Research

Population dynamic consequences of parasitised-larval competition in stage-structured host–parasitoid systems

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Parasitism can influence many aspects of the host's behaviour and physiology, which in turn can have a profound impact on their population and evolutionary ecology. In many host–parasite interactions there is often a time lag between infection and the death of the host, yet little is known, experimentally or theoretically, about the effects that intra-class competition between parasitised and unparasitised hosts have on the host–parasite population dynamics.

In this article we address this gap in our understanding using a stage-structured mathematical model for a host–parasitoid interaction, which has been parameterised for the *Plodia–Venturia* experimental system.

In the case where parasitised larvae do not compete and do not cannibalise unparasitised larvae, our model predicts a wide range of host–parasitoid dynamics, ranging from host–parasitoid generation cycles, to host generation cycles with parasitoid half-generation cycles, to host–parasitoid equilibria, to host generation cycles with parasitoid extinction.

However, when parasitised larvae can compete with their unparasitised larvae counterparts, the host–parasitoid population dynamics can dramatically change. In particular, we show that high levels of competition exerted by unparasitised larvae upon parasitised larvae is more likely to lead to parasitoid extinction.

In addition, we demonstrate that unparasitised host larvae that are sufficiently susceptible to intra-class competition, or parasitised host larvae that are sufficiently strong competitors, can have a stabilising effect on the host–parasitoid population dynamics.

The implications of these theoretical results are discussed in light of our understanding of host–parasitoid interactions and host–parasite systems in general.

Host–parasite interactions are pervasive throughout the natural world, forming a critical component of nearly all plant and animal community assemblages. Many parasites play an important role in host ecology, and in most cases, the parasite has a deleterious affect on the host's population growth rate. Hence, numerous parasite species have been used successfully as biological control agents (biocontrol) for a wide range of target host species.

In particular, in the combat against insect pests, parasitoids have been proven to be highly successful in controlling their hosts' numbers (Van Driesche and Bellows Jr. 1996). Parasitoids are an extremely large and diverse group, and are believed to exert a profound impact on their hosts' population and evolutionary ecology (Godfray 1994). The ways in which parasitoids

assert themselves are often varied, ranging from the timing at which a parasitoid attacks, leading to important age-dependent effects of parasitism (Hassell 2000), to the physiological and behavioural effects of parasitism (Gross 1993). These effects potentially have significant consequences for the host.

From the perspective of the host, there are two distinct types of parasitoid; idiobiont parasitoids which paralyse and prevent further growth of the host, and koinobiont parasitoids, which allow the host to continue growing and feeding until an apparent optimal time, whereupon it is consumed (Askew and Shaw 1991). In both cases the host insect is usually killed.

One feature that is common to nearly all parasite interactions is the time delay before death that follows

infection, resulting in a mixed population of uninfected and infected hosts (Sisterson and Averill 2003, Cameron et al. 2005). In the case of idiobiont parasitoids, the unparasitised hosts may be able to cannibalise the paralysed parasitised hosts. In the case of koinobiont parasitoid species, the parasitised host may still be capable of competing with its unparasitised counterparts, a dynamical interaction that closely resembles many host−pathogen systems, including viruses, bacteria and fungi (Fuxa and Tanada 1987). In the case of microparasites, speed of kill is a key component of the infection probability and has led to the use of transgenic techniques to reduce the lag between infection and death (Hails et al. 2002). However, these phenomena have been largely ignored, experimentally and theoretically, and very little is known about the intra-class competition exerted by the parasitised larvae on the unparasitised larvae (and vice versa) and the role it plays in host−parasitoid population dynamics.

Intuitively we might expect that parasitised hosts exert a weaker competitive effect on healthy, unparasitised hosts. However, in a recent study, Cameron et al. (2005) found the opposite effect in a laboratory host population of Indian meal moths, *Plodia interpunctella* (Hübner) (Lepidoptera: Pyralidae) (hereafter *Plodia*), parasitised by a solitary endoparasitic koinobiont wasp, *Venturia canescens* (Gravenhorst) (Hymenoptera: Ichneumonidae) (hereafter *Venturia*). The putative mechanism, in this case, is that parasitism reduces the resource requirements of the parasitised hosts, allowing them to survive periods of strong resource competition at the expense of healthy host survival. Recent work in other systems has begun to demonstrate the importance of infected competitors in enemy−victim dynamics and in the structuring of ecological communities. For example, in lepidopteran pest communities, Bernstein et al. (2002) found that parasitised hosts (*Ephestia kuehniella* (Lepidoptera: Pyralidae) parasitised by *Venturia*) were less susceptible to competition than their healthy counterparts. In bacterial communities Kusch et al. (2002) found that infected bacterial strains out-competed the uninfected strains. These studies, and others (Prévost 1985, Wajnberg et al. 1985), suggest that intra-class host competition between infected and uninfected hosts are common place. However, few studies have focused on how competition between infected and healthy individuals may influence the dynamics between interacting host and parasite populations.

The mechanisms of stabilisation in host−parasite systems have created a great deal of interest in both the ecological and mathematical modelling communities (Beddington et al. 1978, May et al. 1981, Bernstein et al. 2002). However, little attention has been paid to intra-class competition between infected and uninfected hosts as a factor promoting stability.

Historically, host−parasitoid systems often use a discrete-time framework (Bailey et al. 1962, Hassell 1978), which assumes that the host and parasitoid have discrete, non-overlapping generations, and in the simplest case assume that the parasitoid's generation is in perfect synchrony with its host's. However, for many systems (*Plodia-Venturia,* for example) the populations have continuous, overlapping generations, and therefore to model the dynamics realistically one must use a continuous-time framework. Moreover, both the host and the parasitoid have distinct life-stages (e.g. egg, larva, pupa and adult) of differing length, therefore it is also appropriate to adopt the stage-structured modelling approach that was pioneered by Gurney et al. (1983).

Since Gurney et al.'s (1983) work, there has been a great deal of modelling using the stage-structured approach (Murdoch et al. 2003). As we have mentioned above, many parasitoid species do not begin development within the host until the host has reached some stage or size in the hosts' own development. 'Delayed development' occurs in many endoparasitoids (Askew and Shaw 1991), including our *Plodia-Venturia* system. This phenomenon was explored mathematically in Gordon et al. (1991), however, the authors found that there was little dynamical difference to a model where delayed development did not occur.

In most models it is assumed that once the host has been parasitised it is functionally dead until the parasitoid offspring emerges (Murdoch et al. 1987, Godfray and Hassell 1989, Rohani et al. 1994). Moreover, due to the nature of many host−parasitoid systems, the host population consists of a mixture of both parasitised and unparasitised larvae which have dynamical impacts upon one another. Hence, to fully understand the population dynamics, it is necessary to model both the unparasitised and parasitised larval classes explicitly. This differs from previous models that have considered the effect of intra-class competition within classes of unparasitised and parasitised larvae rather than between classes. For example, Spataro and Bernstein (2004) derive a model for intra-class host larval competition between parasitised and unparasitised larvae, where it is assumed that parasitised and unparasitised larvae do not mix within the population. This assumption is unrealistic for many host−parasite systems.

