding these effects requires, among others, information on the influence of parasites on host growth, fecundity and survival. There are many examples of adverse effects of parasites on all of these variables, including rates of growth (e.g. Minchella 1985; Crompton 1984; Klingenberg *et al.* 1997; Thompson 1990; Mackenzie *et al.* 1987). However, there are also a few systems in which parasitic infection is apparently associated with an increase, rather than a decrease, in host growth rate. These systems may be particularly useful for understanding host–parasite relationships if we can determine the reasons for such an apparently counter-intuitive effect.

Enhanced growth has been reported in several species of molluscs infected with digenean trematodes (e.g. Rothschild & Rothschild 1939; Sorenson & Minchella 1998; Lim & Green 1991; Probst & Kube 1999). Although there is some doubt as to whether such parasite-induced somatic growth ('gigantism'; Poulin 1998) occurs in the wild (Fernandez & Esch 1991; Taskinen 1998), for at least some systems, this is the case (e.g. Gorbushin 1997). Gigantism is usually associated with, and probably caused by, functional castration of the host, which diverts nutrients from reproduction to somatic growth (e.g. Rothschild & Rothschild 1939; Sorenson & Minchella 1998). In the freshwater snail *Lymnea stagnalis* a peptide released from the central nervous system of individuals infected with the schistosome *Trichobilharzia ocellata* inhibits production of gonadotropic hormones and stimulates the secretion of various hormones that control growth (Hordijk *et al.* 1992). This phenomenon has been interpreted as a parasite adaptation (Baudoin 1975), since parasites gain a longer association with the host, but some data are more consistent with the view that growth enhancement is an adaptive response of the host to infection. For example, infection only increases growth if it occurs before the host reaches reproductive maturity (Keas & Esch 1997), and only in those populations that naturally coexist with the parasite (Ballabeni 1995).

A well-documented growth-enhancing effect has also been described for the non-castrating cestode *Spirometra mansonoides*, which induces strikingly enhanced muscular and skeletal growth in rats, mice, deer mice and hamsters (see Phares 1996). These changes are the result of secretions produced in the external surface of plerocercoids of *Spirometra mansonoides*, including a proteinase and a growth factor similar to mammalian growth hormone. The proteinase activity may facilitate the invasion of host tissue and the growth factor may suppress production of endogenous immunostimulants (Phares 1996).

An influence of macroparasites on growth has therefore been identified in two very different systems. However, other definitive examples have been difficult to detect, partly because many studies are observational rather than experimental. For example, during the first two years of life, whitefish (*Coregonus laveratus*) infected naturally with the cestode *Triaenophorus crassus* grow faster (according to back-calculations) than uninfected fishes, although beyond this age growth rates of infected fishes are relatively low. Fast-growing fishes when young may, by consuming more prey, be more exposed to infected intermediate hosts than are slow-growing fishes (Pulkkinen & Valtonen 1999). An experimental approach is clearly required in order to follow the condition of individual

*Author for correspondence (s.a.arnott@udcf.gla.ac.uk).

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hosts during both their pre-infection as well as post-infection development.

Using such an approach, Ballabeni & Ward (1993) found no changes in growth in European minnows (*Phoxinus phoxinus*) infected with *Diplostomum phoxini*. However, in a later experiment fishes infected with a low parasite dose grew faster than either those with a high dose or uninfected controls (Ballabeni 1994), possibly due to the provenance of the minnows and their housing conditions. Thus, unambiguous examples of parasite-associated growth enhancement in fishes are rare.

