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Revenge of the host: cannibalism, ontogenetic niche shifts, and the evolution of life-history strategies in host–parasitoid systems

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ABSTRACT

Question: How does cannibalism in the host alter the evolution of a parasitoid's oviposition strategy? Can differences in cannibalism risk between parasitized and healthy hosts alter the stage-specific foraging of parasitoids? Can host-specific differences in cannibalistic behaviour explain why parasitoids vary in what host stages they attack?

Mathematical methods: We examined the evolutionary dynamics of a stage-structured host–parasitoid model using two complementary approaches: (1) individual-based numerical simulations of evolutionary dynamics, and (2) the theory of adaptive dynamics focusing on evolutionarily stable strategies (ESSs).

Assumptions: Cannibalism in the host is assumed to be stage structured, with larger stages consuming smaller stages. The consumption of parasitized hosts also results in killing of the parasitoid's offspring. Vulnerability to cannibalism of parasitized versus healthy hosts was allowed to vary. The parasitoid's preference for attacking early versus late host stages was the trait under selection and allowed to evolve.

Results: When cannibalism rates increase relative to the parasitoid's attack rates, the ESS of the parasitoid shifts from attacking only early host stages to attacking only late host stages. This shift occurs at lower cannibalism rates when parasitized hosts are more susceptible to cannibalism than healthy hosts. Under equilibrium conditions, a small boundary area exists between these two regions where attacking only early or only late host stages are alternative ESSs. The threshold and alternative stable ESSs are the result of cannibalism, which creates a positive feedback between the parasitoid's oviposition rate and its own mortality. Intermediate strategies, where parasitoids evolve to attack both stages, occur only when host populations exhibit large population oscillations or when generalist parasitoids that attack both stages have a foraging advantage.

Keywords: cannibalism, life-history evolution, mutual predation, ontogenetic niche shift, role reversal, size structure.

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INTRODUCTION

There is little doubt that ecological interactions such as predation and competition influence the evolution of some physiological and behavioural traits of interacting species (e.g. Hairston *et al.*, 1999; Phillips and Shine, 2004; Grant and Grant, 2006). When studying the evolutionary consequences of species interactions, it is commonly assumed that individuals within a species experience the same type of interaction (for a review, see Abrams, 2000). All individuals, however, change in size during ontogeny, which can modify the type and strength of their ecological interactions with other species (for reviews, see Werner and Gilliam, 1984; Yang and Rudolf, 2010; Miller and Rudolf, 2011). Consequently, ontogenetic shifts in species interactions have been observed in most animal taxa (ranging from invertebrates to mammals) and are ubiquitous in marine, freshwater, and terrestrial systems (Polis, 1991; Rudolf and Lafferty, 2011). Increasing evidence indicates that these ontogenetic shifts in species interactions have important consequences for the dynamics of populations and communities (Miller and Rudolf, 2011). Yet, the evolutionary consequences of such ontogentic shifts in species interactions are largely unknown.

Ontogenetic reversal in predation is common in natural communities and leads to 'mutual predation loops' (Polis *et al.*, 1989). Such loops arise because small stages of the predator are often vulnerable to predation by large stages of their future prey (e.g. Polis *et al.*, 1989; Palomares and Caro, 1999; Woodward and Hildrew, 2002; Magalhaes *et al.*, 2005). Although it is generally assumed that predation loops occur among predatory species, they can also arise in host–parasitoid interactions if the host is cannibalistic. Most parasitoids attack early insect life stages (i.e. egg, larvae or pupae) and emerge before the host becomes an adult, typically resulting in the death of the host. However, when hosts cannibalize infected conspecifics, they also typically kill the parasitoid's offspring within the infected conspecific. Thus, with cannibalism in the host, the host–parasitoid interaction is essentially that of reciprocal predation (see Fig. 1).

Cannibalism, or intraspecific predation, is prevalent in a diverse array of natural systems and is very common in invertebrate 'hosts' where older/larger stages consume younger/smaller conspecifics (Fox, 1975; Polis, 1981; Richardson *et al.*, 2010). Cannibalism can determine the dynamics of populations (for a review, see Claessen *et al.*, 2004), alter community dynamics (e.g. Persson *et al.*, 2003; Rudolf, 2007a, 2007b), and recent studies indicate that it can also drive the evolution of life-history traits within a single population (Wakano *et al.*, 2002; Rudolf *et al.*, 2010). Thus, cannibalism could have important consequences for the evolutionary dynamics of host–parasitoid interactions.

Cannibalism in the host species can affect the parasitoid population in at least two ways: (1) it determines the host population dynamics, carrying capacity, and size structure, and (2) it can also increase the mortality rate of the parasitoid via consumption of infected hosts. Cannibalism differs from simple stage-specific background mortality of the host because it creates a positive feedback loop between parasitoid attack and parasitoid mortality. By attacking and killing early host stages, the parasitoid also kills its future predators, thus linking stage-specific predation of the parasitoid directly with its own mortality due to cannibalism. Such density-dependent feedback loops arising from ontogenetic niche shifts often lead to complex dynamics and alternative stable population states that cannot be predicted by classical unstructured models (e.g. De Roos and Persson, 2002; De Roos *et al.*, 2008; Schreiber and Rudolf, 2008). Previous studies also indicate that cannibalism has a significant impact on the dynamics of host–parasitoid and predator–prey dynamics

(for a review, see Claessen *et al.*, 2004), and can alter the conditions for species co-existence in host–parasitoid and other consumer–resource or host–disease interactions (Reed *et al.*, 1996; Rudolf, 2007b; Rudolf and Antonovics, 2007). Cannibalism could therefore also have important evolutionary consequences for parasitoid life-history traits. However, limited work has been done to identify how this common intraspecific interaction can drive the evolution of interactions between a host and its natural enemies.

Evidence from natural systems suggests that there is often an interaction between cannibalistic behaviour and parasitism. Some species exhibit preferential cannibalism of parasitoid-infected larvae (Reed *et al.*, 1996), whereas other species show reduced cannibalism rates of virus-infected larvae (Boots, 1998). Increased rates of cannibalism in parasitoid-infected larvae may arise, because immature parasitoids alter host behaviour and morphology (Godfray, 1994), which can make them more conspicuous and prone to cannibalistic attacks. In addition, infected hosts often show reduced developmental rate and size (Strand *et al.*, 1988; Harvey *et al.*, 1994a, 1996), making them less able to defend themselves from conspecific attacks. This difference in cannibalistic behaviour, ranging from less cannibalism to preferential consumption of parasitized conspecifics, could result in differences in host–parasitoid population dynamics and drive the evolution of parasitoid foraging and egg-laying behaviour.

We used a modelling approach to determine whether ontogenetic changes in intraspecific and interspecific interactions can drive the evolutionary dynamics in a host–parasitoid system. In particular, we tested whether variation in cannibalism rates and selective consumption of parasitized conspecifics, within a size-structured host, can alter the evolution of parasitoid oviposition behaviour. Our results demonstrate that the strength of cannibalism in the host and vulnerability of parasitized hosts to cannibalism determine the evolutionarily stable strategy (ESS) for parasitoid preference for attacking early or late host stages. This suggests that variation in cannibalism rates in the host may explain differences in stage-specific parasitoid foraging strategies in natural populations.

