Optimal growth strategies of larval helminths in their intermediate hosts

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Abstract

We consider optimal growth of larval stages in complex parasite life cycles where there is no constraint because of host immune responses. Our model predicts an individual's asymptotic size in its intermediate host, with and without competition from conspecific larvae. We match observed variations in larval growth patterns in pseudophyllid cestodes with theoretical predictions of our model. If survival of the host is vital for transmission, larvae should reduce asymptotic size as intensity increases, to avoid killing the host. The life history strategy (LHS) model predicts a size reduction <1/intensity, thus increasing the parasite burden on the host. We discuss whether body size of competing parasites is an evolved LHS or simply reflects resource constraints (RC) on growth fixed by the host, leading to a constant total burden with intensity. Growth under competition appears comparable with ''the tragedy of the commons'', much analysed in social sciences. Our LHS prediction suggests that evolution generates a solution that seems cooperative but is actually selfish.

Introduction

Many parasites have complex life cycles, with larval stages in intermediate hosts followed by reproduction of the mature adult in the definitive host. Here, we consider how life history parameters of larval stages may become optimized by natural selection in cases where there is no constraint on the time available for growth because of the immune responses of the host.

Our model aims to predict an individual's final (asymptotic) size in its intermediate host, with and without competition from other larvae of the same species. Our approach has been devised mainly with cestode larvae in mind, but would apply directly for most acanthocephalan larvae, perhaps nematodes and any other parasite larvae where there is no asexual reproduction in the intermediate host. In particular, we seek to explain the growth patterns for which data are available when larval parasites occur concurrently in the same intermediate host, enabling qualitative comparison with theoretical predictions. This has parallel with studies on the evolution of virulence, which have considered the fact that parasites face a trade-off between prudent exploitation and rapid reproduction or growth rate (Frank, 1996), for which there is some empirical evidence (e.g. Ebert, 1998).

If a given host has no value to a parasite larva other than to provide it with food, we might expect with increasing benefits to size that the parasite may grow until it consumes all the host's tissues; this situation appears to be approached with many insect parasitoids (Godfray, 1994). If, however, some degree of host viability is essential to allow the larva to be transmitted to its next host, as in most helminths, the benefits of increased size will be constrained evolutionarily by the decline in host viability. Our model trades off the benefits of continued growth against the costs of reduced prospects of transmission to the next host, and seeks an optimal asymptotic size that maximizes the parasite's expected future reproductive success. Under competition from conspecifics in a given host, there are two possible interpretations of growth patterns. The first is that different growth patterns under different levels of competition simply reflect the constraints of the available host resources. This has been the classical interpretation of growth differences in helminths under competition (e.g. for procercoids; Michajlow, 1953; Rosen & Dick, 1967).

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The second is that a larva can respond to cues of the presence and number of competing larvae by changing its growth pattern in a way that is optimized by selection. This second interpretation is the basic assumption of our model, which is essentially prospective. We discuss the predictions of this model qualitatively using growth data from the procercoids of pseudophyllid cestodes. Quantitative tests would require detailed application of the trade off to the life cycle selected; such investigations are currently in progress for *Schistocephalus solidus*.

Application of standard life history strategy theory to parasite life cycles is in its infancy (Poulin, 1996; 1998). Recently, Gemmill *et al.* (1999) have investigated the optimal switch from growth to reproduction for nematode parasites, showing that observations in nematodes are generally consistent with theoretical predictions. Although their analysis relates to optimal age of maturity of a single adult parasite in its definitive host, we consider growth strategies of larval parasites competing in an intermediate host.

A prospective model for larval parasite growth strategy

Larval parasite growth (mass or volume) eventually levels off towards an asymptotic maximum (e.g. Pool, 1985) with time or age (Fig. 1a). We assume that the expected future reproductive gains, G, of a given parasite, at the stage it becomes infective, increase monotonically with maximum volume $v_{\max}$ (Fig. 1b). In most parasites, the fecundity of females often increases approximately linearly (or sometimes more than linearly; Schärer *et al.*, 2001) with body mass, but the fitness value of greater larval size is not easy to predict and may differ widely depending on the species. Although G is likely to increase with $v_{\max}$, the change in slope of the relation could be either increasing or decreasing. If the larval size is not much smaller than the adult size, $G(v_{\max})$ probably increases with decreasing returns, but if the larva is typically much smaller than the adult, $G(v_{\max})$ may be more likely to show increasing returns.