In this article, we report an experimental demonstration of parasite-associated growth enhancement in fishes using a well-studied host–parasite system, the cestode *Schistocephalus solidus* and its second intermediate host, the three-spined stickleback *Gasterosteus aculeatus*. Three-spined sticklebacks are small (30–100 mm) fishes that inhabit a diverse range of aquatic habitats across the Northern Hemisphere (Wootton 1976). In certain lacustrine populations, sticklebacks serve as intermediate hosts for *S. solidus*. The definitive host of the parasite is usually a piscivorous bird, which becomes infected after ingesting infected sticklebacks. Eggs pass out with the faeces of the avian host and hatch to release free-swimming first-stage larvae (coracidia), which develop into procercoid larvae in the haemocoel of cyclopoid copepods that eat them (Smyth 1962). Sticklebacks acquire plerocercoid larvae after eating infected copepods and these then grow inside the fish's body cavity. Infected sticklebacks suffer a number of adverse effects. The fish's abdomen becomes grossly distorted as the plerocercoid grows inside it, sometimes matching its host in weight (Arme & Owen 1967), and behavioural changes may occur that make them more vulnerable to predation (Giles 1983; Milinski 1985; Barber & Huntingford 1995). Infected fishes are more vulnerable to starvation (Walkey & Meakins 1970), and in the wild lose condition more rapidly during autumn and gain condition more slowly in the spring (Tierney *et al.* 1996). Reproduction can be impaired in heavily infected fishes (Pennycuick 1971; Tierney *et al.* 1996), although this is not always the case (McPhail & Peacock 1983).

These studies used naturally infected fishes at different stages of infection, so it is hard to disentangle cause and effect. In our present study, we exposed laboratory-reared sticklebacks to a controlled dose of *S. solidus*, the parent populations of each originating from a site where *S. solidus* infection is prevalent (Tierney *et al.* 1996). In addition, we provided food at a standardized, high level and housed the fishes individually to avoid the reported effects of infection on competitive ability (Cunningham *et al.* 1994; Barber & Ruxton 1998). This approach allowed us to monitor the condition of individual fish both before and after exposure to the parasite. In particular, we have addressed the following questions. How does *S. solidus* infection affect (i) the growth rate of three-spined stickleback hosts, and (ii) resource allocation between somatic and gonadal body compartments of the fish?

2. METHODS

(a) *Culture of* Schistocephalus solidus

Adult *S. solidus* were cultured *in vitro* from infective (> 50 mg) cestode plerocercoids using a technique similar to that described by Smyth (1962). Plerocercoids were removed aseptically from naturally infected sticklebacks (Inverleith Pond, Edinburgh, UK; 55°55′ N, 03°10′ W) and transferred to 6.3 mm diameter dialysis tubing (Visking, UK) filled with horse serum (Sigma H1138; Sigma Aldrich Ltd, Poole, UK). The tubing was suspended within a 100-ml test-tube filled with horse serum and placed in a darkened shaking incubator at 40 °C for 65 h. Eggs laid by the cestodes were removed, rinsed in distilled water, transferred to a sealed vial of tap water and kept in the dark at 25 °C for four to six weeks before exposing them to light to induce hatching. Laboratory-reared cyclopid copepods (*Cyclops strenuus abyssorum*; Sciento, Bury, UK) were infected with *S. solidus* by mixing them with hatched coracidia and maintaining them at 25 °C for a further period of six weeks. The copepods were individually examined under a compound microscope (immobilized by placing them in a drop of carbonated water) and segregated according to the number of procercoids each harboured. By this stage, procercoids had developed a cercomer, which indicates that they are sufficiently developed to be infective to sticklebacks (Smyth 1969).

(b) *Experimental infections and husbandry*

The fishes used in this experiment were the offspring of *in vitro* fertilizations of nine female and 18 male freshwater sticklebacks from Inverleith Pond, which has a recorded history of *S. solidus* infection (Tierney 1994; Tierney *et al.* 1996). Broods from each female were tended by hand in separate incubators until hatching. Fry were maintained at 15–17 °C on a 16 L:8 D cycle and initially fed on *Artemia* until they were large enough to accept frozen chopped bloodworms (larval chironomids).

After about three months, 76 fishes were chosen at random and isolated within individually labelled 1-l containers with perforated walls. Groups of four to six containers were accommodated together (i.e. shared water) in a series of 12-l tanks. Fishes were kept at 12–14 °C on a 12 L:12 D cycle and fed to satiation one to two times a day on frozen bloodworms. Seventy of the fishes were measured (total length) and weighed (after 24 h starvation) four times: on day 1 (the day they were moved into isolation), day 71, day 90 and day 150. The remaining six fishes were measured and weighed only on the last three occasions, having been isolated slightly later than the initial batch.