THE MODEL

Here, we consider a stage-structured extension of the classical Nicholson-Bailey (1935) host–parasitoid system where the host goes through three developmental stages: an early larval (or egg) stage (E) and a late larval stage (L), both of which can be attacked by the parasitoid (P), and an adult host stage (A) that is not vulnerable to parasitism. To represent the naturally size-structured and asymmetric interactions between cannibals and their conspecific prey observed in natural systems, late (and thus larger) larval stages (L) cannibalize early (smaller) larval stages (E), but no cannibalism occurs within a stage (Fig. 1). The system represents a variety of natural host–parasitoid systems and is a natural extension of previous host–parasitoid models (Hassell, 2000; Murdoch *et al.*, 2003). The host population is regulated by cannibalism and parasitoid-mediated mortality, with Poisson-distributed attack rates of parasitoids (Nicholson and Bailey, 1935) and cannibals (Costantino *et al.*, 1997). Here we focus on hosts with discrete reproductive events (i.e. cohorts of non-mature individuals are synchronized) and iteroparous adults. Such life histories are widespread in temperate regions (Hassell, 2000). We follow previous models (Hassell, 2000) by making the simplifying assumptions that parasitoids are not egg limited, and they produce one offspring per parasitized host that survives to the next time step. The full dynamics of the model are given by:

$$E_{t+1} = fA_t$$

$$L_{t+1} = E_t e^{-aqP_t - ckL_t}$$

$$A_{t+1} = L_t e^{-a(1-q)P_t} + s_A A_t$$

$$P_{t+1} = E_t[(1 - e^{-aqP_t})e^{-c(1-k)L_t}] + L_t(1 - e^{-a(1-q)P_t}) + s_P P_t$$

where f = the number of offspring produced per adult host, a = per capita attack rate of the parasitoid, c = per capita cannibalism rate of the host, and s_A and s_P = the proportion of adult hosts and parasitoids respectively that survive to the next time step. Note that both parasitized and unparasitized (healthy) early host stages (E) can be cannibalized, as observed in natural systems (Reed *et al.*, 1996; Boots, 1998). We found no empirical records suggesting that cannibalism of parasitoids results in infection, although this is possible for some infectious diseases (Rudolf and Antonovics, 2007). Thus, cannibalizing an infected host is equivalent to consuming the offspring of parasitoids. Consequently, cannibalism can dramatically alter the host–parasitoid relationship by introducing mutual predation (Fig. 1).

In natural systems, parasitized hosts can differ in their probability of being cannibalized from unparasitized hosts (Reed *et al.*, 1996; Boots, 1998). To allow for this differential rate of cannibalism, we introduced a preference parameter, k, with $0 \leq k \leq 1$. If $k < 0.5$, parasitized hosts are more likely than unparasitized hosts to be cannibalized, while the reverse is true if $k > 0.5$, and $k = 0.5$ indicates no preference. Note that differences in k can either represent active preference by cannibals for either prey type, or the differences can be passive because

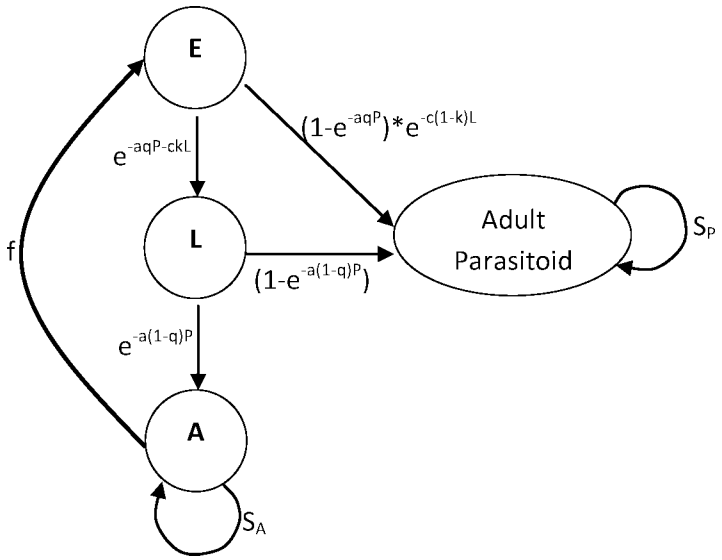

Fig. 1. The life-cycle of the host and its parasitoid. The host has three developmental stages: early (egg or larval) stage (E), late stage (L), and adult stage (A). Parasitoids are able to attack early and late host stages (but not adults) with the preferences q and $(1 - q)$ respectively. Cannibalism is size-structured, where late larvae host stages (L) consume early host stages (E). k and $(1 - k)$ indicate the preference of cannibalizing unparasitized or parasitized early host stages respectively. A detailed description of the model is given in the text.

one stage is simply more prone to being cannibalized (e.g. parasitized hosts are often less mobile and more conspicuous). By varying k we are able to test how differences in the vulnerability to cannibalism of parasitized relative to unparasitized hosts affects the evolution of the parasitoid oviposition strategy.

Explicit solutions to the general population dynamics and stability regions are often difficult to obtain. Thus, we used extensive numerical simulations over a large parameter space to examine the dynamics of the model. In general, increasing the cannibalism rate (c) stabilizes population dynamics. Specifically, increasing cannibalism decreases population oscillations and high levels of cannibalism can lead to stable point equilibria for the host and parasitoid. This is consistent with previous studies demonstrating that simple density-dependence in the host (Hassell, 2000; Murdoch *et al.*, 2003) and cannibalism can often stabilize stage-structured predator–prey dynamics (Rudolf, 2007a). If cannibalism rates are high relative to the search efficiency of the parasitoid (a), the parasitoid cannot invade the host system and stable co-existence of the host and parasitoid is not possible. The exclusion of the parasitoid is largely driven by a decrease in the host density and a concurrent increase in parasitoid mortality rates resulting from high cannibalism of infected hosts. The general population dynamic results found here follow those of similar models that are described, reviewed, and analysed in detail by Murdoch *et al.* (2003). Here, we focus on the evolutionary dynamics of the system, as a detailed analysis of the full ecological dynamics is consistent with previous published results and beyond the scope of this paper.

CANNIBALISM AND THE EVOLUTION OF PARASITOID OVIPOSITION STRATEGIES

Model analysis

To understand how cannibalism in the host influences the evolution of parasitoid oviposition behaviour, we allowed the parasitoid oviposition strategy to evolve between a preference for attacking early or late stages given by q and $(1 - q)$ respectively, with $0 \leq q \leq 1$. Here, q is assumed to be under selection while the per capita attack rate of the parasitoid remains constant. However, we do not make any explicit assumptions about the benefits of cannibalism to the host (e.g. higher growth rates or increased fecundity). Here, we only focus on results for the range of static (non-evolving) cannibalism rates where host and parasitoid can co-exist. However, these results can be used to make predictions about the co-evolution of host cannibalism and parasitoid preference, as evolution of cannibalism in our model is independent of the presence or absence of the parasitoid (or its oviposition strategy). Thus, for any given assumption on the benefits of cannibalism to the host, the co-evolution of host and parasitoid can be predicted from the results here on parasitoid preference and corresponding results for the evolution of the cannibalism rate under different scenarios (e.g. Rudolf *et al.*, 2010).