In this paper, we address this gap in our understanding of host−parasitoid dynamics and theory, and explore the effects of parasitised larval competition using a stage-structured mathematical model with parameters estimated from the *Plodia−Venturia* system. Parasitism of the larval stages of *Plodia* by *Venturia* is strongly age-dependent (Harvey et al. 1994), though wasps prefer to attack the larger stages (Sait et al. 1997). However, regardless of which instar is attacked, *Venturia* can only complete its own development

once the host has reached the final instar stage (Harvey et al. 1994). Thus, a variable but potentially considerable fraction of the parasitoid life-cycle is intimately coupled with the competitive ability and survivorship of the host. *Venturia* exhibits a numerical, density-dependent response to changes in host abundance (Begon et al. 1995) and several mathematical studies of this system demonstrate that host−parasitoid dynamics depend on the strength of larval competition and egg-cannibalism, as well as the parasitoid developmental lag, which promote host generation cycles (Bjørnstad et al. 1998, 2001, Briggs et al. 2000, Wearing et al. 2004b). This pattern in host−parasitoid abundance is more robust with the addition of host demographic stochasticity (Wearing et al. 2004a). Thus, the relative impact of parasitised hosts on their unparasitised counterparts is likely to be sensitive to host−parasitoid densities and resource availability.

Mathematical models

We begin by reviewing the model of Godfray and Hassell (1989), and extend this to include unparasitised host larval competition. We regard this model as our base model, to which we compare further extensions and to test hypotheses.

Godfray and Hassell model

The Godfray and Hassell model assumes that the life-cycle of a female host can be divided into four stages: an egg stage, a larval stage when the parasitoid is able to attack, a pupation stage and an adult stage. It is also assumed that the female parasitoid has only two stages: a juvenile stage for which the larvae remains within the host and an adult stage which can attack the host larvae. Moreover, host egg and pupation stages do not have density-dependent interactions, and so these stages are not explicitly modelled. It is assumed that reproduction by both the hosts and parasitoids is limited to a fixed period of time.

For continuity, we denote the density of host larvae, adult hosts and adult parasitoids by $L(t)$, $A(t)$ and $P_A(t)$ time t respectively. Then the Godfray and Hassell model is given by the system of delay differential equations

$$\frac{dL}{dt}(t) = R_L(t) - M_L(t) - D_L(t) \tag{1a}$$

$$\frac{dA}{dt}(t) = R_A(t) - M_A(t) - D_A(t) \tag{1b}$$

$$\frac{dP_A}{dt}(t) = R_{P_A}(t) - M_{P_A}(t) - D_{P_A}(t) \tag{1c}$$

where the right hand side of the differential equation is given in Table 1 and the parameters for which are defined in Table 2.

The authors show that low values of the clumping parameter, k (a measure of density-dependence in the rate of parasitism), both enhance stability and lead to continuous (equilibrium) rather than discrete generations (generation cycles). Moreover, depending on the ratio of host to parasitoid generation times, the

Table 1. The terms in the Godfray and Hassell model (1). All parameters are defined in Table 2.

Description	Expression
Larval host recruitment rate	$R_L(t) = bA(t - \tau_E)\sigma_E$
Egg host time-independent survival probability	$\sigma_E = \exp(-d_E\tau_E)$
Larval host death rate	$D_L(t) = [f(P_A(t)) + d_L] \, L(t)$
Probability of parasitism	$f(P_A(t)) = k \, \ln\left(1 + \dfrac{aP_A(t)}{k}\right)$
Larval host maturation rate	$M_L(t) = R_L(t - \tau_L)S_L(t)\sigma_L$
Larval host time-independent survival probability	$\sigma_L = \exp(-d_L\tau_L)$
Larval host time-dependent survival probability	$S_L(t) = \exp\{-\int_{t-\tau_L}^{t} [f(P_A(x)) + cL(x)]dx\}$
Adult host recruitment rate	$R_A(t) = M_L(t - \tau_P)\sigma_P$
Pupal host time-independent survival probability	$\sigma_P = \exp(-d_P\tau_P)$
Adult host death rate	$D_A(t) = d_A A(t)$
Adult host maturation rate	$M_A(t) = R_A(t - \tau_A)\sigma_A$
Adult host time-independent survival probability	$\sigma_A = \exp(-d_A\tau_A)$
Adult parasitoid recruitment rate	$R_{P_A}(t) = f(P_A(t - \tau_{P_L}))L(t - \tau_{P_L})\sigma_{P_L}$
Adult parasitoid time-independent survival probability	$\sigma_{P_L} = \exp(-d_{P_L}\tau_{P_L})$
Adult parasitoid death rate	$D_{P_A}(t) = d_{P_A}P_A(t)$
Adult parasitoid maturation rate	$M_{P_A}(t) = R_{P_A}(t - \tau_{P_A})\sigma_{P_A}$
Adult parasitoid time-independent survival probability	$\sigma_{P_A} = \exp(-d_{P_A}\tau_{P_A})$

Table 2. Model parameters with their descriptions, values, and sources of estimates of values. Sources: A, Sait et al. (1994); B, Reed (1998); C, Harvey et al. (1994).

Parameter	Description	Value	Source
τ_E	Duration of host egg stage	4.3 days	A
τ_L	Duration of host larval stage	25 days	B
τ_I	Duration of host parasitised larvae stage	20 days	C
τ_P	Duration of host pupal stage	7 days	B
τ_A	Duration of host adult stage	5.5 days	B
τ_{P_A}	Duration of parasitoid adult stage	2 days	C
b_A	Daily host adult fecundity number of eggs)	21 day^{-1} when fixed	A
c	Competition mortality of L by L	Varied	
c_{LL}	Competition mortality of L by L	Varied	
c_{LI}	Competition mortality of L by I	Varied	
c_{II}	Competition mortality of L by I	Varied	
c_{IL}	Competition mortality of I by L	Varied	
k	Interference parameter	0.01 when fixed	
a	Parasitoid attack rate	0.01	
d_E	Background host egg mortality	0.017 day^{-1}	A
d_L	Background host larval mortality	0 day^{-1}	A
d_I	Background host parasitised larvae mortality	0.1 day^{-1}	
d_P	Background host pupal mortality	0 day^{-1}	A
d_A	Background host adult mortality	0.1 day^{-1}	
d_{P_L}	Background parasitoid juvenile mortality	0.1 day^{-1}	
d_{P_A}	Background parasitoid adult mortality	0.1 day^{-1}	

maximal value of k such that the parasitoid stabilises the hosts, is much smaller when the parasitoid generation time is approximately 0.5 or 1.5 times that of the host. This is clearly important in the *Plodia–Venturia* system, where the *Venturia* generation time is approximately half that of *Plodia.* Thus, under the assumptions of the Godfray and Hassell model, one would expect to see generation cycles unless the parasitoid has very low values k. It has also been shown that for hosts with a short reproductive adult stage (as is the case with *Plodia,* since adults do not feed) the host–parasitoid system is likely to exhibit generation cycles. However, hosts with a relatively long adult stage are more likely to exhibit continuous generations (a stable equilibrium).

Other life-history parameters in the Godfray and Hassell model have little affect on the stability regions, and only affect the density levels of the population outbreaks or equilibria.

Parasitised-larval competition model

In the above Godfray and Hassell model it is assumed that the host larvae do not encounter any intra-class competition. However, as discussed above, in many host systems, larvae experience competition for a limited supply of resources. In the following model, we assume, for simplicity, that the host larvae undergo uniform intra-class competition (Gurney et al. 1983, Briggs et al. 2000) and combine this with the Godfray and Hassell model; each larval host can assert the same level of competition upon its competitors, independent of the age of the larval stage. Relaxing this assumption in order to have varying degrees of competition throughout the stage-structure can have a significant effect on the population dynamics (Briggs et al. 2000, Wearing et al. 2004b).