All of the experimental fishes were exposed to a single dose of infective *S. solidus* procercoids because previous experiments indicate that infectivity is typically low (Tierney 1991). Each fish was fed a single copepod infected with *S. solidus* on day 83 of the experiment, at which time the mean fish length was 41 mm (range 32–51 mm). Each fish was starved for 24 h and then placed in a small Perspex receptacle with 250-ml water and a single copepod that had been scored for infective procercoids. The number of procercoids per copepod was either one ($n = 34$), two ($n = 36$) or three ($n = 6$), depending upon availability. Fishes were left for 3–5 h, but before being returned to their holding tanks, each receptacle was visually examined twice under a high-power light source to ensure that the copepod had been eaten. (Blind tests on 30 receptacles without fishes, but allocated 15 copepods between them at random, indicated that this method was 100% accurate.) Once returned, fishes were maintained and fed to satiation daily on bloodworms.

(c) *Post-mortem examination*

The fishes were killed by an overdose of benzocaine anaesthetic on day 150 and dissected immediately. The gender of

each fish and the number of plerocercoids in its body cavity were recorded. Wet-weight measurements (to 0.001 g) were taken of the following: total weight (fish + parasites, if present), *S. solidus* plerocercoid(s) (if present), liver, gonads, spleen, visceral fat, carcass (i.e. the remaining fish parts minus intestines). In addition, dry-weight measurements of the liver and carcass were obtained after freeze-drying to the point of constant weight.

(d) *Data analysis*

All statistical calculations were performed using Minitab 12.2 software (Minitab Inc., PA, USA). Daily specific growth rates (SGR) for all fishes were calculated for the pre-exposure period (up to day 71), the infection period (days 71 to 90) and the post-infection period (days 90 to 150) according to the formula

$$\mathrm{SGR}(\%\mathrm{day}^{-1}) = 100[\ln(W_t) - \ln(W_0)]/t,$$

where W_0 represents weight in grams at the beginning of the growth period, W_t the weight at the end and t the number of growth days. For infected fishes, weights of the fish alone were used, so growth estimates do not include changes in parasite weight.

SGR was negatively related to fish length over each experimental period (linear regressions of $\log_e$-transformed data, $p < 0.004$ in all instances) and so the general linear model function of Minitab was used to examine the relationship between infective status and growth. For each growth period, infection category (infected fishes versus uninfected) was entered as a fixed factor, with fish gender as a nested factor and fish length as a covariate. Differences between slopes were initially tested by including all interactive terms in the model. If a significant gender interaction was found, further analysis was performed separately for data from male and female fishes (for brevity, only the lowest p-value has been quoted for interactive terms); otherwise, elevations were tested by removing the interactive factors from the model.

The relative growth performance (RGP) of an individual fish with respect to all others was quantified using the standardized residuals (i.e. residual/s.d. of residuals) from SGR versus length regressions. A fish's RGP was low if it grew slower than average for its size (negative residual) and high if it grew faster than average (positive residual). Pearson product-moment correlation and one-sample t-tests were used to examine relationships between RGP before and after parasite exposure.

Various body condition parameters were derived from the measurements of whole-fish wet weight (minus parasite weight), visceral fat wet weight, carcass dry weight and liver dry weight. Reproductive condition was derived from gonad wet weight. All variables were related to body length, and the influence of gender and infective status examined as above.

3. RESULTS

(a) *General infection characteristics*

Overall, 21 out of the 76 fishes (27.6%) exposed to procercoids became infected, with no effect of gender (males, 36.4% infected, $n = 33$; females, 20.9% infected, $n = 43$; Fisher's exact test, $p = 0.32$). The majority of fishes contained just one plerocercoid, but three of the fishes had two parasites and one fish contained three. No significant relationship was detected between the number of procercoids consumed by a fish and its likelihood of becoming infected (logistical regression, $p = 0.60$). The mean (±s.d.) wet weight of plerocercoids was 30 (±19) mg, and this was similar in males and females ($t = 1.89$, $p = 0.08$). The spleens of infected fishes were approximately 2.6 times heavier than those of uninfected fishes (means, 0.003 g, cf. 0.001 g, respectively; $F_{1,17} = 91.05$, $p < 0.001$), and the degree of spleen enlargement was positively correlated with total plerocercoid weight ($r = 0.714$, $n = 21$, $p < 0.0001$).