We used two complementary approaches: (1) individual-based numerical simulations of evolutionary dynamics, and (2) the theory of adaptive dynamics focusing on evolutionarily stable strategies (ESSs) (e.g. Metz *et al.*, 1992; Rand *et al.*, 1994; McGill and Brown, 2007). Because we frequently found oscillatory population dynamics for different parameter combinations (especially at low cannibalism rates), we focus on results from individual-based evolutionary simulations that explicitly account for short-term, transient population dynamics and stochastic processes. We refer to results from adaptive dynamics theory when it provided additional insight to explain the observed patterns from the simulations. Individual-based

simulations are explained in detail in the Appendix. Briefly, we simulated scenarios where the parasitoid evolves and its oviposition preference (q) for early (E) versus late (L) larval stages is the trait under selection. We modelled evolution of q as a continuous trait that is driven by the strength of selection and small mutations. Such phenotypic approaches are commonly used in predator–prey models because the genetics that determine complex traits such as parasitoid (or predator) oviposition behaviour are largely unknown (Abrams, 2000). Simulations were run for a large parameter space to determine how differences in cannibalism rates (c) and differences in the vulnerability of parasitized versus healthy hosts to cannibalism (k) alter the evolution of the parasitoid oviposition strategy.

Results

Our simulations revealed that in the absence of cannibalism, the parasitoid is unlikely to co-exist with the host (Fig. 2). When there is cannibalism in the host species, the ESS of the parasitoid oviposition strategy for early or late larvae depends on the cannibalism rate and

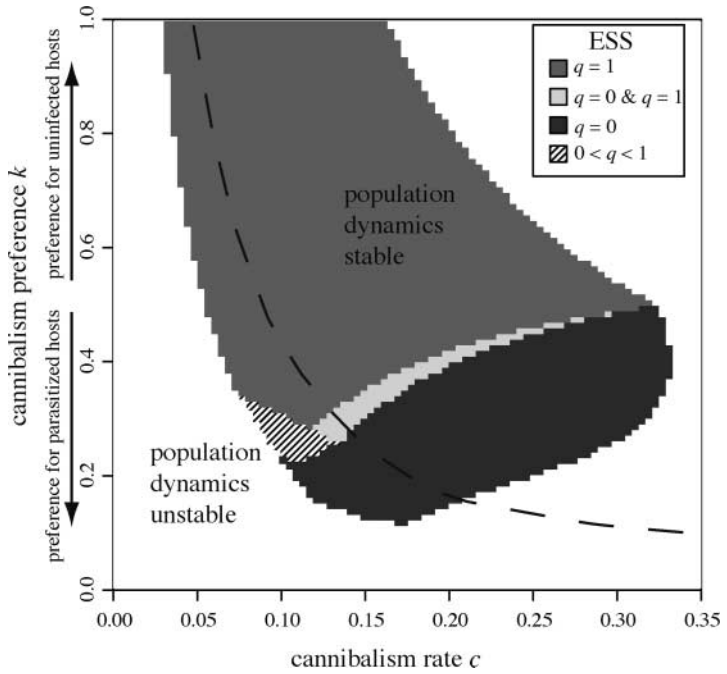

Fig. 2. Plot of ESS values and stability of the population dynamics for dependence on the cannibalism rate (c) and the preference for cannibalizing infected or uninfected hosts (k). The white area is where the parasitoid is unlikely to co-exist with the host. The other areas represent the stable ESS for parasitoid preference q. The two larger areas show where the only stable ESS is either $q = 0$ or $q = 1$ as indicated. In between there is a smaller area (light grey) where both $q = 0$ and $q = 1$ are stable ESS. The hatched area indicates where there is a stable intermediate ESS between 0 and 1. Above the dashed line, the underlying population dynamics are stable. Below the dashed line, the population dynamics are fluctuating for at least some values of q. This figure was produced based on simulations of the evolutionary dynamics as described in the Appendix. Examples of temporal dynamics for the four different types of ESS are given in Fig. 3. Other parameters are $f = 1.2$, $a = 0.2$, $s_A = 0.2$, and $s_P = 0.5$.

differences in the vulnerability of parasitized versus unparasitized hosts to cannibalism (Fig. 2). When parasitized and unparasitized hosts are equally susceptible to cannibalism ($k = 0.5$), or if parasitized hosts are more prone to cannibalism ($k > 0.5$), attacking early host stages ($q = 1$) is always the only stable ESS, even at high cannibalism rates (Fig. 2). However, if cannibalism rates are high and parasitized larvae experience only slightly more cannibalism ($k < 0.5$), the ESS of the parasitoid oviposition strategy shifts from oviposition on early host stages to oviposition on late host stages. In general, lower cannibalism rates in the host require a higher vulnerability of parasitized larvae to cannibalism for this shift to occur. Importantly, this threshold can occur at relatively low cannibalism rates (i.e. at substantially lower cannibalism rates than attack rates of parasitoids) given a sufficient higher vulnerability of parasitized larvae to cannibalism relative to unparasitized larvae. The results were robust to changes in life-history parameters of both hosts and parasitoids. In general, any change in the parameters that improve host fitness (i.e. directly through an increase in f and s_A or indirectly through a decrease in s_P) increases the level of cannibalism within the host, where the parasitoid exists and increases the cannibalism preference for infected hosts that cause a switch to late host stages. By non-dimensionalizing the basic equations of the system (see Appendix), it becomes apparent that the threshold depends on the ratio of the cannibalism rate and attack rate of the parasitoid (i.e. c/a); when the parasitoid attack rate is increased, the shift to the late host stage occurs at higher cannibalism rates. These results were confirmed by additional numerical simulations (not shown here).

To determine whether the observed effect of cannibalism was simply driven by an increase in the mortality of the early stage, we expanded the model to include a density-independent mortality rate in the larval stage. Increasing the mortality rate of early stages relative to late stages slightly increases the range of the ESS that attacks late host stages (i.e. it occurs even if parasitized hosts are less vulnerable to cannibalism). Additional simulations revealed that a very high density-independent mortality rate of early stages can also result in a shift in the parasitoid to attacking late host stages. However, the mortality rate has to be extremely high, and the effects on parasitoid oviposition strategies are negligible relative to only small changes in cannibalism rates. In general, this indicates that it is not simply the mortality rate of early stages *per se* that favours the ESS of attacking late host stages. Instead, evolution of the parasitoid's oviposition strategy is largely driven by the ratio of the attack rate of parasitoids and cannibalism rates.