One stringent assumption of the Godfray and Hassell model, and all other stage-structured models, is that once the host larvae have become parasitised they are functionally dead until the adult parasitoids emerge. We aim to test this assumption by explicitly modelling the parasitised larval stage which is affected by density-dependent effects in the form of competition with unparasitised larvae. Ultimately, we want to answer the question 'do competing parasitised larvae alter the host–parasitoid population dynamics?'

In our parasitised-larval competition model, the host population has two distinct stages: larval and adult. However, we assume that the larval stage has two sub-populations: one where the larvae have been parasitised and the other where the larval host is susceptible to parasitism. The parasitoid population has only one stage; adults.

This formulation allows us to explicitly model a mixed population of unparasitised and parasitised hosts. Moreover, since these stages are explicitly modelled, it is possible to include density-dependent effects that act upon both the parasitised and unparasitised hosts (e.g. competition for resources, predation etc).

We denote the density of healthy host larvae, parasitised host larvae, adult hosts and adult parasitoids by $L(t)$, $I(t)$, $A(t)$ and $P_A(t)$ time t respectively.

Our parasitised larval competition model has the same form as the Godfray and Hassell model, except an additional equation is required for the parasitised larvae, given by

$$\frac{dI}{dt}(t) = R_I(t) - M_I(t) - D_I(t) \qquad (2a)$$

The terms on the right hand side of the differential equations are given in Table 1 except for:

- The unparasitised larval death rate, $D_L(t)$, which is given by

$$D_L(t) = [f(P_A(t)) + c_{LL}L(t) + c_{LI}I(t) + d_L]L(t) \qquad (2b)$$

where c_{ij} the coefficient of competition mortality that stage j has on stage i (i, j $\in$ {L,I}). Hence, the time-dependent survival probability becomes

$$S_L(t) = \exp\left\{-\int_{t-\tau_L}^t [f(P_A(x)) + c_{LL}L(x) + c_{LI}I(x) + c_{LI}I(x)]dx\right\} \qquad (2c)$$

- The rate at which larval hosts move into the parasitised class, $R_I(t)$ is instantaneous (i.e. there is no time-lag) and is given by the rate at which they are parasitised,

$$R_I(t) = f(P_A(t))L(t) \qquad (2d)$$

- The rate at which the parasitised larvae die is given by

$$D_I(t) = [c_{II}I(t) + c_{IL}L(t) + d_I]I(t) \qquad (2e)$$

where d_I is the background mortality rate of the parasitised class. Note that we assume that parasitised larval hosts are not parasitised by other parasitoids.

- Parasitised larval hosts mature out of the stage at a rate $M_I(t)$ which depends on the probability of surviving the stage and the time spent in that stage. The maturation rate of the parasitised larval hosts is simply

$$M_I(t) = R_I(t - \tau_I)S_I(t)\sigma_I \qquad (2f)$$

where τ_1 is the duration of the parasitised larval class, $\sigma_I = \exp(-d_I\tau_I)$ is the time-independent survival probability and $S_I(t)$ is the time-dependent survival probability, given by

$$S_I(t) = \exp\left\{-\int_{t-\tau_I}^t [c_{II}I(x) + c_{IL}L(x)]dx\right\} \qquad (2g)$$

- Those larvae that have been parasitised go on to produce one parasitoid. We assume that the time taken from when the parasitoid emerges from the host to the time of sexual maturity is negligible. This is indeed the case in our system, as in many others, since the adult wasp is parthenogenetic and
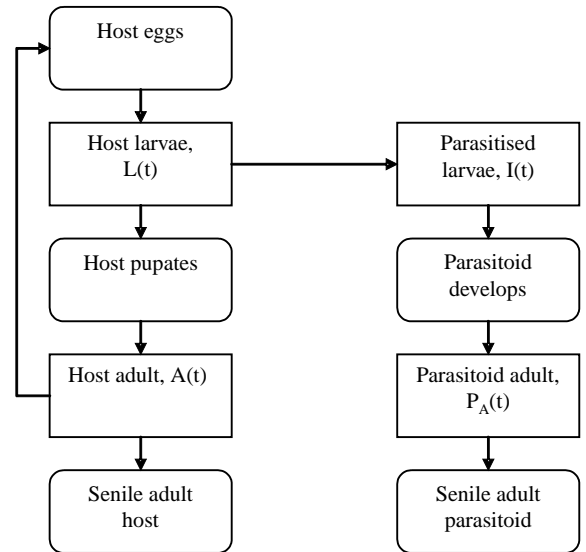

Fig. 1. A schematic interpretation of the life-cycle of the basic host–parasitoid model. Square boxes denote density-dependent stages and rounded boxes denote density-independent stages.

ecloses with mature eggs. Hence the recruitment rate of adult parasitoids, $R_{P_A}(t)$, is simply equal to, the maturation rate of the parasitised hosts, that is,

$$R_{P_A}(t) = M_I(t) \qquad (2h)$$

Equation (2), along with the initial conditions and history (Appendix 1), fully define our parasitised-larval competition model and a schematic interpretation can be found in Fig. 1.

Model predictions

Analytical examination of the above parasitised-larval competition model is difficult, and does not reveal a great deal about the oscillatory dynamics of the systems. We therefore carry out extensive numerical simulations on the model using parameters for the *Plodia–Venturia* system (Table 2). Details of our numerical approach can be found in Appendix 1.

Base model

As our base model we consider the case where there are no density-dependent effects acting upon, or imposed by, the parasitised larvae, so that they are functionally dead until parasitoid emergence. Mathematically, this is equivalent to setting $c_{IL} = c_{II} = c_{LI} = 0$, and $c_{LI} = c > 0$ is the uniform intra-class competition coefficient (Briggs et al. 2000).
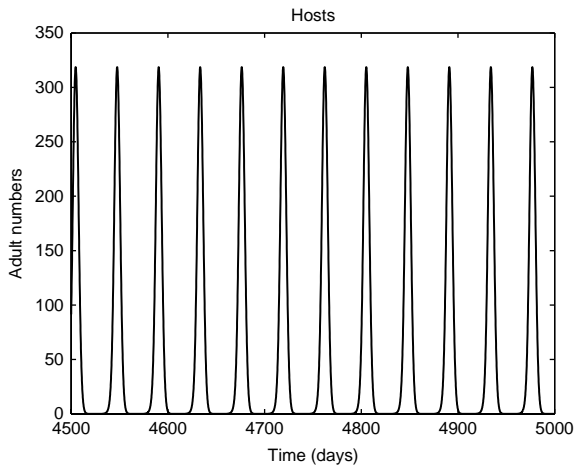
Fig. 2. Host only dynamics predicted from the base model. The parasitoids have not been initialised and all other parameters are given in Table 2 except $c = 7.5 \times 10^{-5}$. We show the adult host population time series after a transient period of 4500 days. From performing the spectral analysis of the time series the period of oscillations is 43.1 days.

In the absence of parasitoids our base model reduces to the uniform larval competition model considered in Briggs et al. (2000). With the parameters given in Table 2, the model predicts generation cycles with a period of approximately 43 days (Fig. 2), which are marginally longer than those observed experimentally (Sait et al. 1994), as found in Briggs et al. (2000). These generation cycles occur due to intense larval competition of the offspring of the adults at the peak, and thus the offspring from the adults that are at the tail experience relatively little competition, and in turn give rise to the next peak in adult numbers. Increased larval competition decreases outbreak population levels, and does not alter the cyclic dynamics.