Increased total mass of larval parasites within a given host is likely to reduce that host's survival (e.g. Anderson & Gordon, 1982; Rousset *et al.*, 1996). For simplicity, we assume that if there is multiple infection, this occurs at approximately the same time so that the larvae are roughly synchronized in their development. Such synchrony is frequently attained in helminth life cycles (see Discussion). Let N represent the level of parasite intensity (the number of individuals of a particular parasite species in each infected host; Bush *et al.*, 1997). The total parasite mass at intensity N, assuming all parasites play the same growth strategy, is $V = Nv_{\max}$. Of interest for our model is the way in which V affects the parasite's probability, P, of being transferred successfully between its present host and its next host in its life cycle. A unit increase in mass by a focal parasite results in a unit reduction in host

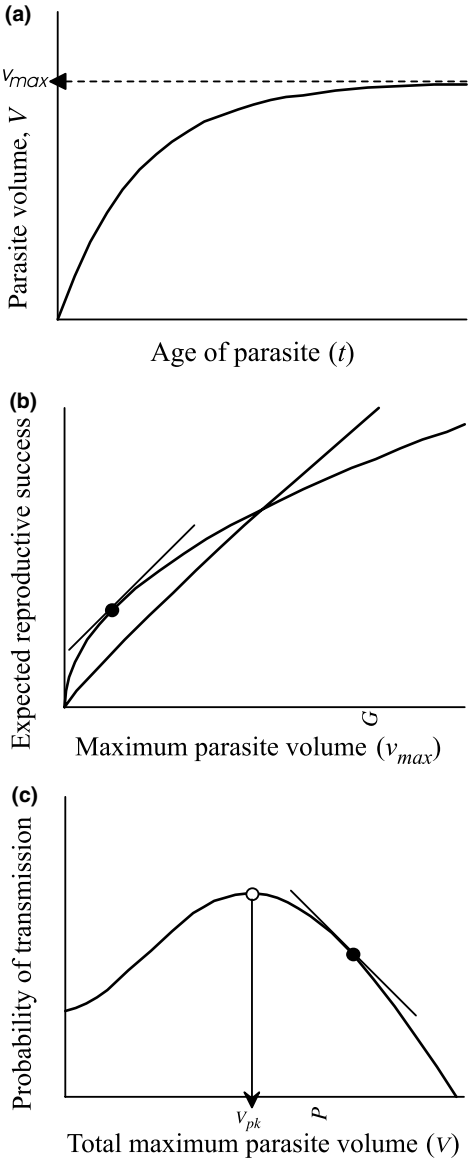

Fig. 1 (a) Form of relationship between parasite volume and age of larva (from time t) following infection. The maximum (asymptotic) size is $v_{\max}$. The curve shown follows $v = v_{\max}(1 - e^{-ct})$, where c is a positive constant determining the rate of growth. (b) Relation between expected future reproductive gains, G, and the maximum volume of the larval parasite, $v_{\max}$, following equation (3a). The line is for power $x = 1$, and the curve is for $x = 0.5$. The gradient of this relation is $G'(v_{\max})$, which is shown for one value of $v_{\max}$ as the tangent to $G(v_{\max})$ at the filled circle on the curve for $x = 0.5$. For the line ($x = 1$), the gradient is constant. (c) Relation envisaged between probability, P, of transmission to the next host and the total maximum parasite volume, $V = Nv_{\max}$. P may first increase because small parasite burdens increase the vulnerability of the current host to predation by the next host, then reach a peak at $V = V_{pk}$, after which P declines. However, the shape of $P(V)$ before V_{pk}, is not important because the ESS growth strategy occurs at a value for V greater than V_{pk}, and is defined in terms of the gradient $P'(V)$, shown by the tangent to $P(V)$ at the filled circle.

survival that is felt equally by all N parasites in that host, i.e. it is a 'summed cost' *sensu* Godfray & Parker (1992). A plausible possibility is that P first increases with V, because small parasite loadings increase the vulnerability of the present host to the predator that acts as the next host. Also, larval parasites induce behavioural changes in their hosts to enhance transmission probability (e.g. Poulin, 1995). However, with very large parasite loadings, the effect will be increasingly to kill the host before it can be exposed to the predator, i.e. to increase 'non-beneficial' host death from the parasite perspective (Fig. 1c).