Interestingly, for most of the parameter region where the population dynamics have stable point equilibria, there is only one stable ESS, at either $q = 0$ or $q = 1$ (Fig. 2). There is, however, a small boundary area between these two regions where both $q = 0$ and $q = 1$ are alternative stable ESSs (Fig. 2). In between the two strategies there is an evolutionarily singular point that is a repeller, i.e. a point the evolutionary dynamics always move away from in time. Furthermore, there is also a small boundary area at lower cannibalism rates, where an intermediate strategy of attacking both host stages to some degree is favoured. Interestingly, the latter case only occurs when populations fluctuate but not when populations have a stable point equilibrium (Fig. 2).

To gain more insight into why only the two extreme oviposition strategies are ESSs when the populations exhibit stable point equilibria, we used adaptive dynamics theory (e.g. Rand *et al.*, 1994; McGill and Brown, 2007) and focused our analyses on ESSs that occur under equilibrium conditions (i.e. non-zero population densities). To identify the ESS, imagine that the 'resident' parasitoid population employs the strategy q and that the host and resident

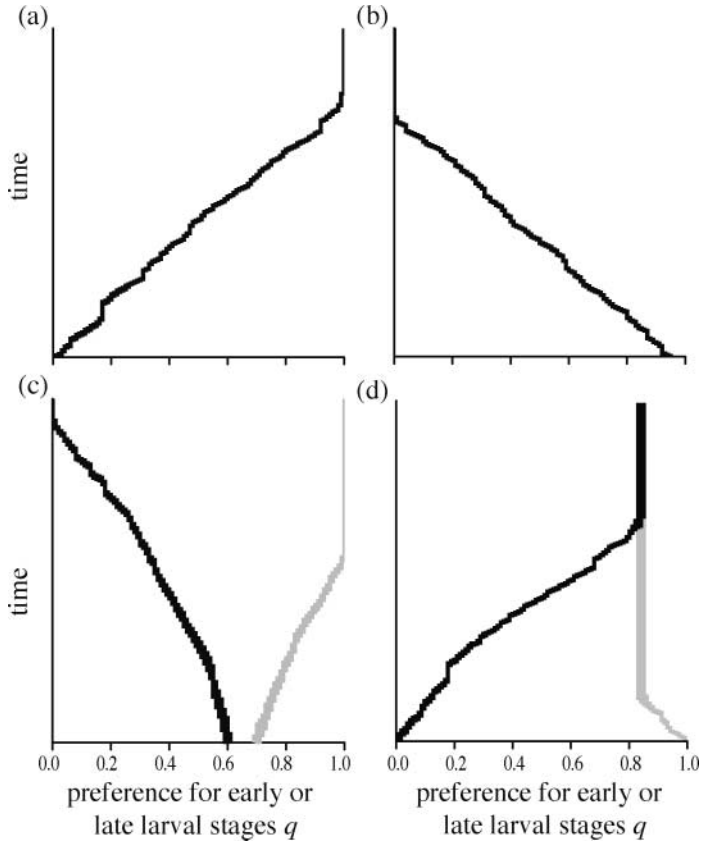

Fig. 3. Plots of evolutionary dynamics for four pairs of values for the cannibalism rate (c) and the preference for cannibalizing infected or uninfected hosts (k). In (a) $k = 0.3$, $c = 0.3$; this is in the ESS $q = 1$ region in Fig. 2. In (b) $k = 0.4$, $c = 0.15$; this is in the ESS $q = 0$ region in Fig. 2. In (c) $k = 0.32$, $c = 0.15$; this is in the ESS $q = 0$ and $q = 1$ region in Fig. 2. In (d) $k = 0.27$, $c = 0.11$; this is in the ESS $0 < q < 1$ region in Fig. 2. The two lines in (c) and (d) are for two separate simulations starting with initial strains differing in preference q. Other parameters are $f = 1.2$, $a = 0.2$, $s_A = 0.2$, and $s_P = 0.5$. The explanation of how these plots were produced is given in the Appendix.

parasitoid populations are at their respective equilibria ($\hat{E}$, $\hat{L}$, $\hat{A}$, and $\hat{P}$). If a new mutation arises in the population leading to a small population of new phenotypes (P_m) that have a different oviposition strategy, q_m, then the invasion rate of the mutant in the population is given by

$$\lambda(c, k, q_m) = \hat{E}\left(\frac{q_m}{q\hat{P}}\right)\left(1 - e^{-aq\hat{P}}\right)e^{-c(1-k)\hat{L}} + \hat{L}\left[\frac{1-q_m}{(1-q)\hat{P}}\right]\left(1 - e^{-a(1-q)\hat{P}}\right) + s_P.$$

Here, the mutant phenotype will invade the resident population if $\lambda(c, k, q_m) > 1$. Note that the invasion rate is a direct extension of the criteria derived in unstructured host–parasitoid models without cannibalism (e.g. van Baalen and Sabelis, 1993; Schreiber *et al.*, 2000). Analyses of this invasion criterion are consistent with the three main outcomes from our individual-based simulations under equilibrium conditions (see Appendix Fig. A1). To gain

more insight into why a mutant can or cannot invade a resident population, we can simplify the invasion conditions. First, we let $P_E = \hat{E}\left(1 - e^{-aq\hat{P}}\right)e^{-c(1-k)\hat{L}}$ and $P_L = \hat{L}\left(1 - e^{-a(1-q)\hat{P}}\right)$. Then, $P_E(P_L)$ becomes the density of new parasitoids that result from attacks on early (late) larval stages at each time step at equilibrium. Rewriting the mutant invasion rate with this notation gives

$$\lambda(c, k, q_m) = \left(\frac{q_m}{q\hat{P}}\right)P_E + \left[\frac{1 - q_m}{(1-q)\hat{P}}\right]P_L + s_P.$$

From the equations that determine the dynamics of the model, we know that at equilibrium $\frac{(P_E + P_L)}{\hat{P}} + s_P = 1$. Subtracting this from the previous equation gives:

$$\lambda(c, k, q_m) - 1 = \frac{1}{\hat{P}}\left(\left(\frac{q_m}{q} - 1\right)P_E + \left(\frac{1 - q_m}{1 - q} - 1\right)P_L\right) = \frac{(q_m - q)}{\hat{P}}\left(\frac{P_E}{q} - \frac{P_L}{(1-q)}\right).$$

From this we know that if $P_E/q > P_L/(1-q)$, only mutants with $q_m > q$ can invade and if $P_E/q < P_L/(1-q)$, only mutants with $q_m < q$ can invade. This can be interpreted by considering that q is determined by how the parasitoid divides its effort (or another resource, e.g. time) between searching for early and late larval hosts. The quantity $P_E/q[P_L/(1-q)]$ is then a measure of the reproductive success per amount of effort for the early (or late) strategy, and is thus a measure of the efficiency of the preference strategy. The only mutants that can then successfully invade a resident population are those that put relatively more effort into the strategy that gives the highest reproductive success per amount of effort. In other words, only a mutant that has a preference for the most efficient strategy can invade. This confirms our individual-based simulation results indicating that only the extreme oviposition strategies (attacking only early or only late host stages) can be ESS when populations are at a stable equilibrium.