The effects from the addition of the parasitoids can be seen in Fig. 3 and Fig. 4. In Fig. 3a and 3b, we see that low levels of larval competition lead to generation cycles of both the host and parasitoid. As larval competition increases, as in Fig. 3c and 3d, the parasitoid exhibits half-generation cycles, that is, there are two parasitoid peaks for every single peak in the host population, which exhibits generation cycles. This phenomenon occurs since every adult parasitoid cohort, and their offspring, are able to parasitise the same generation of hosts. Extreme larval competition causes the parasitoids to go extinct and the system returns to the host-alone generation cycle dynamics (Fig. 3e and 3f). This behaviour is summarised in the period diagrams in Fig. 4. The period of the host cycles remain unchanged, no matter what the behaviour of the parasitoid is, only the magnitude of the host peaks are affected by the strength of larval competition and host fecundity.

The interference parameter (a measure of the degree of interference in the distribution of parasitism among host individuals) k, generally tends to stabilise host–parasitoid dynamics (May 1978, Godfray and Hassell 1989), with low values k (less than unity) giving rise to stable equilibria. However, in Fig. 5a and 5b it is clear that the stability region depends on the level of larval competition. For low levels of larval competition the parasitoids are able to stabilise the hosts if their k value lies within a small window. Increasing larval competition leads to an increase in the size of the stabilising window. However, for sufficiently high levels of larval competition host density becomes small such that the rate of parasitism becomes negligible, and the parasitoids have little influence on the host dynamics. Hence, for sufficiently high levels of competition the parasitoids are unable to stabilise the hosts for any value of k. Note that for very small values of k the parasitoids are unable to stabilise the hosts, independent of the level of competition, since small values of k correspond to a low parasitism rate, and therefore the presence of parasitoids has little effect on the host dynamics.

This behaviour from the base models differs from that of the Godfray and Hassell model, where the hosts do not undergo any competition for resources. Firstly, the Godfray and Hassell model can not predict the eradication of the parasitoids, and parasitoids either persist in equilibrium or exhibit generation cycles. Secondly, under intermediate levels of larval competition, the base model predicts that parasitoids exhibit half-generation cycles.

Whilst it is clear that intensive larval competition would lead to the eradication of parasitoids, it is not intuitively clear as to why moderate levels of competition lead to parasitoid half-generation cycles. However, by studying Fig. 6 one can gain more insight into this phenomenon.

We see in Fig. 6a that with low levels of uniform larval competition we observe generation cycles. The explanation for the mechanism behind the generation cycles where no competition occurs has been put forward by Godfray and Hassell (1987, 1989), and the case where low levels of competition occur does not deviate from this explanation. Consider a peak in the host population which will tend to generate a second peak one generation (41.8 days) later. The first peak also provides a large number of potential hosts for parasitism, and hence produces a large number of adults one parasitoid generation (20 days) later. However, since the parasitoid generation length is approximately half that of the host generation length, the parasitoid progeny of the first peak fail to coincide with the progeny of the first peak in host numbers, and accelerate the decline of the host larvae between successive generations. Thus, the parasitoids have 'feasts' and 'famines' which accentuate the naturally
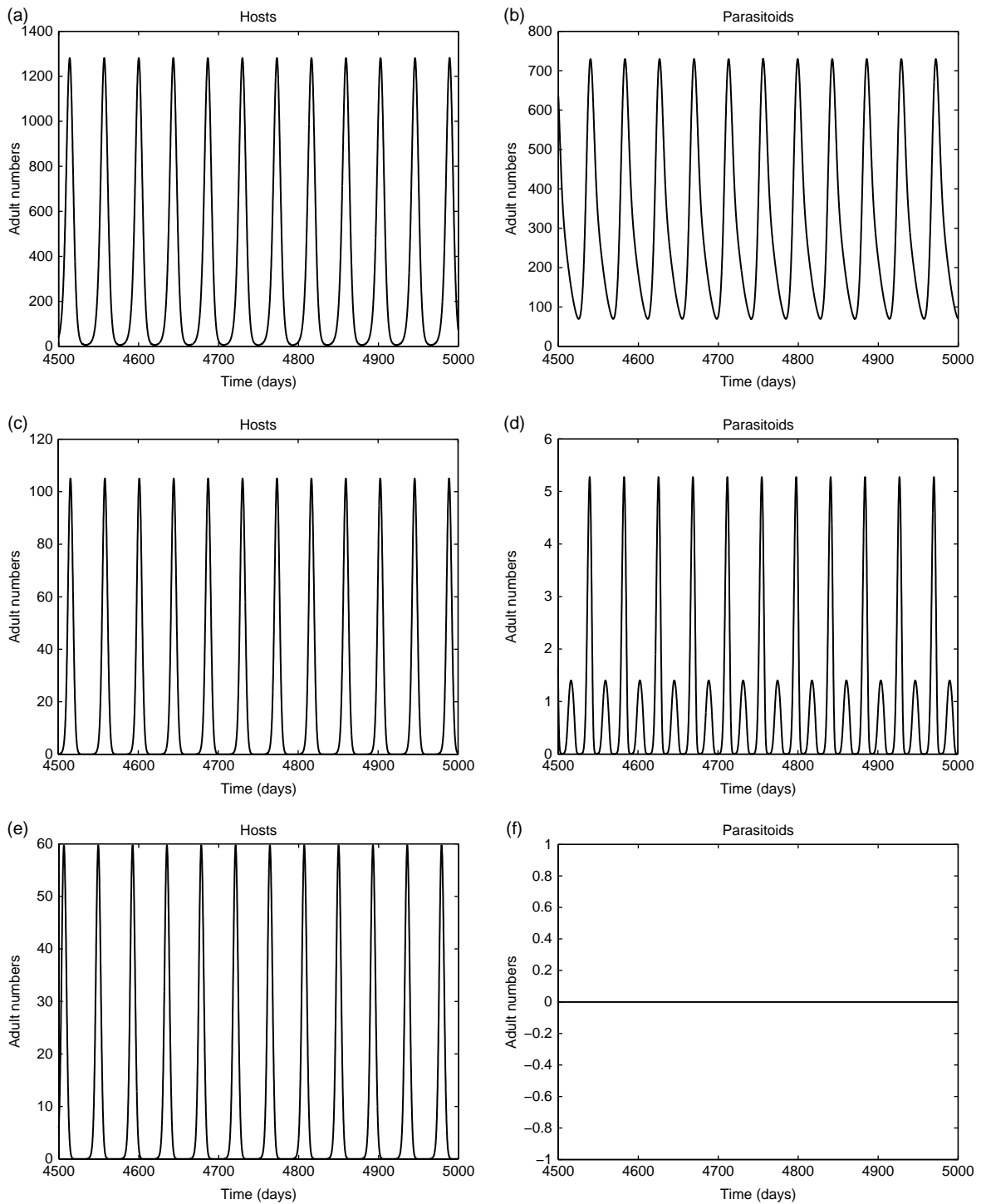
Fig. 3. Typical time series output from the base model as the level of uniform larval competition increases. All parameter values used in the base model are given in Table 2 except $b = 21$ and in (a) and (b) $c = 5 \times 10^{-6}$, in (c) and (d) $c = 2 \times 10^{-4}$ and in (e) and (f) $c = 4 \times 10^{-4}$.
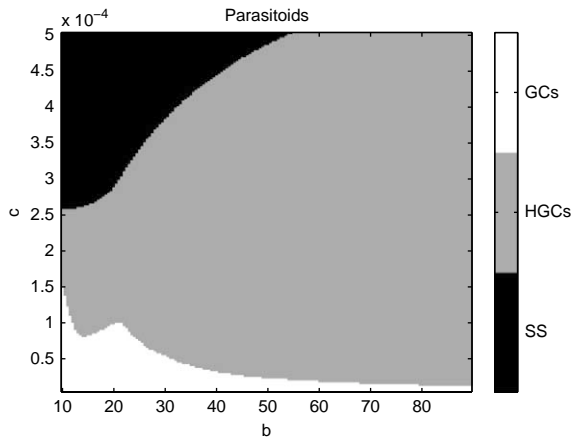
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Fig. 4. Behaviour of the base model. The different shades in the figure show the period of the parasitoid population cycles as the host fecundity b, and larval competition, c, are varied. The host population only exhibit generation cycles for the parameters considered. Key: SS; steady state, HGCs; half-generation cycles, GCs; generation cycles. Regions in parameter space where the hosts exhibit GCs and the parasitoids exhibit SS correspond to parasitoid extinction.

occurring peaks and troughs in host larval (and adult) numbers, giving rise to generation cycles.

