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SPECIAL FEATURE

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Evolutionary and population dynamics of host–parasitoid interactions

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Abstract The role of evolutionary dynamics in understanding host–parasitoid interactions is interlinked with the population dynamics of these interactions. Here, we address the problems in coupling evolutionary and population dynamics of host–parasitoid interactions. We review previous theoretical and empirical studies and show that evolution can alter the ecological dynamics of a host–parasitoid interaction. Whether evolution stabilizes or destabilizes the interaction depends on the direction of evolutionary changes, which are affected by ecological, physiological, and genetic details of the insect biology. We examine the effect of life history correlations on population persistence and stability, embedding two types, one of which is competitively inferior but superior in encapsulation (for parasitoid, virulence), in a Nicholson–Bailey model with intraspecific resource competition for host. If a trade-off exists between intraspecific competitive ability and encapsulation (or virulence, as a countermeasure) in both the host and parasitoid, the trade-off or even positive correlation in the parasitoid is less influential to ecological stability than the trade-off in the host. We comment on the bearing this work has on the broader issues of understanding host–parasitoid interactions, including long-term biological control.

Key words Trade-off · Virulence · Encapsulation · Intraspecific competition · Population persistence and stability · Polymorphism

Introduction

Parasitoids are insects that lay their eggs on, in, or near the bodies of other arthropods. Their biology is tightly coupled to their hosts for all aspects of their development and survival. Parasitoids account for almost a fifth of all Metazoa known and exhibit a complex and wide variety of physiologies, behaviors, and life strategies. This variation in parasitoid life history characteristics can occur at the individual level, the population scale, and the regional level, and analyzing the expression of this genetic variation on which selection acts is grist to the mill for life history evolution (Roff 1992). Not only do parasitoids provide model organisms for understanding the life history evolution and population dynamics of predator–prey interactions (Hassell 1978; Godfray 1994; Quicke 1997), but also these organisms are of considerable economic importance in the control of pest populations (Waage and Greathead 1986; Gauld and Bolton 1988).

In this article we explore the population and evolutionary dynamics of host–parasitoid interactions. We begin by examining the constraints on genetic covariance and trade-offs in parasitoid wasps. We then provide an overview of some previous theoretical and empirical studies before introducing a general host–parasitoid model and demonstrating how evolution can alter the dynamics of host–parasitoid interactions. We then consider a simple evolutionary interaction between hosts and parasitoids, examining the conditions for the evolution of parasitoid virulence. In the final section we comment on directions for future work.

Trade-offs and genetic covariance

At the level of the individual wasp, many phenotypic traits are highly variable. For example, egg size, shape, and number in many parasitoids such as the Ichneumonidae or Chalcidae are highly variable (Iwata 1958, 1962, 1966). How these morphological and reproductive strategies of wasps (how many eggs to lay and what sex ratio to produce) impinge on life history strategies have been the focus of much research (Charnov et al. 1981; Iwasa et al. 1984; Waage and Godfray 1985; Godfray 1987).

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Parasitoid fecundity, for instance, is known to be correlated with the stage of the host attacked (Price 1973, 1974). Wasps attacking earlier host stages have greater fecundity, and this was originally explained in terms of the *balanced mortality hypothesis.* Parasitoids that lay their eggs in early host stages but delay development until the host reaches maturity suffer increased levels of mortality compared to parasitoids which attack later host stages. Thus, the balanced mortality hypothesis predicts that parasitoid fecundity should be the inverse of juvenile parasitoid mortality. Price (1974), for example, showed for ten parasitoids of the pine sawfly *Neodipiron swainei* that this relationship accounts for more than 90% of the variation in the data.

Parasitoids with high fecundities are found in areas supporting relatively low host populations, often at the edge of the host species range. In marginal habitats, the correlation between fecundity and juvenile mortality is very strong (Price 1974) and could be explained by the balanced mortality hypothesis. For example, although the positive correlation between fecundity and host stage attacked for the ichneumonids attacking *N. swainei* is substantial, the lack of taxonomic or phylogenetic controls may inflate the degree of correlation between related species.

In comparison, a taxonomic controlled analysis showed that for 474 Hymenoptera fecundity was not correlated with host stage attacked (Blackburn 1991). Moreover, size (measured as adult length) and fitness (defined in terms of the fecundity of the adult) were not correlated. Similarly, no significant correlations between adult life span and fecundity were identified. Blackburn suggests that size is less important in wasps because the major determinant of mortality is host ecology rather than parasitoid morphology (Price 1974).

Such phenotypic correlations even with taxonomically controlled analyses may not necessarily imply the existence of a physiological or genetic trade-off. Although natural selection works on individual phenotypes, the effectiveness of selection in changing the composition of a population or population interactions depends on the heritability of phenotypic characteristics. Levins (1968) outlined that trade-offs must constrain an organism. However, observations of strong correlation between traits do not necessarily imply a genetic covariance and the existence of a trade-off; this is clearly dependent on the degree of heritability of the characters. If heritability is low then any phenotypic correlation may arise primarily through environmental correlations, and the sign and magnitude of a genetic correlation between two characters cannot be determined simply from the phenotypic correlations (Falconer 1989).

It may be, for instance, that in marginal habitats the correlation between fecundity and juvenile mortality is strong because of environmental constraints rather than physiologically derived trade-offs. *Size–fecundity relationships may be confounded by environment or phenotype–environment interactions*. We urge caution in attributing correlations between particular life history traits and suggest that a more rigorous approach to understanding the existence of genetic covariance in parasitoid life history schedules is now required.