Model extensions and sensitivity analysis

Although our main model is a straightforward extension of the well-studied, classical Nicholson-Bailey (1935) model, which has been successfully applied to a large range of host–parasitoid systems, we introduce several model extensions that reflect a range of different life histories in order to test how robust our model predictions are to the specific model assumptions.

First, in addition to cannibalism, there are several other types of density dependence that can regulate host populations and could influence the dynamics of the system. Thus we considered two common additional forms of density dependence within our model framework: (1) density-dependent regulation of host fecundity, where the reproductive output of adult hosts decreases with increasing adult host density: $E_t + 1 = fA_t \exp(-wA_t)$; and (2) density-dependent regulation of larval mortality, where the mortality of larvae in both host stages increases with an increase in the total density of host larvae: $L_t + 1 = E_t \exp(-aP_tq - ckL_t - w(E_t + L_t))$ and $A_t + 1 = L_t \exp(-aP_t(1-q) - w(E_t + L_t)) + s_A A_t$. In both cases, w is positively associated with the strength of the density dependence. As expected, we found that in both scenarios, varying the strength of density dependence (w) changed the region where the parasitoid was able to persist and where oscillations occurred. However, there was no qualitative change in the general evolutionary outcomes, and the

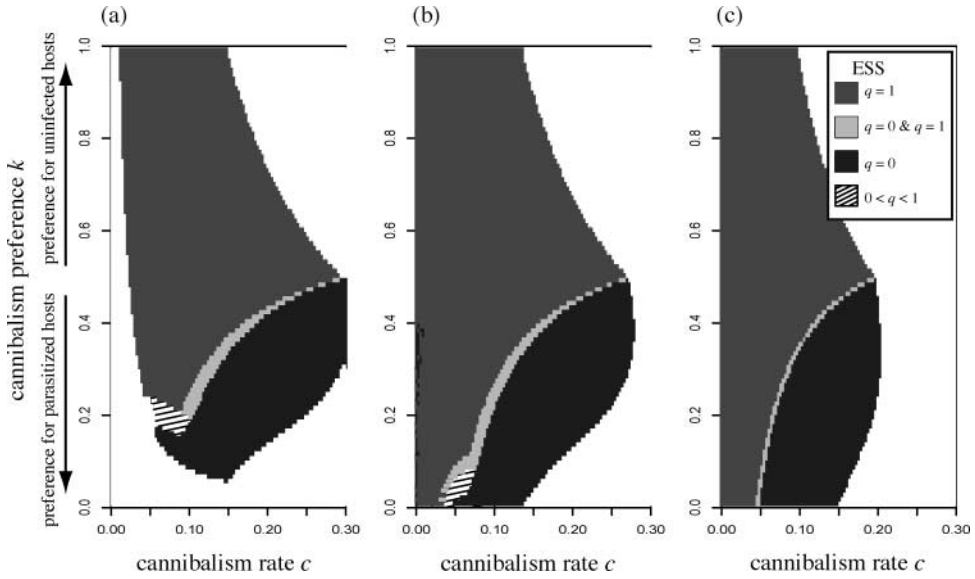

Fig. 4. Example for ESS values for different levels of density-dependent regulation of parasitoid fecundity. Larger values of the parameter w increase the strength of dependence of fecundity on density and result in reduced parasitoid populations. (a) $w = 0.01$, (b) $w = 0.02$, (c) $w = 0.05$. The shaded areas represent the stable ESS for the parasitoid preference q (see legend to Fig. 2). The figure was produced based on individual-based simulations of the evolutionary dynamics with the parameter values: $f = 1.2$, $a = 0.2$, $s_A = 0.2$, and $s_P = 0.5$.

parasitoid ESS depended on the rate of cannibalism and relative cannibalism risk of infected larvae (Fig. 4).

Second, in some systems, parasitoids that attack large host stages may have a fitness advantage. For instance, early host stages may be more likely to die when parasitized, thus reducing the success of parasitoids to complete development in small hosts (Vet *et al.*, 1993). Therefore, we introduced such a fitness advantage by reducing the proportion of parasitoids that emerged from small host stages relative to large host stages by the factor h (with $0 < h < 1$), with $P_{t+1} = E_t(1 - \exp(-aP_tq))\exp(-c(1-k)L_t)h + L_t(1 - \exp(-aP_t(1-q))) + s_PP_t$. As expected, we found that the evolutionary dynamics remained largely unchanged with one exception. Increasing the fitness advantage that parasitoids gain from attacking late host stages (i.e. decrease h) resulted in a preference for late host stages at lower preference for cannibalizing infected hosts (Fig. 5).

Finally, our basic model follows previous formulations (Murdoch *et al.*, 2003) in assuming a linear trade-off between preying on early versus late host stages. However, in some instances, generalists that attack both stages may have a fitness advantage, resulting in a non-linear trade-off. Here, we introduce such a non-linear trade-off by changing the parasitoid preference to: $q + \theta q(1-q)$. Given this formulation, generalists have a higher attack rate and thus a fitness advantage. This advantage increases with θ. Given this trade-off, the strength of cannibalism and the preference for cannibalizing infected hosts still determines the ESS of the relative preference for a specific host stage (Fig. 6), indicating that cannibalism in the host still drives the evolution of parasitoids. However, instead of having an abrupt switch between two opposing ESSs ($q = 0$ vs. $q = 1$), the trade-off results

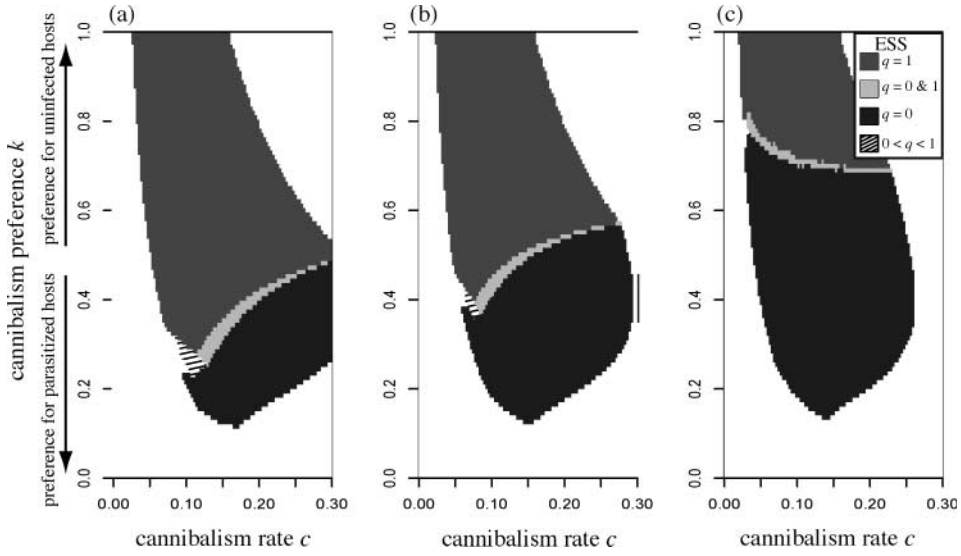

Fig. 5. Plots for ESS values for parasitoid oviposition strategy in systems where parasitoids that develop in small host stages have lower survival rates, giving parasitoids that attack large host stages a fitness advantage. *se* indicates the fraction of parasitoids completing development in early stages. In (a) $se = 1$, so all parasitoids from attacked early stages complete development as before. In (b) $se = 0.9$ and (c) $se = 0.8$, an increasingly smaller fraction of parasitoids completes development in early stages. The shaded areas represent the stable ESS for the parasitoid preference q (see legend to Fig. 2). The figure was produced based on individual-based simulations of the evolutionary dynamics with the parameter values: $f = 1.2$, $a = 0.2$, $s_A = 0.2$, and $s_P = 0.5$.