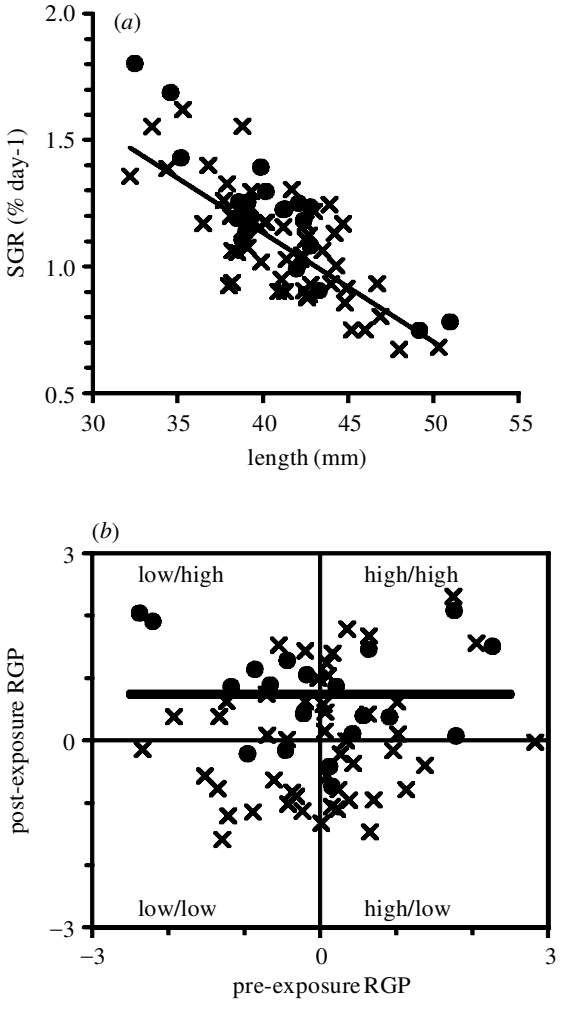

Figure 1. Effects of *S. solidus* upon specific growth rate (SGR) of three-spined sticklebacks. (*a*) Relationship between SGR and fish length (day 90) during the period after (days 90–150) being exposed to *S. solidus*. The fish that went on to develop infections (filled circles) had higher SGRs than those that did not develop infections (crosses and fitted regression line). (*b*) Relationship between relative growth performance (RGP, see § 2 for details) before exposure (days 1–71) and after exposure (days 90–150) to *S. solidus*. Pre- and post-exposure RGP were not related in uninfected fishes (crosses), whereas post-exposure RGP of infected fishes (filled circles) lay significantly above the origin (bold line, mean infected post-exposure RGP).

(b) *Infection and growth*

Between days 1 and 71, before being exposed to the parasite, the slopes of the relationship between SGR and fish length differed between male and female fishes within infection categories ($p < 0.05$). Testing the genders