We seek an optimal asymptotic or maximum volume, $v^*_{\max}$, for the parasite. When more than one parasite occupies the same host, $v^*_{\max}$ will be an evolutionarily stable strategy (ESS; Maynard Smith, 1982) because an individual's fitness will depend on the growth strategies of conspecific competitors in the same host.

Provided that competing parasites are unrelated, we will assume that the fitness of a given juvenile parasite, W, is the product of the probability that it will survive to the next host and its expected fitness should it survive. The expected fitness of a mutant playing $v_{\max} \neq v^*_{\max}$ is, therefore,

$$W(v_{\max}, v^*_{\max}) = P(V) \cdot G(v_{\max}), \tag{1a}$$

where the total parasite biomass is $V = [(N-1)v^*_{\max} + v_{\max}]$. As competing parasites may have an average coefficient of relatedness, r, greater than zero, we can derive an inclusive fitness version of (1a) as

$$W(v_{\max}, v^*_{\max}) = P(V) \cdot G(v_{\max}) + P(V) \cdot G(v^*_{\max}) r(N-1) \tag{1b}$$

The first term on the right hand side of (1b) is self's (the focal mutant's) fitness and the second term adds the fitness of the $(N-1)$ competing relatives scaled by r, the probability that each of these competing parasites share the same mutant allele as the focal parasite. This inclusive fitness or 'marginal Hamilton's Rule' approach has for previous cases been found to give the same ESS solutions as one based on genetics (Godfray & Parker, 1992; Mock & Parker, 1997; Parker, 2000) and we have obtained the same solution for the case of $N = 2$ for the present model.

We find the ESS by the usual procedure (e.g. Maynard Smith, 1982) of setting

$$\frac{\partial W(v_{\max}, v^*_{\max})}{\partial v_{\max}} = 0; \quad \text{subject to } \frac{\partial W(v_{\max}, v^*_{\max})^2}{\partial^2 v_{\max}} < 0$$

for $v^*_{\max}$ to be a maximum. This gives

$$\frac{G'(v_{\max})}{G(v^*_{\max})} = -\frac{P'(V^*)}{P(V^*)}[1 + r(N-1)] \tag{2}$$

Equation (2) states that at the ESS, the gradient of G scaled by its absolute value, must equal minus the gradient of P scaled by its absolute value, multiplied by the inclusive fitness parameter, $[1 + r(N-1)]$, which is equal to 1, if parasites are unrelated ($r = 0$). As $G(v_{\max})$ is always expected to be positive, this means that at the ESS the total mass of parasite in the host must be such that it causes 'non-beneficial' mortality of the host, i.e. the region of $P(V)$ where its gradient is negative. The benefits of increased mass to self must be offset by decreased transmission probability of self plus relatives to the next host.

As $V^* = Nv^*_{\max}$, each parasite will grow to a maximum of $v^*_{\max} = V^*/N$ under competitive conditions. What does this imply for growth under competition? To gain further insights we resort to explicit functions for $G(v_{\max})$ and $P(V)$. Specifically, we assume that these are simple power relations:

$$G(v_{\max}) = a v^x_{\max} \tag{3a}$$

$$P(V) = P_{pk}(1 - b(V - V_{pk})^y \tag{3b}$$

where V_{pk} is the parasite volume that gives the maximum probability of transmission, P_{pk}. As we require that $P'(V_{pk})=0$, and $P(V)$ is likely to decline to zero with increasing gradient, $y > 1.0$. Equation (3b) serves as an approximation to the part of the curve (Fig. 1c) that lies beyond the peak, as we know that an ESS must occur at a V higher than that which gives a transmission probability, P_{pk}. In order to change the shape of $P(V)$ without altering the value V_0 at which no transmission occurs [where $P(V) = 0$], we set $b = 1/V_0^y$. This gives a family of curves obtained from different values of y as shown in Fig. 2a.

From (3a), (3b) and (1b), remembering that $V^* = Nv^*_{\max}$, we obtain

$$\frac{x}{v^*_{\max}} = \frac{by(Nv^*_{\max} - V_{pk})^{y-1}}{1 - b(Nv^*_{\max} - V_{pk})^y[1 + r(N-1)]} \tag{4a}$$

For present purposes, let $V^* \gg V_{pk}$. Substituting $b = 1/V_0^y = 1/(N^y v_0^y)$, we can obtain for this case

$$\frac{v^*_{\max}}{v_0} \approx \left(\frac{Nx}{Nx + [1 + r(N-1)]y}\right)^{\frac{1}{y}} \tag{4b}$$

From (4) it is clear that y is critical in determining the way that $v^*_{\max}$ changes with the number of competitors, N.