in a gradual shift across a range of mixed oviposition strategies, and the alternative stable states are lost (Fig. 6). The weaker the non-linear trade-off, the stronger the preference towards one host stage.

DISCUSSION

Traditionally, evolutionary analyses of consumer–resource models have assumed that the ecological role of species in these interactions remains constant during the ontogeny of individuals (for a review, see Abrams, 2000). However, because of changes in size during development, the type of interaction can also change, leading to ontogenetic niche shifts in species interactions in many systems. Here we show that an ontogenetic reversal in a consumer–resource interaction due to cannibalism can alter the evolutionarily stable strategy of a consumer's foraging behaviour and lead to alternative stable states. In general, this emphasizes the importance of accounting for ontogenetic shifts in species interactions for predicting evolutionary dynamics.

Evolutionary dynamics with ontogenetic niches shifts and mutual predation loops

Ontogenetic niche shifts in species interactions can create complex feedbacks that lead to counterintuitive community dynamics (e.g. De Roos and Persson, 2002; De Roos *et al.*, 2003; Schreiber and Rudolf, 2008). Our results suggest that this might also be the case for evolutionary dynamics.

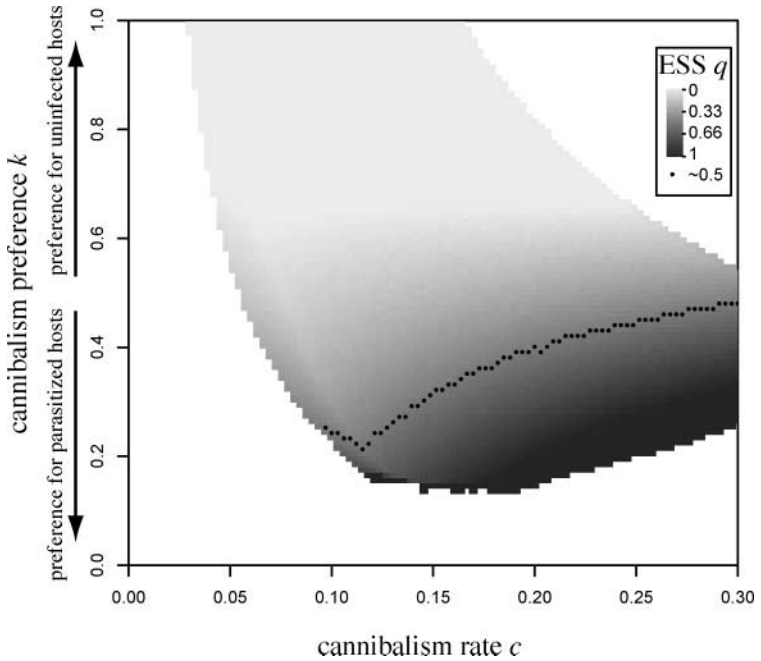

Fig. 6. Plot for ESS values for the parasitoid oviposition strategy when there is a non-linear trade-off for parasitoid preference. The white area is where the parasitoid is unlikely to co-exist with the host. The shaded areas represent the stable ESS for the parasitoid preference q. The black dots approximate the position where the parasitoid has equal preference for the host stage types, that is, $q = 0.5$ is a stable ESS. The figure was produced based on individual-based simulations of the evolutionary dynamics with the parameter values: $f = 1.2$, $a = 0.2$, $s_A = 0.2$, $s_P = 0.5$, and $\theta = 0.1$.

In the absence of cannibalism (or generalist advantage), evolution generally favours parasitoids (i.e. consumers) that attack early larval host stages. This makes intuitive sense, since the consumption of early host stages reduces the availability of later host stages (i.e. $E > L$). Because the reproductive rate of a parasitoid increases with the number of available hosts, parasitoids that attack the early stage have a competitive advantage over parasitoids that attack the late larval stage. This result is consistent with other theoretical studies on parasitoid competition (Briggs, 1993), and the competitive exclusion observed in the red scale system where the parasitoid *Aphytis lingnanensis* was competitively excluded by the parasitoid *Aphytis melinus* because the latter was able to attack earlier host stages (Murdoch *et al.*, 1996).

Our results suggest, however, that cannibalism can alter the evolutionary oviposition strategy of the parasitoid, leading to a switch from attacking early to attacking late host stages. This switch occurred at higher cannibalism rates and when parasitized hosts were more vulnerable than unparasitized hosts. It is important to note that even high rates of cannibalism did not trigger a shift in the ESS to attack late host stages in our model, as long as cannibalism was random or unparasitized larvae were more susceptible to cannibalism. This result clearly indicates that parasitoids did not switch to attack the cannibalistic stage of the host simply because cannibalism increases mortality of the early cannibalized host stage. This result makes sense in light of the linear foraging trade-off and the complex

feedbacks between host and parasitoid mortality due to the mutual predation loop resulting from cannibalism in the host. In this system, the relative abundance of stages is just as important as the relative mortality rate of the parasitoid within each host stage. With cannibalism in the host species, parasitoids that attack early stages effectively reduce the density of cannibals, thereby increasing their own survival. Thus, as long as cannibalism rates are low (relative to parasitoid attack rates) and random, the parasitoid is able to reduce the cannibal density to low enough levels that still produce a higher reproductive output than would result from attacking late host stages. In addition, 'mutant' parasitoids that preferentially attack late host stages also reduce cannibal densities, thus indirectly providing a benefit for the competitors that attack early host stages. However, once cannibalism rates are high enough, a threshold is reached where parasitoids are not able to suppress the cannibal density to the level where the costs of attacking early stages outweighs the benefits. At this point, parasitoids shift all their effort into attacking late host stages. This is consistent with our findings that: (1) it is not the absolute rate of cannibalism, but its relative strength compared with the parasitoid attack rate, that determined the evolutionarily stable oviposition strategy of the parasitoid; and (2) that only one strategy is optimal under equilibrium conditions given a linear foraging trade-off.