Parasitism and coevolution

At large geographic scales, both abiotic and biotic processes influence the variation in parasitoid life history characteristics and the evolutionary interaction between hosts and parasitoids. For example, across Europe the interaction between *Drosophila melanogaster* and *Asobara tabida* shows phenotypic variation in resistance to host encapsulation ability, sex ratio, host selection, and diapause (Kraaijeveld and van der Wel 1994; Kraaijeveld and van Alphen 1994, 1995).

Correspondingly, at a more local scale (within populations), hosts also have considerable variation in their ability to encapsulate or defend themselves against attack by parasitoids (Kraaijeveld and Godfray 1997). However, this defense is costly to the host (*D. melanogaster*): hosts that are good at encapsulating are poor competitors against conspecifics. Recently, further work has shown that the trade-offs in hosts that suffer multiparasitism may be even more complex (Fellowes et al. 1998).

From a population dynamic perspective, encapsulation by host insects can stabilize otherwise unstable interactions (Godfray and Hassell 1991) by allowing a fraction of the hosts to be effectively in a refuge from parasitism (Hassell 1978). The evolution of encapsulation within the framework of a standard host–parasitoid model (e.g., Nicholson 1933; Nicholson and Bailey 1935) has been shown to lead to the evolution of a single monomorphic type. If additional ecological factors limit population growth, then polymorphisms in encapsulation may be more likely (Godfray and Hassell 1991). The coexistence of these polymorphisms arises simply because the population regulatory factors are stronger within the host (e.g., self-regulation) than the factors operating between the host and parasitoid (e.g., parasitism).

More recently, Hochberg (1997) has shown, in a theoretical study, that the costs of defense versus the cost of concealment in a refuge can have different consequences for the coevolution of the host–parasitoid interaction. In the absence of parasitoid evolution, concealment evolves to frequencies equal to or greater than the fraction that encapsulates. If parasitoids are allowed to coevolve then the frequency of concealers is always greater than the frequency of encapsulators. This asymmetrical relationship is brought about by differential loss of reproductive effort by the parasitoid: it is more costly to parasitoids to have eggs encapsulated than to miss an encounter as the result of concealment.

Host–parasitoid population model

Here, by incorporating host density dependence into a discrete-time host–parasitoid model we develop a simple scenario for exploring the role of natural selection in the interaction between a host and a specialist parasitoid. All numerical simulation of models in this paper was done by MATLAB version 5.0.0.4064 (Math Works 1996).

In this model, the host density dependence acts after parasitism and the model is of the form:

$$H_{t+1} = \lambda H_t g(H) f(P)$$
$$P_{t+1} = H_t\left(1 - f(P)\right) \quad (1)$$

where λ is the growth rate of the host population (H), $g(H)$ is a function for the intraspecific effects, and $f(P)$ is a function describing the host's escape rate from the parasitoid (P) at time t. When explicit functions for competition and parasitism are considered, the model can be recast as

$$H_{t+1} = \lambda H_t \exp(-\beta H_t)\left(1 + aP_t/k\right)^{-k}$$
$$P_{t+1} = H_t\left[1 - \left(1 + aP_t/k\right)^{-k}\right] \quad (2)$$

where $g(H)$ is replaced by a simple function for intraspecific competition of the host $\exp(-\beta H_t)$ in which β is the strength of competition. The nonlinear function for parasitism follows May (1978) and assumes that some form of density dependence acts on the foraging ability of the parasitoid. Here, k is the clumping parameter of the negative binomial distribution and a is the searching efficiency of the parasitoids. In the absence of parasitoid, the equilibrial abundance of the hosts is

$$H^* = \ln\lambda/\beta \quad (3)$$

In the presence of parasitoids but in the absence of density dependence, the abundance of the host population at equilibrium is

$$H_p^* = \frac{k \cdot \left(\lambda^{1/k} - 1\right) \cdot \lambda}{a \cdot (\lambda - 1)} \quad (4)$$

From these, a simple criterion $q = H^*_p/H^*$ characterized by β/a (Beddington et al. 1975, 1978) can be used to assess the impact of the parasitoid on the host population (H^*_p) with respect to the host population in the absence of parasitism (H^*). The population dynamics of these model have been thoroughly investigated (Beddington et al. 1975; May et al. 1981; Hastings 1984; Bonsall et al. 1999). The stability properties and impact of the natural enemy of this model are shown in Fig. 1. It is clear that the populations can show a range of dynamical behaviors from regions in which the interaction is not persistent through limit cycle dynamics to stable host–parasitoid interactions (Beddington et al. 1975; Hastings 1984).