In Fig. 6b the competition coefficient has been increased and we observe parasitoid half-generation cycles. This can be explained since the increase in competition leads to a natural decrease in the amplitude of the host larval (and adult) peaks. Thus a peak in the host larvae will still produce a peak in the parasitoids one generation later. However, these progeny do not 'fast' since the decay of the host peak is not as quick as in the case where competition is less (compare the decreasing host slopes in Fig. 6a and 6b). At first this may seem counterintuitive, but the larval competition rate ($-cL^2$) is less, despite having a higher competition coefficient, since the larval numbers are much smaller in Fig. 6b than in 6a, due to the more intensive larval competition. Therefore, this leads to greater number of potential hosts for the parasitoids to parasitise in Fig. 6b than in 6a, and hence there are sufficient larval hosts to produce a secondary (and smaller) parasitoid peak. This secondary parasitoid peak is then able to parasitise the next host peak (progeny from the previous generation). The cycle then continues, thus giving rise to half-generation cycles.

Parasitised-larval competition model

Under the assumption that $c_{IL} = c_{II} = c_{LI} = 0$ the parasitised-larval competition model collapses to the base model, since there is no density-dependent mortality acting upon the parasitised larvae.

Therefore, we begin our analysis of the parasitised-larval competition model by assuming the parasitised larvae do not compete, and that competition exerted by the unparasitised larvae on themselves is equal to that on the parasitised larvae (a scenario that closely resembles many idiobiont host−parasitoid systems), that is, by setting $c_{LL} = c_{IL} = c$, say, and $c_{IL} = c_{LI} = 0$. The extra mortality causes a shift in the stability regions (compare Fig. 4 and Fig. 7), and hence, it is more likely that the parasitoids will die out. This is due to the increased competition asserted by the unparasitised larvae on the parasitised larvae, suppressing potential parasitoid adult numbers, and causing the parasitoid population to go extinct. We support this result in Fig. 5c and 5d where we see that the parasitoids are unable to stabilise the hosts for any value of k in the given competition coefficient range.

Note that the assumption $c_{LL} = c_{IL} = c$ is robust, and that having unequal competition coefficients has little effect (not presented here) on our results. Significant deviation from our findings are only observed when there are large differences between c_{LI} and c_{LL}. With $c_{LL} \gg c_{LI}$, the dynamics closely resemble that of the base model. However, with $c_{LL} \gg c_{LI}$ the parasitoids are more likely to become extinct due to excessive mortality of the parasitised hosts.

In order to fully understand the effects of parasitised-larval competition in our model, where the parasitised larvae compete with the unparasitised larvae, one must vary all four competition coefficients c_{ij} $(i,j \in \{L,I\})$. To do this it is convenient to set three of the parameters as scaled multiples of the fourth by setting $c_{LI} = \chi c_{LL}$, $c_{IL} = c_{LL}/\psi$ and $c_{II} = \chi c_{LL}/\psi$. Thus χ represents the ratio of competitive effects and ψ represents the ratio of competitive sensitivities (Briggs et al. 2000, Wearing et al. 2004b).

By varying χ and ψ, we see in Fig. 8 that if χ and ψ are sufficiently large then the parasitoids are able to stabilise the host population, since in the absence of the parasitoids the hosts exhibit generation cycles. Recall that increasing the ratio of competitive effects χ increases the competition from the parasitised larvae, and increasing the ratio of competitive sensitivities, ψ, decreases the competition on the parasitised larvae. Hence, host stabilisation occurs when the unparasitised hosts are sufficiently susceptible to competition and parasitised hosts are sufficiently strong competitors.