The complex feedback between parasitism and cannibalism can also explain the alternative stable states we observed at the boundary of the transition between the two oviposition strategies. Here, attacking early or late host stages were both alternative stable ESSs. However, it is important to note that the final outcome depended on the initial preference (i.e. starting condition) of the resident parasitoid. Only if the resident phenotype initially showed a strong preference for early stages was this strategy also the ESS (e.g. Fig. 3c). This is consistent with the above explanation, that under these conditions, if parasitoids show a strong enough preference for early host stages, they are able to suppress the number of cannibals below a threshold where the benefits of attacking the early stage outweigh the costs of being cannibalized. However, if parasitoids prefer large stages initially, they are not able to suppress cannibal numbers, and invading parasitoids that attack early stages face too much cannibalism and lose the evolutionary race to the resident population of parasitoids that prefer late-stage hosts.

While we also observed intermediate strategies, where parasitoids attacked both early and late host stages, these intermediate strategies were only observed when populations were oscillating and never when populations were at a stable density. We suggest that these oscillations result in population structures and densities that favour parasitoids that attack early stages on some occasions and parasitoids that attack late stage hosts on other occasions. As a consequence, the optimal oviposition strategy evolves to an intermediate level between the two extremes. It is important to note, however, that intermediate, mixed oviposition strategies were the norm when generalists had a fitness advantage due to a non-linear trade-off in attack rates. This suggests that the dramatic shift in the ESS oviposition preference for early or late stages was driven by the linear trade-off in attack rates. However, the fraction of parasitoid attacks on early versus late stages was still driven by cannibalism in the host. The main difference was that the transition between preferentially attacking early versus late stages smoothly transitioned through an intermediate strategy. Thus, regardless of the specific trade-off, cannibalism still drove the evolution of the stage-specific oviposition strategy of the parasitoid. In general, this indicates that complex feedback between species due to ontogenetic niche shifts can result in evolutionary dynamics that cannot be predicted from classical unstructured predator–prey systems.

Stage-specific parasitoid oviposition strategies in natural systems

While there is considerable variation in the oviposition strategy (i.e. what host stages are attacked) of parasitoid species in nature, it is unclear which factors are driving this variation. For instance, the size of the host is known to have a strong influence on selection by parasitoids (for reviews, see Vinson, 1976; Vinson and Iwantsch, 1980). Several previous studies have explained this variation based on host or parasitoid specific life-history traits or the presence of other species that can act as hyperparasitoids (Vinson and Iwantsch, 1980). For example, different host stages may represent different levels of resource availability for parasitoids (i.e. the bigger the host, the more resources that are available) (Harvey *et al.*, 1994). However, in many instances, parasitoids are able successfully to use a large range of host sizes for reproduction (e.g. Harvey *et al.*, 1994; Sait *et al.*, 1997; Akinkurolere *et al.*, 2009), and host size is not always correlated with host quality (Harvey *et al.*, 2004). Our results extend previous studies by demonstrating that variation in a stage-specific host behaviour (i.e. cannibalism) could explain why parasitoid species vary in their preference for attacking different developmental stages of their host.

In general, our model predicts that when hosts exhibit a certain level of cannibalism, parasitoids should avoid small host stages and instead attack larger stages that have a lower risk of being cannibalized, especially when parasitized larvae are more vulnerable to cannibalism. This shift is expected to be even more likely when parasitoids infer some fitness advantage from attacking late host stages. While this has not been tested experimentally, this prediction is consistent with the limited information available from natural host–parasitoid systems. For example, in the Indian meal moth (*Plodia interpunctella*), older, larger larvae heavily cannibalize smaller conspecifics (Boots, 1998). In addition, parasitized larvae are more likely to be cannibalized (Reed *et al.*, 1996). Consistent with model predictions, several parasitoid species of *Plodia* (*Venturia canescens*, *Habrobracon hebetor*) preferentially attack late larval stages that are less vulnerable to cannibalism (Sait *et al.*, 1997; Wearing *et al.*, 2004; Akinkurolere *et al.*, 2009). This preference was observed even though parasitoids can successfully reproduce in all but the smallest of the five larval stages. Similarly, in the cannibalistic fall army worm (*Spodoptera frugiperda*), large instars were more likely to be attacked by parasitoids than smaller stages (Chapman *et al.*, 2000). While these examples are consistent with our predictions, other potential mechanisms cannot be ruled out and explicit experimental tests are needed to test the model. However, given the high frequency of cannibalism in a large range of invertebrate species, including non-carnivorous species (Fox, 1975; Polis, 1981; Richardson *et al.*, 2010), and the propensity for cannibalism to vary substantially within and across species (e.g. Wagner *et al.*, 1999; Michaud, 2003; Burgio *et al.*, 2005), our results suggest that cannibalism is likely an important selective agent that determines the evolution of parasitoid oviposition strategies in natural communities.

CONCLUSIONS

It is clear that individuals change in size during their development and such changes alter the strength and often the type of ecological interactions (Werner and Gilliam, 1984; Yang and Rudolf, 2010). While there is increasing evidence that such ontogenetic shifts alter the dynamics of populations and communities (Miller and Rudolf, 2011), the evolutionary consequences of such ontogenetic shifts are rarely investigated. Our results clearly indicate that a cannibalism-mediated ontogenetic niche shift can drive the evolution of life-history strategies in

consumer–resource systems. This result suggests that examining how other types of ontogenetic niche shifts can influence the evolution of life-history traits will be a valuable topic of research and provide novel insight into the evolutionary consequences of species interactions in natural populations.

ACKNOWLEDGEMENTS

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APPENDIX

Individual simulations

Here we explain how the evolutionary dynamics of the parasitoid were simulated. The population dynamics equations can be adapted to include multiple strains of the parasitoid. When there are n strains of parasitoid, the equations become:

$$E_{t+1} = fA_t$$

$$L_{t+1} = E_t e^{-aP_t^T - ckL_t}$$

$$A_{t+1} = L_t e^{-aP_t^S} + s_A A_t$$

$$P_{t+1}^j = \frac{P_t^j q_j}{P_t^T} E_t \left[\left(1 - e^{-aqP_t^T}\right) * e^{-c(1-k)L_t} \right] + \frac{P_t^j (1-q_j)}{P_t^s} L_t \left(1 - e^{-aP_t^S}\right) + s_P P_t^j$$

$$P_t^T = \sum_{j=1}^{n} P_t^j q_j$$

$$P_t^S = \sum_{j=1}^{n} P_t^j (1 - q_j),$$

where P_t^j is the density of the jth strain of parasitoid at time t and q_j is the corresponding preference. The simulations are the results of iteratively applying these equations to compute the population dynamics for a long period of time. What makes this evolutionary is that the simulation starts with a single strain of parasitoid, $n = 1$, with new strains being generated occasionally as rare, small random mutations of an existing strain. If the new mutants have an advantage over the old strains, the mutants will increase in density and the old strains will decrease, eventually becoming practically non-existent. Typically, one strain will eventually appear that outcompetes the older strains and any further mutations. This will have a preference value close to an ESS.