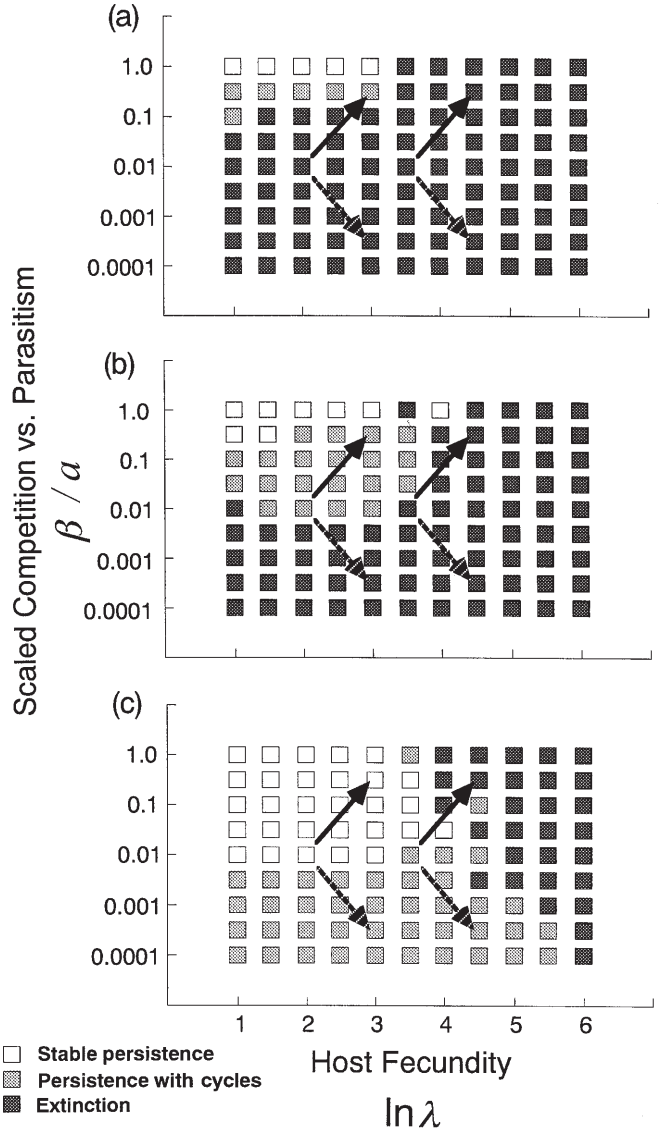

Fig. 1a–c. Numerical simulations with the direction of evolution (*solid* and *striped arrows*) for parasitoids showing varying degrees of aggregation: (**a**) absent, i.e., random attack ($k = \infty$); (**b**) moderate ($k = 2.0$); and (**c**) strong ($k = 1.0$). As the parasitoid aggregates strongly at high density ($k \leq 1$; May 1978), the parameter region for persistent and stable interactions increases

r–K selection

Models of evolutionary processes can be formulated in terms of trade-offs in life history variables or, in a more heuristic way, in the form of processes acting on species near their population equilibria (*K*-selection) or on species perturbed away from the equilibrium (*r*-selection) (MacArthur and Wilson 1967; Mueller 1988; Pianka 1988). Here we adopt the second formulation and assume that evolution tends to maximize population size (described in Eq. 2). If selection for increased host fecundity or increased parasitoid searching efficiency acts unhindered, then it can alter the stability properties of the population interaction from stable equilibria to limit cycles (striped arrows in Fig. 1c).

Empirical evidence provides some support that shifts in population dynamics are a consequence of evolutionary changes. Recently, Tuda and Iwasa (1998) and Tuda (1998) demonstrated that changes in the population dynamics between the bruchid beetle *Callosobruchus maculatus* and its parasitoid *Heterospilus prosopidis* can occur because of evolutionary shifts in the demography of the host population. Under the initial starting conditions, *C. maculatus* larvae showed scramble-type competition and the host–parasitoid dynamics were oscillatory. However, through the

course of the experiment, the competition type changed to a contest type and the population dynamics were stabilized. The evolution toward the contest-type competition is promoted when resource units are small for competitors (Tuda and Iwasa 1998). The direction of the observed evolution could be projected onto Fig. 1a; upward to the stable region.

These results are obviously influenced by other physiological, ecological, and behavioral constraints. If, for example, resources are more or less continuously distributed, the competition evolves toward scramble-type or more efficient resource users (Nicholson 1957; Stokes et al. 1988). By contrast, when a population is subdivided and isolated from each other, the resultant structured deme can favor scramble competitors (Colegrave 1997).

If only a fraction $(1 - p)$ of the host population is susceptible to parasitism, with (p) protected by means of a spatial refuge, invulnerable host stage(s), or immunity, then this allows the host–parasitoid population to persist by preventing the parasitoid from overexploiting its host (Bailey et al. 1962; Godfray and Hassell 1991). This effect can emerge as a consequence of parasitoid behavioral tendency: parasitoid aggregation allows a probabilistic refuge for the host (Fig. 1). At high parasitoid densities, host's refuge $p = (1 + aP_t/k)^{-k}$ (governed by the strength of nonlinearity, or aggregation, in parasitism) can favor persistent and stable host–parasitoid interactions (Fig. 1c). This effect can be accompanied by the effects of pseudointerference (Free et al. 1977).

Again, evolutionary effects can change the stability properties of the interaction (Fig. 1a–c). If the parasitoid shows marked density dependence (e.g., through spatial aggregation to particular host types) then it is likely that changes in the demographic parameters of the host and parasitoid will lead to observed changes in both the population and evolutionary dynamics. The equilibrial abundance of hosts and parasitoids can also be affected by changes in host or parasitoid demographic characteristics.

It is important, however, to realize that natural selection acts on the demographic characteristics of individuals such as the competitive ability of the host or the searching efficiency of the parasitoid and not on populations. The changes in stability of the population interaction arise as a *consequence* of life history evolution.

Parasitism and the evolution of virulence

It is known empirically and theoretically that hosts can show genetic variation for defense against parasitism (Godfray and Hassell 1991; Kraaijeveld and Godfray 1997) and, given certain ecological constraints, populations may be polymorphic for encapsulation ability (Godfray and Hassell 1991). What is less clear is whether parasitoids are likely to show potential genetic variation on which natural selection can act to overcome host defense mechanisms or invasion criteria. By defining a particular physiological trade-off, we examine the dynamics of virulence in a simple model for parasitoid virulence.