Equation (4b) therefore gives the ESS asymptotic volume, $v^*_{\max}$, expressed as a proportion of the individual parasite volume, v_0 which results in zero transmission [$(P(V) = 0)$] if shown by all N competitors. Note that $V^*/V_0 = Nv^*_{\max}/Nv_0 = v^*_{\max}/v_0$. This means that equation (4b) also tells us the ESS total parasite biomass expressed as a proportion of the biomass that results in zero transmission. We can call the term $v^*_{\max}/v_0$ $(= V^*/V_0)$ the 'host exploitation' parameter.

As $v^*_{\max}/v_0$ increases, the probability of transmission for each of the N competitors decreases and when $v^*_{\max}/v_0$ rises to 1.0 – none of the parasites survives to reach the next stage. However, it is clear that (4b) has an asymptotic of 1.0, so that in the present model, if we assume that parasites can detect each other's presence

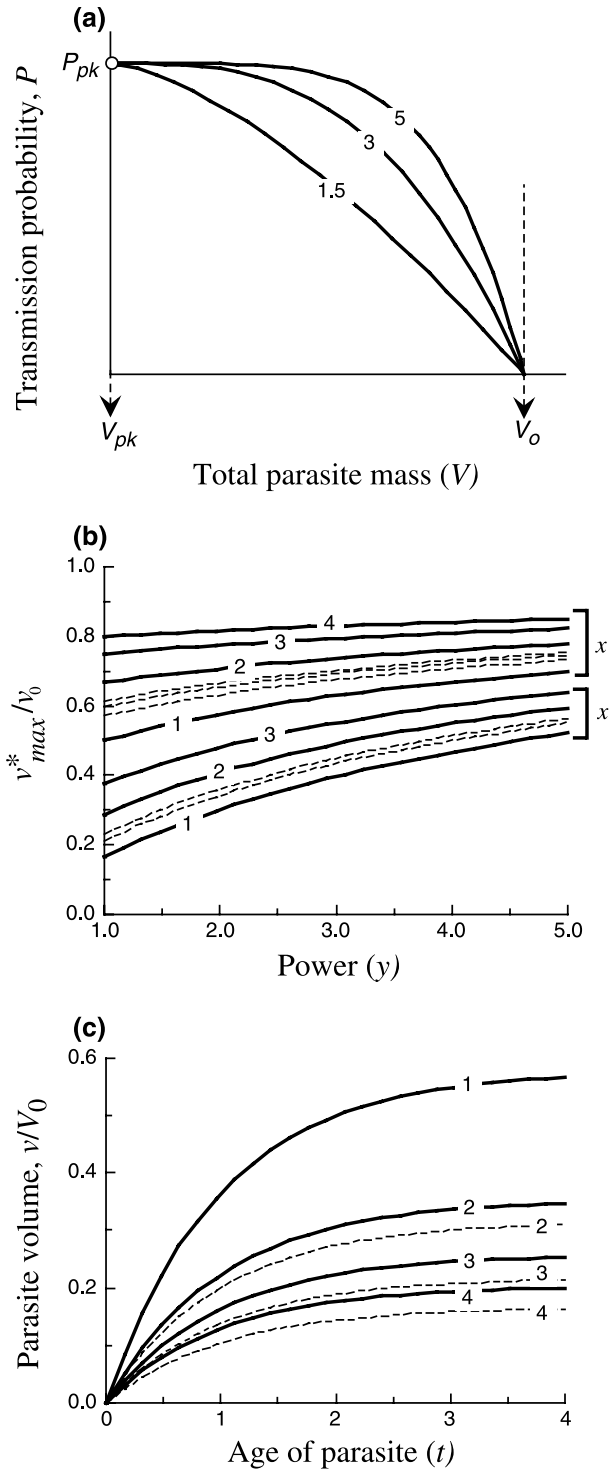

Fig. 2 (a) Form for $P(V)$ used to generate the results in (b), following equation (3b) with $P_{pk} = 1.0$, $V_{pk} = 0$. The three curves are for the three values of y (1.5, 3, 5), which are constrained to have the same intercept on V at V_0 by setting $b = 1/V_0^y$ (see text). (b) Relation between host exploitation index, v_{max}/v_0 the maximum size scaled by the size that would cause death of the host, and power y as explained in (a), calculated from equation (4b). The sets of curves are for two values of the power x (1.0, 0.2) used in the gain function, equation (3a). The continuous curves are for single infections, or multiple infections (intensities shown on curves) where competitors are unrelated ($r = 0$), and the broken curves for cases where competitors are full sibs ($r = 0.5$). The broken curves are for the same intensities as the continuous curves at a given x, and have the same qualitative ordering as the continuous curves but lie much closer together across the set of intensities shown. (c) Growth trajectories expected under growth as in Fig. 1a with $c = 1.0$, calculated for v_{max}/v_0 as in equation (4b), with $x = 1.0$ and $y = 2.0$. Continuous curves are for unrelated ($r = 0$) competitors (intensities shown on curves), and the broken curves for cases where competitors are full sibs ($r = 0.5$).

and modify their growth strategy accordingly, they should never kill the host by overexploitation.

We show some results of (4b) in Fig. 2b. The main findings are:

1 The degree of host exploitation, v^*_{max}/v_0 increases as the number of competing parasites, N, increases. As a result of scramble competition for the limited host resources (Nicholson, 1954), increased competition causes increased risks of transmission failure because of greater host exploitation.

2 Increasing the power x from 0 to 1.0 reduces the initial rate of increase in G with v^*_{max} (Fig. 1b); this increases host exploitation.

3 The effect of increasing power y, which causes $P(V)$ to become more bowed between V_{pk} and V_0 (Fig. 2a), is to increase the degree of host exploitation.