These findings are emphasised in Fig. 5c−h. We see that in Fig. 5c and 5d where parasitised larvae do not compete, the parasitoids are not capable of stabilising tne host dynamics for any value of k in the given competition coefficient range, since competition drives the parasitoids to extinction. In Fig. 5e and 5f, where parasitised and unparasitised larvae are equally competitive, the parasitoids go extinct and are unable to stabilise the host dynamics. However, as the ratio of
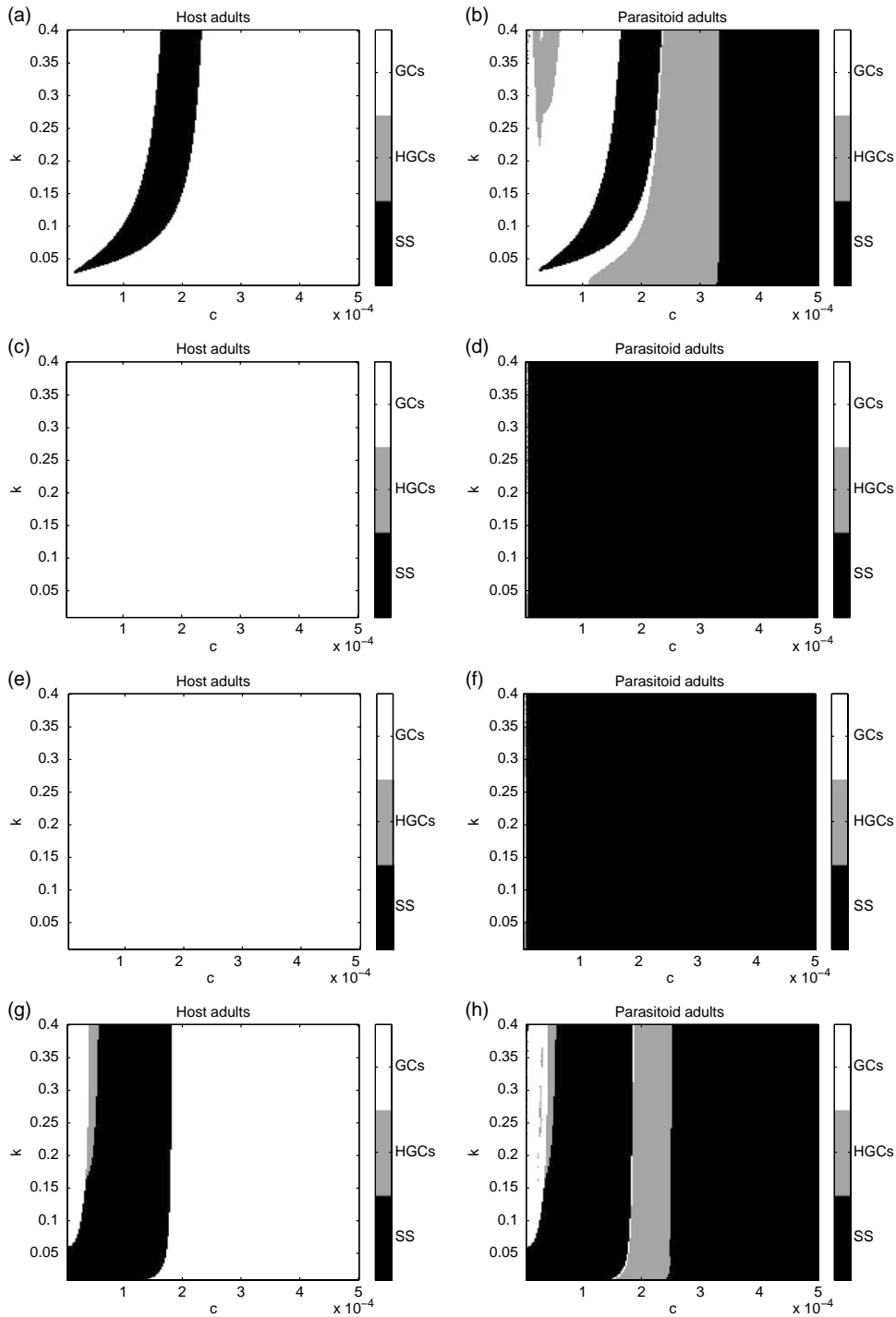
Fig. 5. Stability regions in (c, k)-space. The different shades in the figures show the period of the host and parasitoid population cycles as the host larval competition (c or c_{LL}) and parasitoid clumping (k), are varied in the base model ((a) and (b)) and the parasitised larval competition model ((c) to (h)). The parameter values are given in Table 2 except: in (c) and (d) $c_{LL} = c_{IL}$ and $c_{II} = c_{LI} = 0$; in (e) and (f) $\psi = \chi = 1$; in (g) and (h) $\psi = \chi = 20$. Key: SS; steady state, HGCs; half-generation cycles, GCs; generation cycles. Regions in parameter space where the hosts exhibit GCs and the parasitoids exhibit SS correspond to parasitoid extinction.
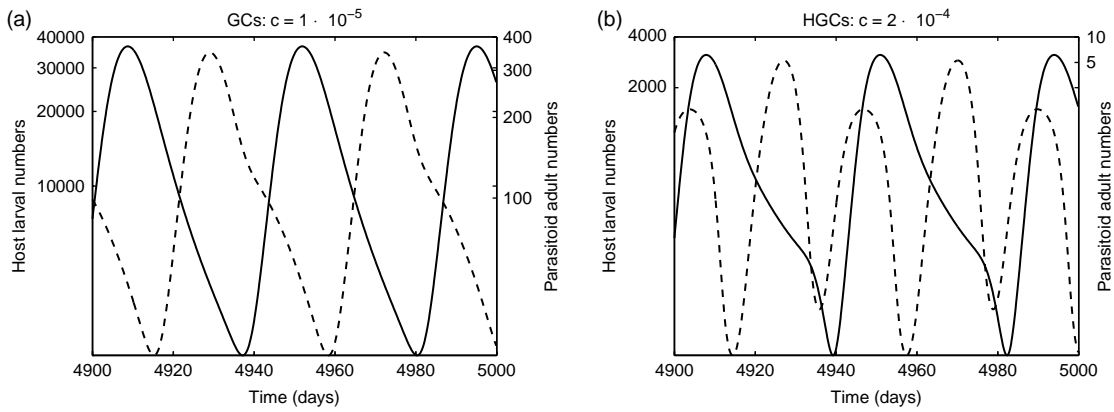
Fig. 6. Typical time-series output from the base model explaining the occurrence of parasitoid half-generation cycles. Solid lines indicate host larval numbers and dashed lines indicate parasitoid adult numbers. In both plots the y-axis has been log transformed. All parameter values used in the base model are given in Table 2 except in (a) $c = 1 \times 10^{-5}$ and in (b) $c = 2 \times 10^{-4}$.

competitive effects (χ) and the ratio of competitive sensitivities (ψ) are increased, a window of stability appears (Fig. 5g and 5h). Moreover, comparing Fig. 5a and 5g, we see that the window of stability has increased in size, allowing parasitoids to stabilise the host dynamics for much lower values of k.

Thus the major question arising is 'why does an increase of susceptibility to competition in the unparasitised host larvae, or an increase of competitive strength in the parasitised host larvae, lead to increased stability?' An increase in the susceptibility to competi-

Fig. 7. Behaviour of the parasitised-larval competition model. The parameters are given in Table 2 and $c_{LL} = c_{IL} = c$ and $c_{LI} = c_{II} = 0$. The different shades of the figure show the period of the parasitoid population cycles as the host fecundity, b and larval competition, c, are varied. The host population only exhibit generation cycles for the parameters considered. Key: SS; steady state, HGCs; half-generation cycles, GCs; generation cycles. Regions in parameter space where the hosts exhibit GCs and the parasitoids exhibit SS correspond to parasitoid extinction.

tion in the unparasitised larvae, or an increase of competitive strength in the parasitised larvae, increases the effects of parasitism on the host population, with more parasitised larvae surviving development into adult parasitoids. Moreover, since the unparasitised larvae do not fare so well in competition, their outbreak rates are decreased. Hence, combining these two factors leads to increased stability.

Host development time

As Cameron et al. (2005) discuss, there may be other competitive effects from the parasitised larvae, in particular, the shortening of the development time of the unparasitised larvae. This decrease in development time is likely to occur due to a relaxation of interference competition for safe pupation sites i.e. cannibalism free space for vulnerable pupae. A possible explanation for this is that large final instar larvae parasitised by *Venturia* are entering the pre-pupal stage more quickly than a similar sized healthy larva (Harvey et al. 1994), a potential mechanism of risk avoidance from cannibalising larvae. However, the effect on the population dynamics are not clearly understood.

In Fig. 9 we show that a short unparasitised larval development time aids in parasitoid extinction, due to an increase in the avoidance of parasitoid attack. We also show that if there is an increase in the duration of host the parasitised larval stage then this also aids in parasitoid extinction, due to a significant increase in the total amount of competition exerted upon the parasitised larvae. For the parameter ranges that we have investigated, there is little change in the qualitative behaviour of the hosts, which only exhibit generation cycles.
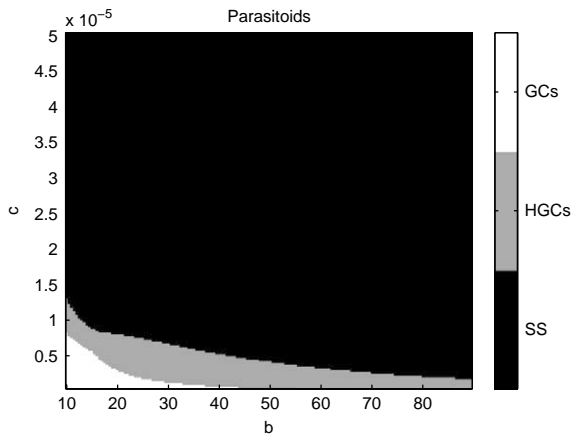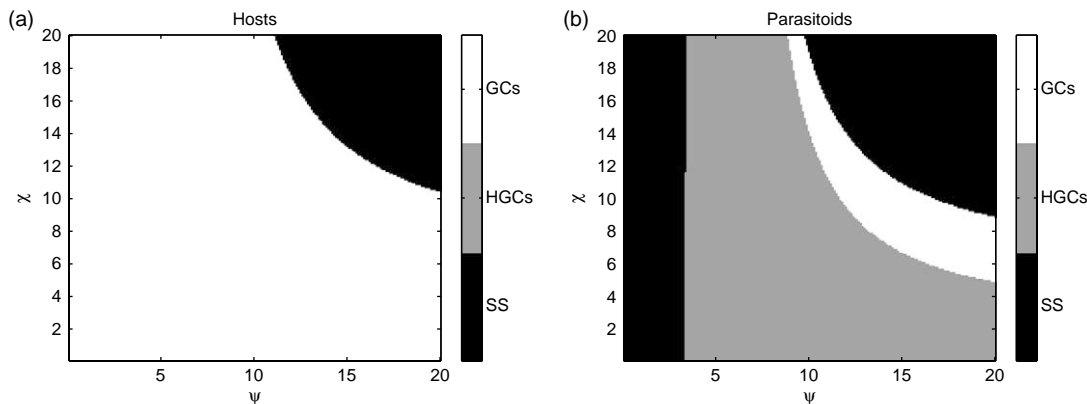
Fig. 8. Behaviour of the parasitised-larval competition model. The parameters are given in Table 2 with b = 50 and $c_{LL} = 1 \times 10^{-4}$. The different shades in the figures (a) and (b) show the period of the host and parasitoid population cycles as we vary the ratio of competitive effects χ, and the ratio of competitive sensitivities ψ. Key: SS; steady state, HGCs; half-generation cycles, GCs; generation cycles. Regions in parameter space where the hosts exhibit GCs and the parasitoids exhibit SS correspond to parasitoid extinction.