We introduce the mutants at time intervals of $t_m = 10{,}000$. Beginning with one strain, $n = 1$, at time $t = 1$, we compute the population dynamics until $t = t_m$. The mutant is generated by setting $n = 2$, $P_{t_m}^2 = \varepsilon P_{t_m}^1$, $q_2 = q_1 + r$, where ε is small (we used $\varepsilon = 0.01$) and r is a small random number, which may be positive or negative (but q_j is restricted between 0 and 1). We now continue computing the population dynamics for $t > t_m$ but with two strains of parasitoid. More mutants are then introduced at $t = 2t_m, 3t_m, 4t_m, \ldots$.

To introduce strain P_t^{n+1}, an existing strain of the population is selected to produce the mutant. The probability that the jth strain is selected is $P_t^j / \sum_{i=1}^n P_t^i$. If the new strain is a mutation of the jth strain, the preference of the new mutant is $q_j + r$, where r is a small random number generated for each mutation. The new mutant is then introduced at density $P_{nT}^{n+1} = \varepsilon P_{nT}^j$.

At the end of the simulation, the average preference is computed as: $\sum_{j=1}^n P_t^j q_j / \sum_{j=1}^n P_t^j$. If the simulation is over enough time steps, this value will lie at an ESS. Typically, the strain with preference closest to the ESS will have considerably larger densities than the others.

We run two simulations for every pair of values for c and k in Fig. 2. One has an initial strain with preference $q = 0$ and the other with $q = 1$. Where there is one ESS, the evolutionary dynamics of the two simulations converge and eventually give the same result. Where $q = 0$ and $q = 1$ are both ESSs, the two simulations do not converge. In both simulations, the initial strain outcompetes all the mutants. There is an unstable ESS in between $q = 0$ and $q = 1$. If the initial strain has a preference q less than this unstable ESS, the evolutionary dynamics will tend to $q = 0$; if it is greater, the evolutionary dynamics tend to $q = 1$.

Non-dimensionalization

Here we show why systems with a fixed ratio of cannibalism to parasitoid attack rate c/a will behave similarly. In particular, their ESS will be identical.

If we define new variables $\hat{E}_t = aE_t$, $\hat{L}_t = aL_t$, $\hat{A}_t = aA_t$, and $\hat{P}_t = aP_t$, the equations become:

$$\hat{E}_{t+1} = f\hat{A}_t$$

$$\hat{L}_{t+1} = \hat{E}_t e^{-q\hat{P}_t - \frac{c}{a}k\hat{L}_t}$$

$$\hat{A}_{t+1} = \hat{L}_t e^{-(1-q)\hat{P}_t} + s_A \hat{A}_t$$

$$\hat{P}_{t+1} = \hat{E}_t \left[\left(1 - e^{-q\hat{P}_t}\right) * e^{-\frac{c}{a}(1-k)\hat{L}_t} \right] + \hat{L}_t (1 - e^{-(1-q)\hat{P}_t}) + s_P \hat{P}_t,$$

where we can see that c and a only appear in the ratio c/a. This means systems with the same ratio c/a will behave similarly.

Two systems with different cannibalism and parasitoid attack rates but the same ratio c/a will have identical population dynamics up to a scaling (by the ratio of their attack rates) if the initial conditions satisfy the same scaling. The evolutionary dynamics of both systems can be determined by the system of equations above. In particular, the ESS for the parasitoid preference q will be the same.

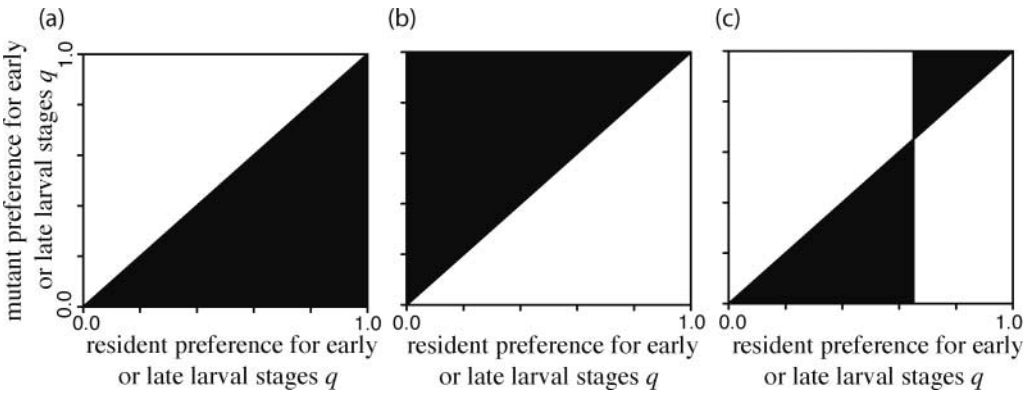

Fig. A1. Pairwise invasibility plots (PIPs) for the parasitoid oviposition strategy under equilibrium conditions based on invasion condition (λ) given in the Results section of the main text. The shaded areas indicate where the mutant phenotype will invade from. The parameter values are $f = 1.2$, $a = 0.2$, $s_A = 0.2$, and $s_P = 0.5$.

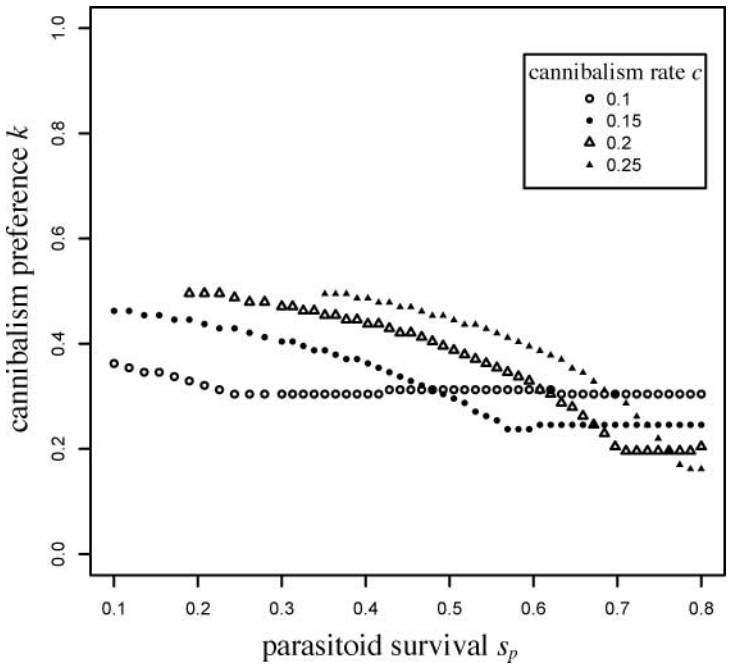

Fig. A2. Plot for the value of parasitoid preference k above which $q = 1$ is a stable ESS, where the parasitoid co-exists with the host. The plot shows how this ESS changes with cannibalism rates for different levels of parasitoid survival s_P. The figure was produced based on individual-based simulations of the evolutionary dynamics with the parameter valus: $f = 1.2$, $a = 0.2$, and $s_A = 0.2$.