4 The effect of competition between kin is to reduce the host exploitation; for the case of full sibs, this reduction is very marked. If the competing larvae are genetically identical, $r = 1.0$ and N cancels from the right hand side of (4b). This means that the same parasite index holds at all N, so that asymptotic larval size is $1/N$ of the v^*_{max} at $N = 1$.

Finally, we can see how the result in (4b) might translate into growth trajectories. Fig. 2c shows how larvae may be seen to grow under the assumption of growth with exponentially diminishing returns as in Fig. 1a (the case given is where $x = 1.0$, $y = 2$). We express the asymptotic mass of an individual parasite as a proportion of the total mass that will cause zero transmission, V_0. This index, v^*_{max}/V_0 declines with the number of competitors, N. As N increases, the decline in v^*_{max}/V_0 becomes weaker: there is more difference between v^*_{max}/V_0 for $N = 1$ and 2, than between $N = 2$ and 3, etc.

Host exploitation is constant if Nv^*_{max}/V_0 remains constant, i.e. if v^*_{max} is proportional to $1/N$ at all N. Our results predict that v^*_{max} should never decline as steeply as this unless competing larvae are identical. Thus the relative total volume of parasites in a host will increase as intensity increases. However, if y is high [steeply bowed

$P(V)$ function] and x also high (escalating fitness returns with increasing size), the host exploitation index is pushed close towards V_0 for all N, so that the decline in v^*_{max} may approximate towards $1/N$, with the relative total volume of parasites remaining constant with intensity.

In summary, we expect the asymptotic size of individual larvae to decrease, but the total volume of parasites to increase, as the number of competing larvae increases in a host. Both effects get weaker as larval intensity increases. Kin selection can greatly dampen increases in total parasite volume.

Discussion

A central question concerns whether body size of competing parasites is an evolved life history strategy (LHS) as we propose here, or is simply a direct reflection of resource constraints (RC) on growth because of the fixed limitations imposed by the host. Under the LHS hypothesis, the major determinant of parasite size is the trade-off between the benefits of being larger, and the costs this imposes on transmission through adverse effects on host survival. Under the RC hypothesis, the limited host resources directly constrain the capacity for parasite growth.

The simplest and most plausible hypothesis for RC is that the relative total volume of parasites remains constant whatever the intensity of infection, so that when there are N larvae, they each have $1/N$th the final size. We acknowledge that there may be deviations from this prediction depending on the exact nature of the host–parasite relationship. Only under extreme circumstances (high relatedness, high values of x and y) can so extreme an effect of intensity as $1/N$th the final size be seen under the LHS model. Thus in both cases, the volume of the parasite should decrease as intensity increases, but under LHS the total parasite volume should increase, whereas under RC it should remain the same. To test between the two hypotheses may be possible experimentally if the stimulus moderating growth under LHS is the presence of other worms.