Discussion

In this paper we have shown that increased competition between parasitised and unparasitised larval hosts leads to increased stability in the host−parasitoid dynamics. When competition from parasitised larvae is not taken into account, or equivalently, when parasitised larvae do not compete with unparasitised larvae, then the dynamics of the host population can be stabilised through competition, but over a smaller parameter range. However, extreme competition leads to parasitoid extinction. We argue that these phenomena may be observed in many host−parasite systems where parasitised hosts are able to compete with their unparasitised counterparts.

As previous theory suggests, a key parameter for controlling the dynamics of the system is the clumping parameter, k (May 1978, Spataro and Bernstein 2004). However, extreme competition from the unparasitised host larvae results in parasitoid extinction, thus rendering the clumping parameter ineffective. Moreover, highly aggregated adult parasitoids (small k) tend to destabilise the system dynamics. As we have demonstrated here, these factors are dependent on the degree to which the parasitised larvae can compete, and parasitised larvae that are strong competitors tend to stabilise the host−parasitoid dynamics. Host suppression and stability have been shown in other experimental systems (Murdoch et al. 2005). Here we have shown that host stabilisation is obtainable by the parasitoid, which has also been shown by Wearing et al. (2004b) and others. However, increased competition from the parasitised larvae increases the parameter region for which equilibrium population dynamics occur.

This theory agrees with Spataro and Bernstein (2004), in that strong parasitised-larval competition enhances stability, despite the major differences between the two models and the experiments from which they were derived (directly competing, mixed populations of parasitised and unparasitised hosts in our model versus unmixed populations). For the Spataro and Bernstein (2004) model the authors claim two important facts. Firstly, that if parasitised hosts are more susceptible to competition than healthy ones, then the
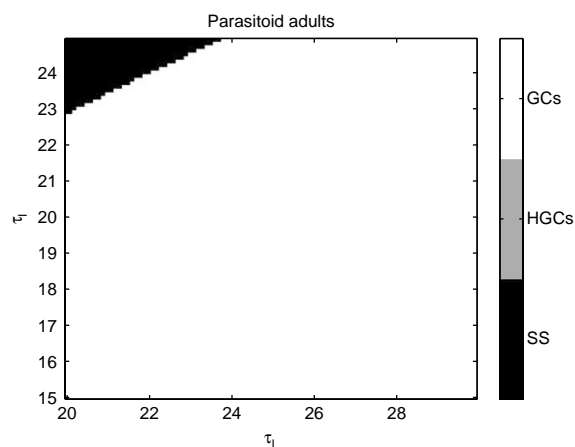

Fig. 9. Behaviour of the parasitised-larval competition model. The parameters are given in Table 2 with $c_{LL} = 1 \times 10^{-6}$ and $\chi = \psi = 1$. The different shades in the figure show the period of the parasitoid population cycles as we vary the duration of host larval stage τ_L and the duration of host parasitised larvae stage, τ_I. Key: SS; steady state, HGCs; half-generation cycles, GCs; generation cycles. Regions in parameter space where the hosts exhibit GCs and the parasitoids exhibit SS correspond to parasitoid extinction.

parasitoid population tends to vanish. This is unsurprising in their model since both types of larvae act independently, and therefore the increased competition simply reduces the parasitoid net growth rate (via intra-class competition) sufficiently so that the parasitoid population is unable to persist. However, in our model the competition on the parasitised larvae comes from both intra- and inter-class competition, and thus the unparasitised larvae can have a significant impact on the parasitoid population. Therefore, the parasitoids can be driven to extinction by strong inter-class competition even if the parasitised larvae exhibit weak intra-class competition. Secondly, Spataro and Bernstein (2004) demonstrate that stability is greatest if intra-class competition between healthy hosts is stronger than that between parasitised hosts. However, we show that whilst strong parasitised-larval competition is an important factor in stabilisation, it is by no means the only route. In Fig. 8 we show that stability is achieved when both the ratio of competitive effects χ and the ratio of competitive sensitivities ψ are large. This corresponds to the parasitised larvae having a large inter-class competitive effect on the unparasitised larvae, the unparasitised larvae having a weak inter-class competitive effect on the parasitised larvae and equal intra-class effects. Hence, we have shown that it is differential inter-class competition effects that promote stability, rather than differential intra-class effects. In conclusion, when the parasitised and unparasitised larvae are lumped into a single stage the mechanisms for stabilisation are vastly different.

Whilst we have shown that the effects from parasitised-larval competition are important, there are other parasitoid induced effects, in particular, host development time. We have demonstrated that if there is a decrease in larval development time then this can cause parasitoid extinction, due to host temporal avoidance of the parasitoid attack. This is akin to the effects of phenological asynchrony between parasitoids and their hosts as a result of environmental change (Godfray et al. 1994, Stireman et al. 2005). However, the host may experience some detrimental effects from the lack of competition they exert upon the parasitised hosts. This competition versus avoidance tradeoff may be important in the dynamics of other host–parasite systems. In addition, other parasitised-larvae induced effects include an increased background unparasitised larval mortality rate (Cameron et al. 2005). However, simulations from our model indicate that this phenomena contributes little to the host–parasite population dynamics, which is consistent with the Godfray and Hassell model.

Very few other models have also considered the effects of parasitised-larval competition. Reed et al. (1996) derived a model to study the potential effects of differential cannibalism between parasitised and unpar-

asitised larvae in the *Plodia–Venturia* system. However, this model does not take into account the age-structured behaviour of the *Plodia–Venturia* system, and cannot predict the full range of cyclic behaviour that is observed experimentally in many host host–parasitoid systems (Gordon et al. 1991). This may be due in part to the fact that their observation of preferential cannibalism of parasitised larvae, akin to increased competition, disappears when resources are provided for the developing larvae. Moreover, like the Spataro and Bernstein (2004) paper, the authors do not explicitly take into account the dynamics and interactions between the parasitised and unparasitised host larvae, which have been shown experimentally to be an important part of the host and parasitoid life-history. We argue that, as experimental results suggest, parasitised larval stages should be included in future models for systems that have competition effects between parasitised and unparasitised larval hosts. Indeed, this approach could be adopted to include host–parasite interactions more generally when there is a lag between infection and death and individuals continue to compete for resources.