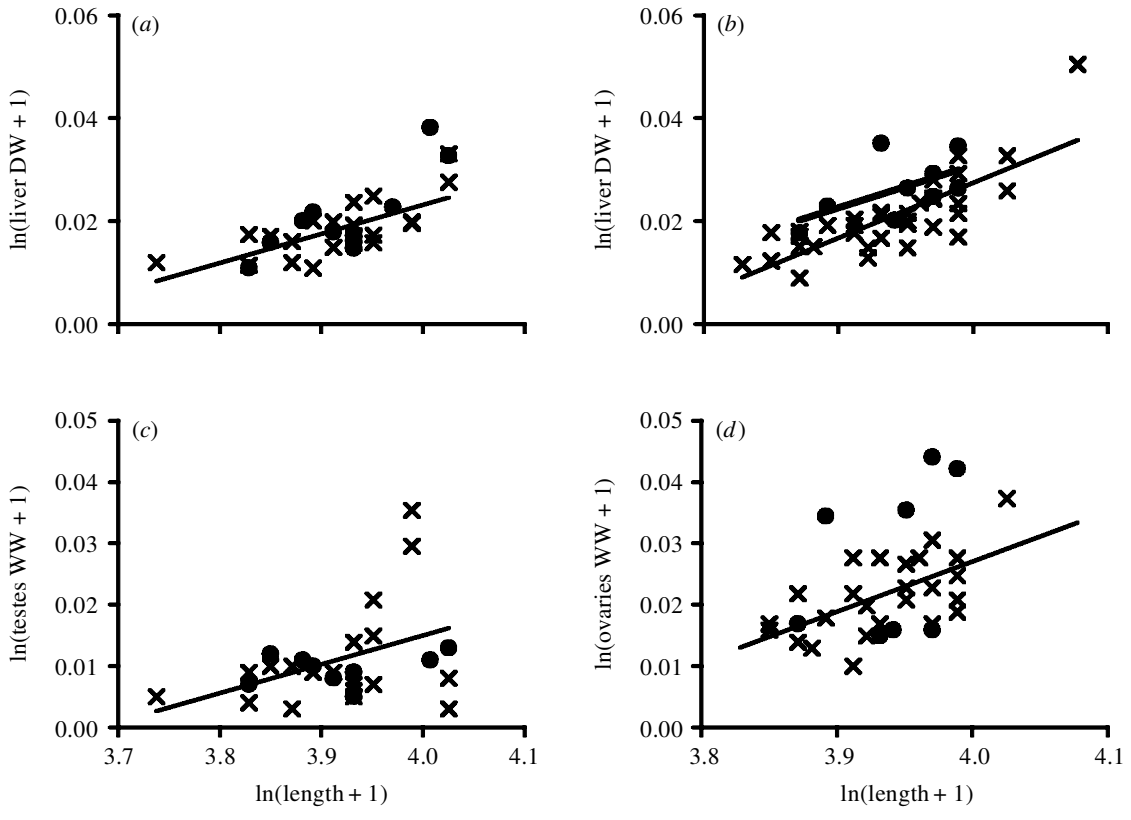

Figure 2. Effects of *S. solidus* upon resource allocation in immature three-spined sticklebacks. Relationships between fish length and (*a*) male liver dry weight (DW), (*b*) female liver dry weight, (*c*) male testes wet weight (WW), (*d*) female ovaries wet weight. With the exception of female livers, infection had no significant effect upon any of the body components. Thin lines, regression fitted to uninfected fishes; bold line, regression fitted to infected fishes (if different).

separately showed no difference in slope (females, $p > 0.7$; males, $p > 0.7$) or elevation (females, $F_{1,36} = 0.07$, $p = 0.786$; males, $F_{1,27} = 1.21$, $p = 0.281$) between future-infected versus future-uninfected fishes.

Between days 71 and 90, spanning the exposure period, no differences were found between the slopes of the SGR relationships ($p > 0.6$). Regression elevations did not differ between male and female fishes ($F_{2,64} = 1.69$, $p = 0.193$), nor between the infection categories ($F_{1,64} = 0.01$, $p = 0.959$). Thus, both before and during parasite exposure, growth rates were similar between the infection categories of fishes.

This is in marked contrast to the growth rates after infection (days 90 to 150). During the post-exposure period, the slopes relating SGR to length did not differ between the experimental groups ($p > 0.5$), and gender had no significant effect upon the regression elevations ($F_{2,71} = 0.08$, $p = 0.925$). However, infection status did have a significant effect ($F_{2,71} = 6.63$, $p = 0.012$), indicating that infected fishes grew faster than uninfected fishes of equivalent length (figure 1*a*). This was not simply the result of the infected fishes having enlarged spleens, since the absolute weight of this organ was so small (SGR ignoring spleen weight; $F_{2,71} = 5.99$, $p = 0.017$).

Among both the infected and uninfected fishes, and RGP of individual fish before exposure did not correlate with RGP after exposure (uninfected, $r = 0.22$, $n = 49$, $p = 0.12$; infected, $r = -0.15$, $n = 21$, $p = 0.52$). However, the RGP of infected fishes after exposure was significantly greater than zero (mean = 0.74, $t_{20} = 4.0$, $p < 0.001$). The reasons for this are twofold; first, future-infected fishes with a high RGP before exposure mostly maintained a high RGP once they become infected. Second, future-infected fishes with low RGPs before exposure shifted towards having high RGPs after infection (see figure 1*b*).