We examined data on pseudophyllid cestodes in their principal first intermediate hosts. There have been numerous investigations of the procercoid stages following Janicki & Rosen's (1918) and Rosen's (1919, 1920) early life cycle studies of *Diphyllobothrium latum* (as *Dibothriocephalus latus*), *Triaenophorus nodulosus* and *Ligula intestinalis* (as *L. simplicissima*), e.g. Dubinina (1980) for Ligulidae, Vogt (1938), Miller (1943) and Kuperman (1981) for *T. crassus* and *T. nodulosus* and von Bonsdorff (1977) for *D. latum*. These sources give important biological information, but do not provide data to compare with our predictions.

By contrast, Guttowa (1961), Halvorsen (1966), Rosen & Dick (1967) and Pool (1985) have examined experimentally the effect of increased intensity of infection on growth, giving size as lengths of procercoids. To conform with our model and for comparison to unpublished data (M. Michaud) we assumed maximum width to be proportional to length and converted to volumes using an ellipsoid approximation. Table 1 shows the asymptotic volumes of individual procercoids at intensities $N = 1$, 2 and 3, divided by the asymptotic volume at $N = 1$. The scaling relative to size at $N = 1$ gives a dimensionless index for comparison with the model's predictions. Table 1 also includes the scaled total volume of parasites for each intensity level. We do not consider these data to be an empirical test of the model, but simply assess whether they conform to our predictions. All data conform to decreasing larval size with increasing intensity, and *S. solidus* and *T. crassus* approach the $1/N$ reduction that leads to a constant total parasite volume. The decline in parasite size is shown in Fig. 3 for *Bothriocephalus acheilognathi* and can be seen to be less than $1/N$.

The evidence from Table 1 and Fig. 3 in favour of LHS or RC is equivocal. The data appear to divide into two groups. The first quite closely approaches constancy of relative total volume of parasites (*T. nodulosus* and *S. solidus*), and could be seen as potential candidates for RC. The second (*Bothriocephalus acheilognathi, D. dendriticum* and *D. latum*) shows a much less steep decrease in

Table 1 Relative procercoid volumes at different intensities of infection of copepods.

	Intensity							
	1		2		3			
Species	V	TV	V	TV	V	TV	H	Source
Bothriocephalus acheilognathi	1	1.00	0.85	1.70	0.71	2.13	2	Pool, 1985
*Diphyllobothrium dendriticum**	1	1.00	0.94	1.88	0.78	2.34	3	Halvorsen, 1966
Diphyllobothrium latum	1	1.00	0.91	1.82	0.68	2.04	3	Guttowa, 1961
Schistocephalus solidus	1	1.00	0.55	1.10	0.37	1.11	3	Michaud, unpublished data
Triaenophorus crassus	1	1.00	0.50	1.00	0.37	1.11	3	Rosen & Dick, 1967

*Halvorsen (1966) studied *D. dendriticum* as *D. norvegicum*. V = asymptotic volume (scaled by the volume at an intensity of 1) of individual infective procercoids in the adult natural first intermediate host (copepods) at intensities of $N = 1$, 2 and 3 parasites. TV = total volume of all parasites in the host (scaled by the volume at an intensity of 1). H = number of hosts in the life cycle.

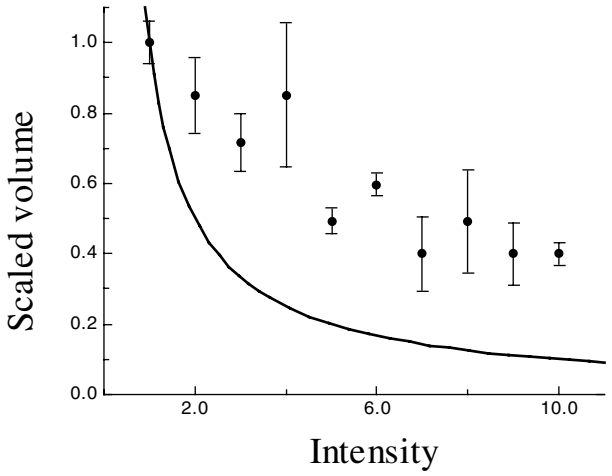

Fig. 3 *Bothriocephalus acheilognathi* procercoid volume (scaled by the volume at an intensity of 1) in relation to intensity of infection (filled circles ± SE). A decrease in volume of $1/N$ is shown by the continuous curve. Data taken from Pool (1985).

body size with intensity (see Fig. 3 for *B. acheilognathi*), and hence clear increases in total parasite volume, which could possibly be taken as circumstantial evidence for LHS. Although the data are insufficient to draw any clear conclusions, it seems unlikely that this difference relates to the number of hosts in the life cycle (Table 1). It is also possible that group 1 are the result of LHS under relatively high values for x and y, coupled with some degree of kin selection. Michaud used coracidia from three worms that may have mated together in her experiments, with $r > 0$.