Virulence: competitive ability trade-off

The evolution of virulence in host–parasitoid interactions is best considered as a trade-off against some other life history characteristic such as parasitoid larval survival or competitive ability. Clear evidence for the existence of trade-offs between intraspecific and interspecific characters in host populations has recently been demonstrated (e.g., Kraaijeveld and Godfray 1997). For parasitoids, however, little empirical evidence exists for trade-offs between characters associated with the interaction with its host. Rather than assuming a particular mechanism for a trade-off (such as energetic cost functions), we concentrate on examining the effect of trade-offs, in the hosts and parasitoids, on the population dynamics of the interaction. A trade-off between competitive ability and virulence is assumed in both the host and parasitoid and also a positive correlation (known for parasite; Ebert 1998) in the parasitoid to allow comparison of the addition of a trophic level. We find that either the trade-off or positive correlation in the parasitoid is less influential to ecological stability than the trade-off in the host.

The dynamics of the monomorphic host–parasitoid interaction correspond to Fig. 1a; as k becomes infinitely large, Eq. 2 becomes the Nicholson–Bailey model with random parasitoid attack. We explore here three host-parasitoid evolutionary scenarios and their consequence on population dynamics. First, we consider a trade-off between host competition and encapsulation, second, a trade-off between parasitoid competition and virulence (as a countermeasure of encapsulation), and finally a positive correlation between parasitoid competition and virulence.

A basic model

Suppose that there are two phenotypes of hosts and two phenotypes of parasitoid. Host phenotype 1 (density H_1) is competitively inferior to the other phenotype (H_2), while it performs better at encapsulating parasitoid eggs. A similar trade-off exists in the parasitoid: the parasitoid phenotype 1 (P_1) is an inferior competitor but has a higher virulence against the host than the mutant phenotype 2 (P_2). This coevolutionary model can be represented as

$$\begin{aligned} H_{1,t+1} &= \lambda H_{1,t} \exp\left(-\beta_1 H_{1,t} - \beta_2 H_{2,t}\right) f_{11} f_{12} \\ H_{2,t+1} &= \lambda H_{2,t} \exp\left(-\beta_2 H_{2,t}\right) f_{21} f_{22} \\ P_{1,t+1} &= c\left[H_{1,t} f_{12}\left(1 - f_{11}\right) + H_{2,t} f_{22}\left(1 - f_{21}\right)\right] \\ P_{2,t+1} &= c\left[H_{1,t}\left(1 - f_{12}\right) + H_{2,t}\left(1 - f_{22}\right)\right] \end{aligned} \tag{5}$$

where λ is the fecundity of host, β_i is the strength of intraspecific (or within-phenotype i) competition, c is the number of offspring of parasitoid per host, and f_{ij} is the escape rate of the host phenotype i from the parasitoid phenotype j. The model assumes that the host phenotype 2 is not affected by any density dependent effect in the other phenotype, and that the parasitoid phenotype 2 always wins in larval competition between the two parasitoid phenotypes. Eggs oviposited and embedded in host tissue are partially protected (by, for instance, injected poly-

dnaviruses; Vinson and Scott 1974), with the rest killed through an encapsulation response by the host. For simplicity, we assume within-patch interactions and that the parasitoid attacks the immature stage of the host randomly rather than aggregatively as in Eq. 2. The function for parasitism follows the Nicholson–Bailey model:

$$f_{ij} = \exp\left(-a \cdot \gamma_{ij} \cdot P_{j,t}\right) \tag{6}$$

where γ is the virulence of the parasitoid or a countermeasure of encapsulation by the host. When trade-offs exist between host competition strength and virulence and also between parasitoid competition and virulence, the virulence is modified by attenuation parameters d_H and d_P ($0 < d_H, d_P < 1$):

$$\begin{aligned} \gamma_{11} &= d_H \gamma_{21} \\ \gamma_{22} &= d_P \gamma_{21} \\ \gamma_{12} &= d_H d_P \gamma_{21} \end{aligned} \tag{7}$$

The two phenotypes of the host are produced by a suite of clones ($l = 1, 2, \ldots, n$), producing a fixed probability $q_{H,l}$ of the mutant phenotype H_2. Given the density of clone l is x_l, then the total host density is given by

$$H = \sum_{l=1}^{n} x_l \tag{8}$$

and the density of the mutant phenotype, H_2:

$$H_2 = \sum_{l=1}^{n} q_{H,l} \cdot x_l \tag{9}$$

The average fraction of host phenotype 2 is given by

$$\overline{q_H} = \frac{H_2}{H} \tag{10}$$

A similar formulation can be applied to the parasitoid. If the density of parasitoid clone m is y_m then the total density of the parasitoid population is

$$P = \sum_{m=1}^{n} y_m \tag{11}$$

and the density of parasitoid phenotype 2 is

$$P_2 = \sum_{m=1}^{n} q_{P,m} \cdot y_m \tag{12}$$

This gives the average fraction of the parasitoid phenotype 2 as

$$\overline{q_P} = \frac{P_2}{P} \tag{13}$$

The evolutionary dynamics of the host phenotypes, in the absence of the parasitoids, can be derived by a straightforward calculation of the fitness conditions for coexistence:

$$w_{H_1}\left(H_1^*, H_2^*\right) = w_{H_2}\left(H_2^*\right) = 1 \tag{14}$$

Here w_{H_1} and w_{H_2} are the fitnesses of phenotype 1 and 2, respectively, estimated at equilibrium (H_1^*, H_2^*). This gives $H_1^* = H_2^* = 0$; otherwise, $w_{H_1} < w_{H_2}$ holds: the competitvely superior phenotype 2 always outcompetes the other in a host-only population (Appendix).