(c) *Infection and body condition*

In considering the whole-body condition of the sticklebacks, no difference was found between fish groups (gender or infection status) with regard to the slopes describing fish wet weight against length ($p > 0.34$ on day 1; $p > 0.61$ on day 90; $p > 0.08$ on day 150). At the start of the experiment, male fishes were the same weight as females for a given length ($F_{2,65} = 0.77$, $p = 0.47$), but by the end males were heavier ($F_{2,71} = 6.55$, $p < 0.002$). The effect of infection status was not significant at any point during the experiment (most conservative occasion, day 90: $F_{1,71} = 0.017$, $p = 0.151$).

At the end of the experiment, carcass dry weight for fishes of a given length was greater in males than in females ($F_{2,70} = 7.11$, $p = 0.002$) and there was a tendency for infected fishes to be slightly heavier than uninfected fishes of the same gender ($F_{1,70} = 3.69$, $p = 0.059$).

Among the uninfected fishes, the slopes of dry liver weight against length differed significantly between male and female fishes ($p < 0.05$), and so each gender was tested separately. Dry liver weights did not differ between infected and uninfected males (slopes, $p > 0.1$; elevations, $F_{1,30} = 0.73$, $p = 0.401$; figure 2*a*), but were approximately 17% heavier in infected compared to uninfected females

(slopes, $p > 0.2$; elevations, $F_{1,41} = 12.4$, $p < 0.001$; figure 2*b*). The degree of liver enlargement in females (i.e. weight–length residual) was also positively correlated with the total wet weight of *S. solidus* found within the fish ($r = 0.748$, $n = 9$, $p = 0.021$), whereas this was not true for males ($r = 0.204$, $n = 12$, $p = 0.525$).

Neither gender nor infection status affected the visceral fat weight of fishes at the end of the experiment (slopes, $p > 0.21$; elevations, gender, $F_{2,71} = 0.37$, $p = 0.69$; infection, $F_{1,71} = 1.57$, $p = 0.214$).

(d) *Infection and gonadal development*

None of the fishes had attained sexual maturity by the time of sacrifice, ovaries comprising, on average, 2.4% of their body mass and testes 1.2% (the immature condition; Wootton 1984). The slopes of gonad weight versus length did not differ between male and female fishes or between infected and uninfected fishes ($p > 0.75$), although the gonads of female fishes were significantly heavier than those of male fishes ($F_{2,59} = 28.79$, $p < 0.001$). However, within each gender, infection status had no effect upon the elevation of the gonad weight against length relationships ($F_{1,59} = 0.33$, $p = 0.568$; figure 2*c*,*d*).

4. DISCUSSION

This study has shown that in the weeks following parasite exposure, with an abundant food supply and in the absence of competition, sticklebacks that become infected with *S. solidus* show greater somatic growth than those that remain uninfected. This is despite growing at similar rates before infection and maintaining similar gonadal investment post-infection.

Levels of infection in our experiment were low (28%), which is consistent with the findings of Tierney (1991). Other stages of the parasite's life cycle also have incomplete success when infecting new hosts (Wedekind 1997; Tierney & Crompton 1992; Smyth 1969). The precise reasons for this remain unclear. It may be that phenotypic differences between individual parasites affect their ability to invade hosts and establish within them. Alternatively, or additionally, potential hosts may vary in their ability to resist infection.

Examination of individual growth rates showed that those fishes with a low RGP before parasite exposure had a high RGP if they became infected, whilst fishes with a high RGP before exposure maintained this high level if they became infected. There was no consistent relationship between RGP of individuals before and after exposure in fishes that did not become infected.

The higher growth rate of infected sticklebacks is especially surprising because it is achieved despite two significant energetic drains. First, the plerocercoid exploits host-derived energy to fuel its own growth from a microscopic larva to *ca.* 30 mg within ten weeks. Second, infected fishes apparently mount an immune response against the infection, evinced by the spleens of infected sticklebacks, which increased in size with respect to plerocercoid weight. Taylor & Hoole (1994) have also reported spleen enlargement in cyprinids infected with the closely related cestode *Ligula intestinalis*. Despite this response, at the end of our experiment, the somatic condition of infected fishes was, if anything, marginally higher compared to uninfected fishes, whilst the livers of infected females were larger than those of uninfected females, and positively related to plerocercoid weight. Liver size is usually indicative of energy reserves in sticklebacks, with females in natural populations storing more energy in their livers than males before the breeding season (Wootton 1984). This inherent physiological difference between genders may account for the different resource allocation responses of infected males and females.