Dubinina (1980) conducted many experimental infections in Ligulidae (i.e. *Ligula, Digramma* and *Schistocephalus* species) and found usually one to three procercoids in a single copepod (maximum 10–17). In experiments up to 50–60 coracidia could be ingested by the principal host, which showed a protective reaction by forming a fibrous shell around the many oncospheres penetrating through the wall of the stomach. Intense infestation did not result in host death, but the larvae that developed caused the crustacean to become less mobile, to be unable to hold itself at the proper level in the water, to sink to the bottom and to fall prey to other, less infected, predatory *Cyclops*. Procercoids at high intensities of infection were typically significantly smaller than those at intensities of 1 or 2. Michajlow (1953) believed that the size of the *T. nodulosus* was primarily constrained by both the limited capability of the host (copepod) to provide them with food, and the limited volume of its body cavity.

We note, however, that while high intensities of procercoids can be readily induced in experimental infections, in nature there are very low prevalences in copepods. Jarecka (1958) examined 48 691 planktonic crustaceans from Druzno Lake, Poland, finding only 228 larvae of a range of helminth species. An argument in favour of the RC hypothesis is that multiple infection may be too rare in nature to constitute a significant selective force shaping LHS adaptation.

The present model must be regarded as prospective. It examines final size in larval parasites when multiple infections tend to occur approximately simultaneously. The validity of this assumption is clearly questionable. However, there are good reasons why synchronous infection may often occur in nature: (i) the host may be vulnerable to infection only at a limited phase of its life (e.g. Smyth, 1963, 1969); (ii) after a first infection, the host develops an acquired immunity (e.g. Vignali *et al*., 1989); (iii) host behaviour such as migration (e.g. Dogiel, 1970) or reproduction (e.g. Dogiel, 1970); (iv) seasonal factors (Chubb, 1980, 1982) and (v) patchy distribution of infectious stages in the habitat (e.g. Jarecka, 1958). Patchy distribution also permits the possibility of ingestion of related larvae (e.g. Vik, 1958).

Where multiple infections occur with high asynchrony, the conclusions derived from our model would be inaccurate. Asynchrony would result in a higher optimal v^*_{max} for the first larva, with the v^*_{max} of each subsequent larva showing a reduction. However, our conclusions would probably in general hold qualitatively if v^*_{max} is taken as the average asymptotic size of the N competing larvae, although this remains to be proven analytically.

The present paper examines only the final size of a parasite larva. A second aspect of growth strategy, unexplored in the present paper, concerns the instantaneous rate of growth, which can be affected by competition (e.g. Royle *et al*., 1999). If parasites can detect the presence of competitors in the same host, selection is likely to favour an increased growth rate (above that which would be optimal for a single parasite), and increasingly so with the intensity of competition, allowing a greater amount of the available host resource to be sequestered at the expense of competitors. This aspect of growth strategy for parasites is currently under investigation.

Our LHS solution that unrelated parasites competing in the same host should overexploit (but not kill) the host is exactly comparable with a problem much analysed and studied in the social and political sciences and in evolutionary game theory called either 'the tragedy of the commons' (e.g. Hardin, 1968; 1998) or the 'public goods problem' (e.g. Ostrom, 1999). The tragedy of the commons describes the problem of sustaining a public resource when everybody is free to overuse it. Public goods experiments, a paradigm for studying this kind of problem, usually confirm that a collective benefit will not be produced. Competing parasites represent an animal example of a public goods problem. If we assume that parasites can detect each other's presence and modify their growth strategy accordingly, our analysis suggests they should never kill the host by overexploitation. This is a non-cooperative solution that appears superficially cooperative.

In principle, the same sort of approach and analysis should be applicable to growth strategies of competing adult parasites, provided that their present reproduction (rather than future, as in larvae) can be constrained by overexploitation of their host. There appear to be many instances where multiple parasite infection results in growth reduction of adult parasites (e.g. Williams & Halvorsen, 1971).

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