With the addition of the parasitoid, we proceed with numerical simulations of population and evolutionary dynamics because the formulation of host–parasitoid population dynamics is too complex to derive intuitive analytical results of stability criteria and persistent invasibility, and because transient behavior far from an equilibrium point is difficult to predict analytically (Case 1995; Morton et al. 1996). We iterate the model for 1000 generations or until either the host or parasitoid is extinct. Extinction occurs when the total population density (the sum of the densities of two phenotypes) becomes less than 0.001. Extinction of particular phenotypes is checked by the same criterion. The baseline parameter set is shown in Table 1.

Ecological stability and persistence

First, the effect of virulence ($0 \le \gamma_{ij} \le 1$) on the host and parasitoid population dynamics is examined with a single phenotype of each (Fig. 2). The parameter boundaries for persistence with cycles and for stable persistence are characterized by $0.27 < \beta_i/a' < 0.73$ and $0.73 < \beta_i/a' < 1.87$, respectively, where $a' = \gamma_{ij}a$, reduced attack rate by trade-offs (Fig. 1a). When the host phenotype 1 interacts with either parasitoid phenotype, with high virulence ($0.365 < \gamma_{1j}$) the parasitoid rapidly goes extinct (Fig. 2a,b). Lower virulence allows persistence with population cycles ($0.137 < \gamma_{1j} \le 0.365$), and even lower virulence allows stable persistence, or damped oscillations ($0.0535 < \gamma_{1j} \le 0.137$). However, the parasitoid goes extinct if the virulence becomes too low ($\gamma_{1j} \le 0.0535$). When host phenotype 2 interacts

Table 1. The definition and baseline values of parameters for Eqs. 5–13

Parameter	Definition	Baseline value
λ	Host fecundity	10
β_1	Strength of host phenotype 1 competition	0.01
β_2	Strength of host phenotype 2 competition	0.1
a	Parasitoid attack rate	0.1
c	Number of parasitoid per host attacked	1
H_0	Initial host density	50
P_0	Initial parasitoid density	10
$\overline{q_{H0}}$	Initial average fraction of host phenotype 2	0.01
$\overline{q_{P0}}$	Initial average fraction of parasitoid phenotype 2	0.01
γ_{ij}	Virulence of parasitoid phenotype j on host phenotype i	0–1
d_H	Decrease in host encapsulation by a trade-off with competition	0–1
d_P	Decrease in parasitoid virulence by a trade-off with competition	0–1