Since the developing parasitoid experiences the same fate as its host until it emerges, being able to compete effectively during periods of limited resources appears to be a clear benefit to the parasitoid individual. At the population level, from a parasitoid point of view, by stabilising the host population the parasitoid population is less likely to go extinct from stochastic events when the parasitoid populations (adult parasitoids and parasitised larvae) are low. Thus, by stabilising the host population the parasitoids are helping to ensure their survival. The same could be said of the hosts, except their generation-length population cycles have been shown to be remarkably persistent and are fairly robust to extinction from stochastic effects, and so there seems to be little benefit from stabilisation (Sait et al. 1994 Begon et al. 1995, Bjørnstad et al. 2001, Bonsall and Benmayor 2005). Moreover, during periods of excessive competition, necessary for parasitoid-derived stability, the host numbers are markedly reduced, which in turn increases the likelihood for parasitoid extinction due to a decreased probability for a successful attack. This possibility of parasitoid extinction could be increased if there is a significant change in the development time for either the host larval or parasitoid larval stages. Therefore, we hypothesise that stabilisation via the intra-class competition mechanism is only a benefit to the parasitoid population, but further modelling and/or experiments are required to test our hypothesis.

Like most population models that incorporate self-regulation, we have assumed that competition is a declining linear function of the larval population density. However, there is little experimental evidence to suggest this functional form is valid, since there may

be other nonlinear effects that influence self-regulation, particularly in cases where larvae exploit resources homogeneously. As Getz (1990) shows for discrete-time population dynamics, the incorporation of non-linear effects, such as density-dependent abruptness, can cause population oscillations. This, coupled with intra-class competition between parasitised and unparasitised larvae may show a rich set of behaviours that are not predicted by current theory, and that could have important ecological and evolutionary consequences.

It should be noted that for the *Plodia–Venturia* parameters used in the above models it is unlikely that stabilisation will occur from parasitised-larval competition unless food resources are sufficiently low that the parasitised larvae can assert their competitive strength sufficiently (Cameron et al. 2005). Possible reasons for the superior competitive effect of the parasitised larvae have been discussed at the individual level (parasitised larvae inflict more fatal injuries; parasitised larvae behave differently than unparasitised larvae; and parasitism results in decreased resource requirements). In effect, the overt consequences of being parasitised for the host appear to be benign from a dynamical perspective. However, this may not be true in other host–parasitoid systems where the life-history parameters may be sufficiently different and the consequences of being parasitised are more dramatic. In many host–pathogen interactions, for example, the host may become increasingly more moribund, and hence non-competitive, as the level of infection or parasite load increases, even before death occurs. In this instance, the competition coefficients of such a parasitised or infected host could take the form of a monotonic decay function, which may have significant long-term effects. For example, this may lead to destabilisation since, as we have shown here, competition from parasitised hosts has a stabilising affect which would decrease as the host becomes more moribund.

There are other important factors that could have a significant influence on population dynamics that incorporate competition between parasitised and unparasitised larval hosts. Cameron et al. (2005) have shown that longer periods of exposure to parasitised larvae resulted in an increase in development time of unparasitised individuals, which were larger when they emerged as adults and, thus, had greater reproductive fitness (Gage 1995). These results have been shown in other systems. Bedhomme et al. (2005) have shown that unparasitised yellow fever mosquito larvae, *Aedes aegypti* (Diptera: Culicidae), took significantly longer to develop when reared in competition with larvae that have been parasitised with the microsporidian parasite *Vavraia culicis* (Microsporida: Pleistophoridae). As Washburn et al. (1991) argue, the regulatory role of the parasites can be modified by a range of factors, including availability of resources, competition among

host larvae and changes in feeding behaviour. The authors show that under certain conditions (such as resource limitation), natural enemies may actually increase the fitness of adult hosts by allowing more or larger individuals, or both, to complete development. Not only will this have an important implication for the ecology of host–parasitoid systems (as we have demonstrated above), but may play a significant role in their coevolution.

Whilst the main results of this paper have been aimed towards the interactions between the host, *Plodia interpunctella,* and its parasitoid, *Venturia canescens,* we argue that the model can be applied to many other host–parasitoid systems. For example, Lane and Mills's (2003) *Ephestia kuehniella–Venturia canescens* laboratory system, has shown that in the presence of parasitoids attacking the late larval stage, competition changed from scramble for food to contest for enemy-free space. Other important extensions include host–parasitoid dynamics of idiobionts, which can be modelled by a simple modification (setting $c_{II} = c_{LI} = 0$). In other areas in biology, for example in endophyte-infected plants, the host plants show increased resistance to herbivores and greater growth and competitive performance relative to uninfected plants (Cheplick 2004), which may have major effects on the population dynamics. Thus, given the clear demonstration of the importance of intra-class competition, the long-term population dynamics of host–parasitoid and host–parasite interactions in general could be investigated using the above model.

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Appendix 1. Numerical simulations and spectral analysis

In order to solve the models numerically we differentiate the time-dependent survival probabilities with respect to time to get a system of constant time delay ordinary differential equations (DDEs), which can be solved using many solvers. Thus for the parasitised-larvae competition model (2g) becomes

$$\frac{dS_1}{dt}(t) = S_1(t)\{[c_{II}I(t-\tau_I) + c_{IL}L(t-\tau_I)]$$
$$-[c_{II}I(t) + c_{IL}L(t)]\}$$

In order to solve the system of DDEs one must specify not only the initial conditions, but also the history. All simulations carried out in this paper have zero history populations and unity for the survival probability history, all initial conditions are equal to the history.

To initialise the adult hosts parasitoids two inoculation functions were used (Gurney et al. 1983). This allows the hosts to establish themselves before the parasitoids are introduced, and they play no part in the long-term dynamics of the system (unless multiple attractors exist). For the hosts in the parasitised-larval competition model the adult host recruitment rate, $R_A(t)$ is replaced with

$$R_A(t) = M_L(t-\tau_P)\sigma_P + I_{H_A}(t)$$

where $I_{H_A}(t) = i_{H_A}$ for $t \in (i_{P_A}\text{start}, i_{P_A}\text{start}+1]$. For all simulations carried out in this paper we set $i_{H_A} = 15$ and $i_{H_A}\text{start} = 0$. For the parasitoids (2h) is replaced with

$$R_{P_A}(t) = M_I(t) + I_{P_A}(t)$$

where $I_{P_A}(t) = i_{P_A}$ for $t \in (i_{P_A}\text{start}, i_{P_A}\text{start}+1]$. For all simulations carried out in this paper we set $i_{P_A} = 2$ and $i_{P_A}\text{start} = 200$.

The delay-differential equations are solved numerically using a Fortran subroutine by Hairer and Wanner based on a fourth order explicit Runga-Kutta method with quintic Hermite interpolation (Hairer et al. 1993). Solution values are uniformly outputted for 1 day periods.

Spectral analysis is performed by taking the fast Fourier transform (FFT) of the last 2^{10} solution values of the adult host densities after a transient period of $(5000 - 2^{10})$ days. The FFT was carried out using Matlab's FFT routine.

All Fortran code was compiled and run on a 32-bit Intel dual-processor machine using the G95 Fortran compiler.