Whether the growth effects we have observed here represents a response by the host to infection, or manipulation by *S. solidus* of the host, remains equivocal. Genital primordia do not develop in *S. solidus* until plerocercoids reach *ca.* 19 mg (Hopkins & McCaig 1963), and eggs are usually only produced by those larger than 50 mg (Tierney & Crompton 1992). Host survival is thus critical to *S. solidus* during its development, as is the ability of the host to eventually grow sufficiently large enough to accommodate a plerocercoid > 50 mg. Therefore, plerocercoid fitness is intimately linked to the payoff between parasite versus host energy allocation.

Enhanced growth in infected sticklebacks may represent an adaptive response that promotes host survival by ameliorating the risk of starvation, particularly over the winter, since the population at Inverleith becomes infected in the autumn and generally does not produce mature plerocercoids until the next spring (Tierney *et al.* 1996). Among fish populations in general, larger individuals have a lower metabolic rate per unit mass than smaller fishes (Clarke & Johnston 1999), making them less prone to overwinter starvation and mortality in temperate habitats (e.g. Berg & Bremset 1998; Post *et al.* 1998; Schultz *et al.* 1998; Gotceitas *et al.* 1999). As such, enhanced growth of infected sticklebacks may act as an 'insurance' that reduces the risk of starvation-induced mortality over the winter, despite the rising energy demands as *S. solidus* develops. Under more natural conditions, the positive effects of infection on growth that we have observed may help counteract (and be masked by) other unavoidable negative effects, such as reduced competitiveness.

The fact that enhanced host growth in some other host–parasite systems can be attributed to parasite secretions (e.g. Phares 1996; Hordijk *et al.* 1992) may implicate parasite adaptation in these instances rather than a host response. The argument that enhanced growth may represent a host response that enables it to outlive infection (Ballabeni 1995) does not hold in Inverleith sticklebacks, since they are an annual population and there is no evidence to suggest that fishes can eliminate a mature plerocercoid once it has formed inside them.

It has been suggested by others that *S. solidus* affects the behaviour of sticklebacks in a manner that makes them more vulnerable to predation by piscivorous birds (e.g. Giles 1983; Milinski 1985; Barber & Huntingford 1995). Similarly, enhanced growth may make host sticklebacks more vulnerable to predatory birds that select the largest available fishes from the population, as do cormorants (*Phalacrocorax carbo*) (Van der Veer *et al.* 1997), which are frequent stickleback predators at Inverleith Pond.

Concerning the mechanisms by which infection generates enhanced growth, food intake is not always enhanced in molluscs exhibiting gigantism, but host activity can be

decreased (Mouritsen & Jensen 1994). Gigantism in molluscs may also depend upon habitat food density (Fernandez & Esch 1991). Mice infected with *Spirometra mansonoides* do not eat more than uninfected animals, but convert food more efficiently (Phares 1996). For logistical reasons, we did not measure how much food our experimental subjects ate, or how active they were, so their enhanced growth could be the result of increased appetite, increased food conversion efficiency, reduced activity, or a combination of these. *S. solidus* is known to influence the feeding behaviour in its stickleback host, but the reported effects are complex and depend on the intensity of infection and environmental factors (e.g. Milinski 1985, 1990; Cunningham *et al.* 1994; Tierney 1994; Barber & Huntingford 1995; Ranta 1995).

Enhanced growth in mice infected with *Spirometra mansonoides* (Phares 1996) and in snails infected with digenean worms (Hordijk *et al.* 1992) is a consequence of secretion of growth enhancers by the parasites. Extracts from *S. solidus* procercoids contain a chymotrypsin-like proteinase that may aid in penetrating the intestinal wall of stickleback intermediate hosts, and the tegument of plerocercoids shows protease activity, possibly to allow peptide degradation into a form that can be absorbed by the parasite (Polzer & Conradt 1994). We are unaware of any studies that have yet investigated hormonal production in *S. solidus*, although the production of specialized plerocercoid growth factors by the related cestode *Spirometra mansonoides* makes this an obvious choice for future investigation.

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