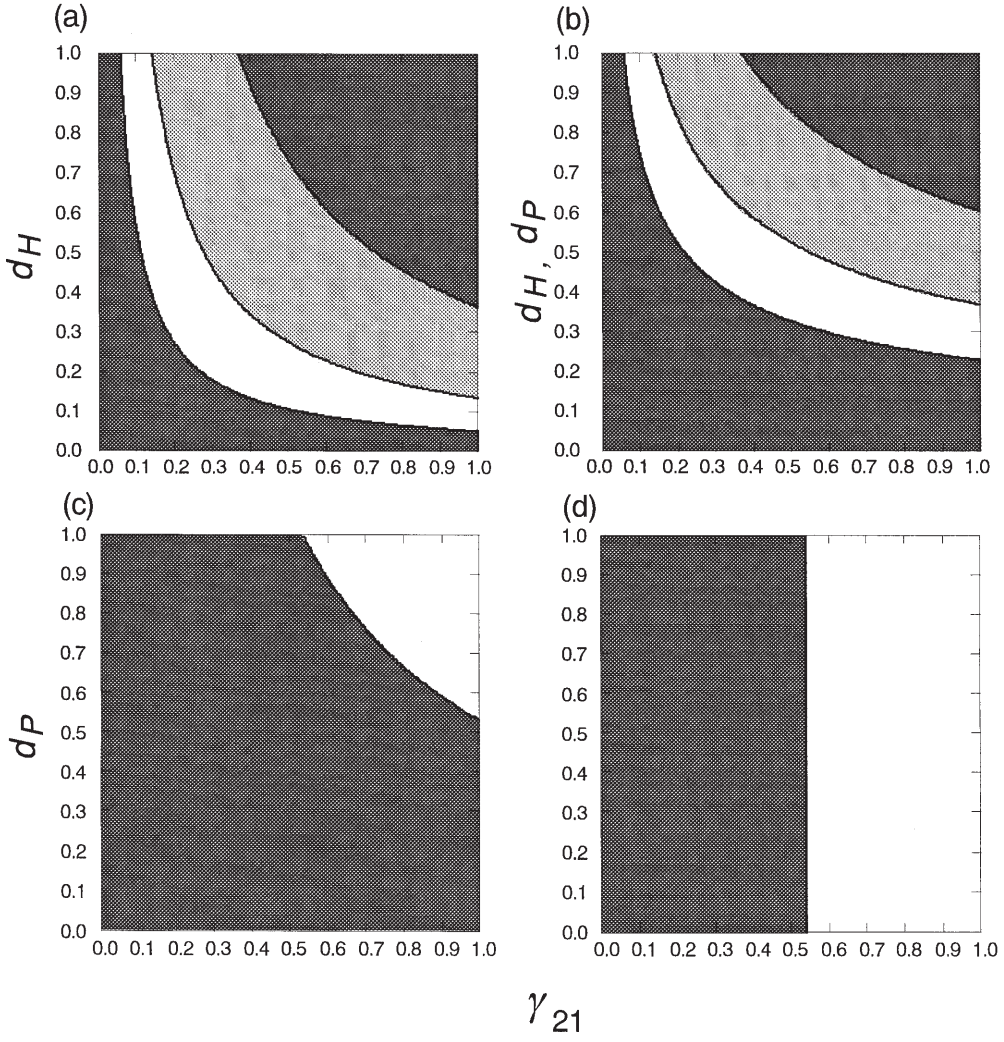

Fig. 2a–d. The general effects of a trade-off between virulence and competitive ability, showing how these effects can influence the ecological dynamics of the monomorphic interactions of (**a**) resident host–resident parasitoid, (**b**) resident host–mutant parasitoid, (**c**) mutant host–mutant parasitoid, and (**d**) mutant host–resident parasitoid. The persistence of the interaction is critically dependent on the strength of the virulence of the parasitoid (and its countermeasure of the host) against the mutant host phenotype (phenotype 2). Only intermediate levels of virulence promote persistence of interactions between the resident host phenotype (phenotype 1) and either parasitoid phenotype (**a** and **b**). For a mutant host, higher virulence is allowed for persistent interaction with the parasitoid (**c** and **d**). *White area*, stable persistence; *shaded area*, persistence with unstable dynamics; *black area*, extinction

with either parasitoid phenotype, stable persistence is possible with high virulence: $\gamma_{2j} > 0.0535$, otherwise, the population goes extinct (Fig. 2c,d).

Consider, first, the persistence of a host–parasitoid interaction when trade-offs exist in both host and parasitoid. As a single-species host population, an initially rare phenotype 2 quickly increases and dominates the population as predicted, which results in bounded fluctuation. When the parasitoid is present, however, the invasion of the mutant host phenotype is only successful under limited conditions, at around persistence–extinction boundaries for monomorphic resident host–resident parasitoid interactions (hatched area in Fig. 3). The polymorphism in this region is maintained either at constant frequencies (Fig. 4a,e) or with periodically oscillating frequencies of the two phenotypes

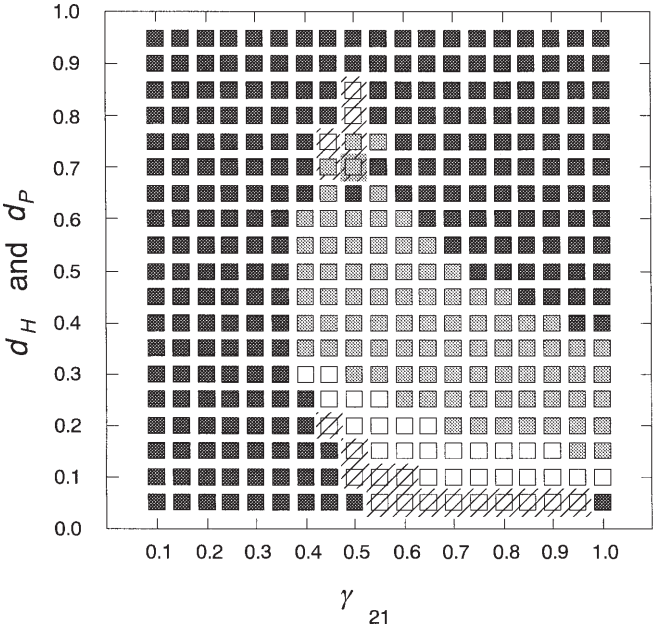

Fig. 3. Only intermediate levels of virulence promote persistence, mostly of the monomorphic host–parasitoid interaction (i.e., between H_1 and P_1). Notice, however, a new stability region emerges between persistence boundaries for monomorphic interactions. *White squares*, stable persistence; *shaded squares*, persistence with unstable dynamics; *black squares*, extinction. *Hatched area*, H_2 invasion; *shaded area*, P_2 invasion

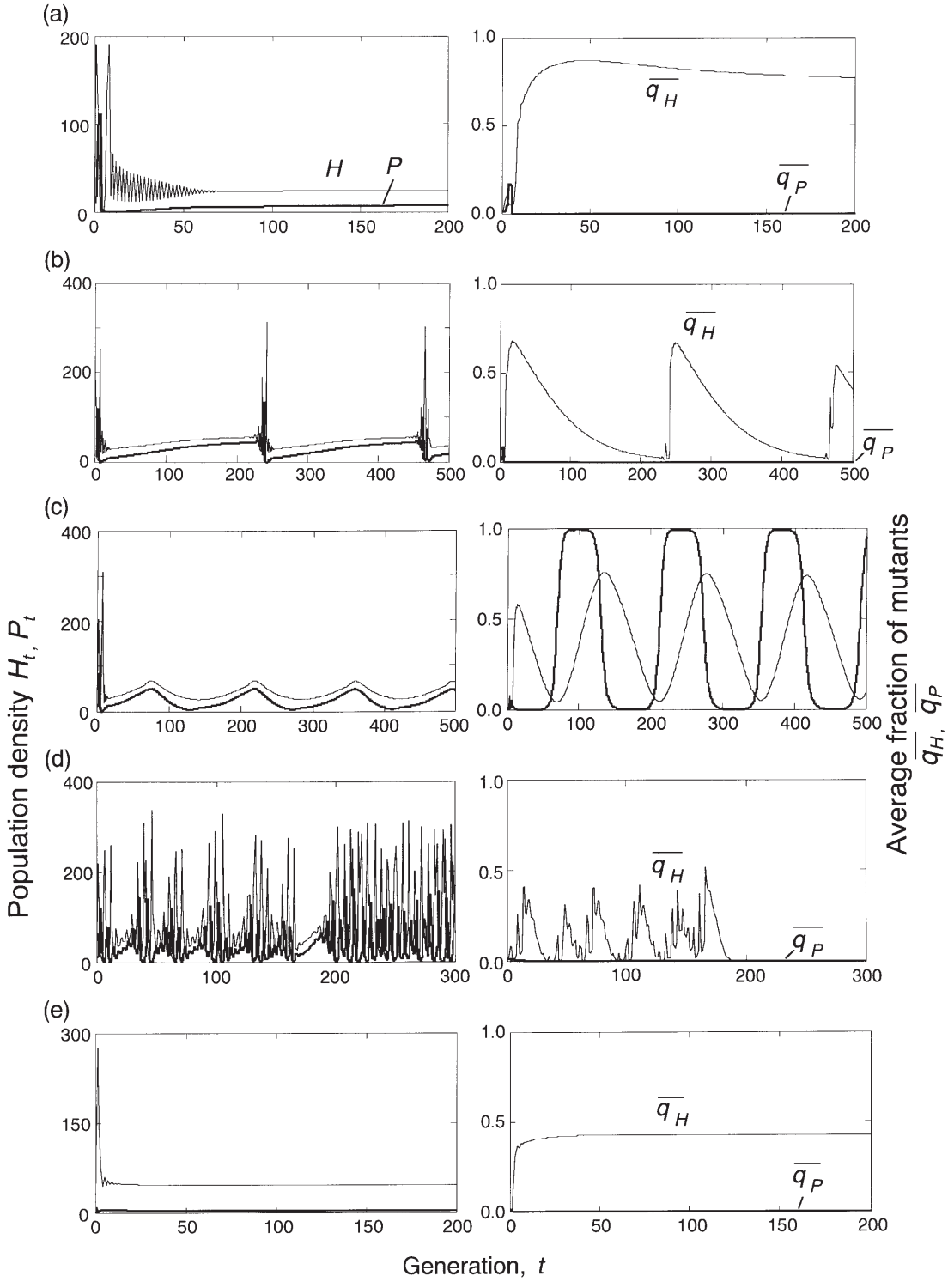

Fig. 4a–e. Examples of evolutionary population dynamics, at $\gamma_{21} = 0.5$ and (**a**) $d_H = 0.85$, stable persistence with H_2 invasion; (**b**) $d_H = 0.75$, persistence with unstable dynamics, H_2 invasion; (**c**) $d_H = 0.7$, persistence with limit cycles, H_2 and P_2 invasion; (**d**) $d_H = 0.55$, persistence with unstable dynamics, no invasion by mutants; and (**e**) $d_H = 0.1$, stable persistence with H_2 invasion

(Fig. 4b,c). For the parasitoid, the invasion of the mutant is highly difficult. Persistence is determined, to a large extent, by the resident host–resident parasitoid interaction (Fig. 3 in comparison with Fig. 2a). The invasion of host mutants, however, promotes the persistence of the interaction through its stronger self-regulation than the resident. Notice that the virulence levels for stable persistence are higher than the levels observed in the monomorphic (nonevolutionary) host–parasitoid interaction (Fig. 2d in comparison with 2a), because of the higher com-

petitive ability, or stronger self-regulation, of the mutant phenotype of the host. Examples of the coupled evolutionary and population dynamics at $\gamma_{21} = 0.5$ are shown in Fig. 4.

Effect of trade-offs on ecological and evolutionary stability

Trade-offs between life history characters are ubiquitous components of life histories, especially in host insects (Roff 1992). To address this, we examine the trade-offs in the host and then the parasitoid population.

The effect of a trade-off acting on the host is examined by removing it from the host (i.e., $d_H = 1$), while retaining it in the parasitoid. Exploration of all the possible d_P's allows us to conclude that the parasitoid is unable to persist (Fig. 5). As for the effect of a trade-off in the parasitoid, this is examined in the same way by removing it from the parasitoid (i.e., $d_P = 1$), while retaining it in the host. Although the persistence region changes only slightly, the mutant parasitoid always invades and wipes out the population in the region (Fig. 6 in comparison with Fig. 3; the area for invasion is not shown). Graphically explained, the persistence regions for resident host–mutant parasitoid now overlaps with that for resident host–resident parasitoid interaction (the persistence region in Fig. 2b now converges to that in Fig. 2a). A parameter region similar to the hatched area in Fig. 3 exists in which the mutant phenotype 2 of the host can invade (results not shown).

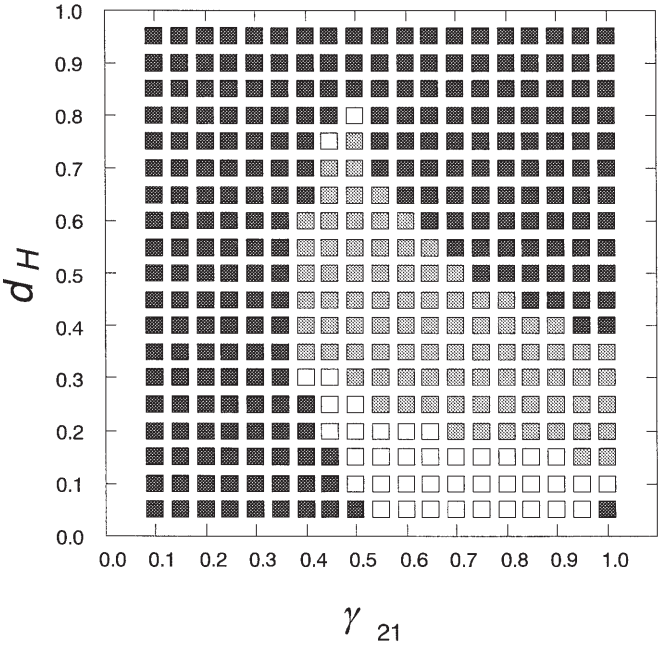

Fig. 6. Population persistence when removing the trade-off effect of competition on virulence in the parasitoid population, while retaining it in the host (i.e., $d_P = 1$, with competitive superiority of mutants over residents in both populations). The mutant parasitoid now always invades in the parameter region for persistence (i.e., *white* and *shaded squares*). *White squares*, stable persistence; *shaded squares*, persistence with unstable dynamics; *black squares*, extinction

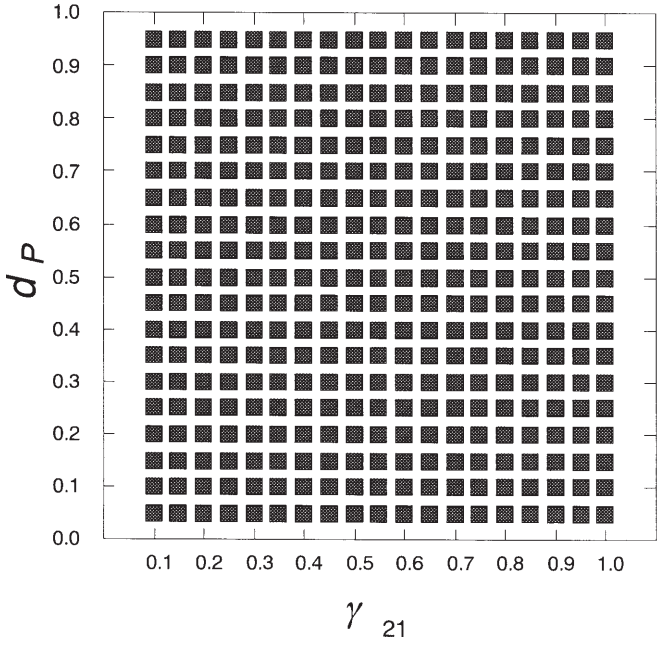

Fig. 5. Population persistence and invasibility of mutant phenotypes when removing the trade-off effect of competition on virulence in the host population, while retaining it in the parasitoid (i.e., $d_H = 1$,with competitive superiority of mutants compared to residents in both populations). In all combinations of two virulence parameters, the parasitoid is lost through extinction. This result shows that a trade-off acting in the host is stabilizing

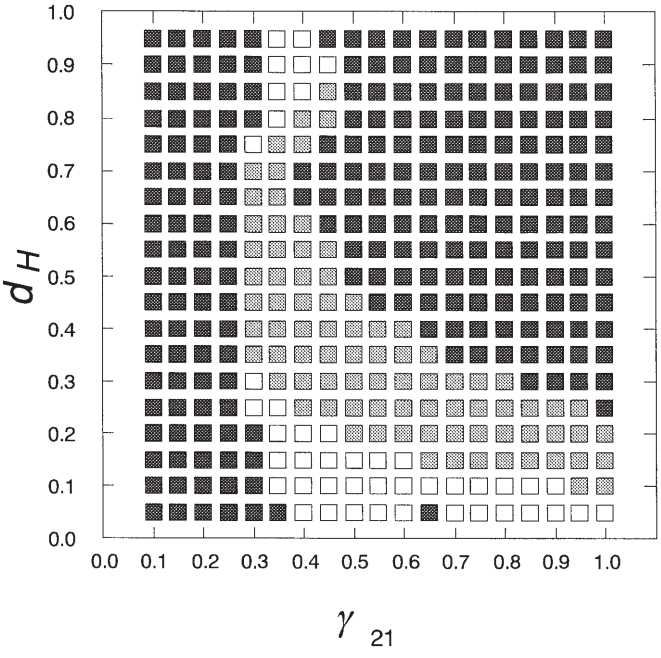

Fig. 7. Population persistence when a positive correlation exists between competition and virulence in the parasitoid population ($d_P = 1.5$), while retaining the trade-off (negative correlation) in the host. The mutant parasitoid always invades in the parameter region for persistence (i.e., *white* and *shaded squares*)

If the correlation between competition and virulence is positive, on the other hand, the region for persistence and stability shifts toward lower virulence to compensate the higher a' of the mutant parasitoid (Fig. 7). Again, the mutant parasitoid always invades and outcompetes the competitively inferior resident, and the region of persistence is determined mainly by resident host–mutant parasitoid interaction (Fig. 8b in comparison with Fig. 7).

The trade-off in the parasitoid is less influential to persistence than that in the host, and the polymorphism is quite rare under the present assumption. The polymorphism in the host, by contrast, emerges around the parameter boundaries for persistence–extinction and promotes population stability. It is clear from this analysis that both the searching efficiency of the adult female wasp and the survival of the juvenile parasitoid (or avoidance of encapsulation) are critical in determining the persistence of polymorphisms. Concomitantly, if juvenile parasitoid survival is influenced by host susceptibility and encapsulation ability (Kraaijeveld and Godfray 1997; Fellowes et al. 1998) then, as we have demonstrated, the coupling of the evolutionary and population dynamics between hosts and parasitoids cannot be neglected in understanding the evolution of parasitoid virulence.

Discussion

It is clear that the evolution of parasitoid virulence is tightly coupled to the population dynamics of the host–parasitoid interaction. Our result showing that genetic variability of the host promotes the stability of the host–parasitoid interaction is also supported by Doebeli (1997) and Holt and Hochberg (1997).

The present study contrasts trade-off effects between the hosts and parasitoids on population dynamics and persistence of the interaction; the trade-off in the host is more influential on persistence of the host and parasitoid interaction than that observed in the parasitoid. This finding could explain the frequent observation of resistance (and associated genetic variability) in the host (Henter 1995; Henter and Via 1995) and the trade-off relationship observed with other life history components (such as competition ability; Kraaijeveld and Godfray 1997). Recently, Doebeli (1997)

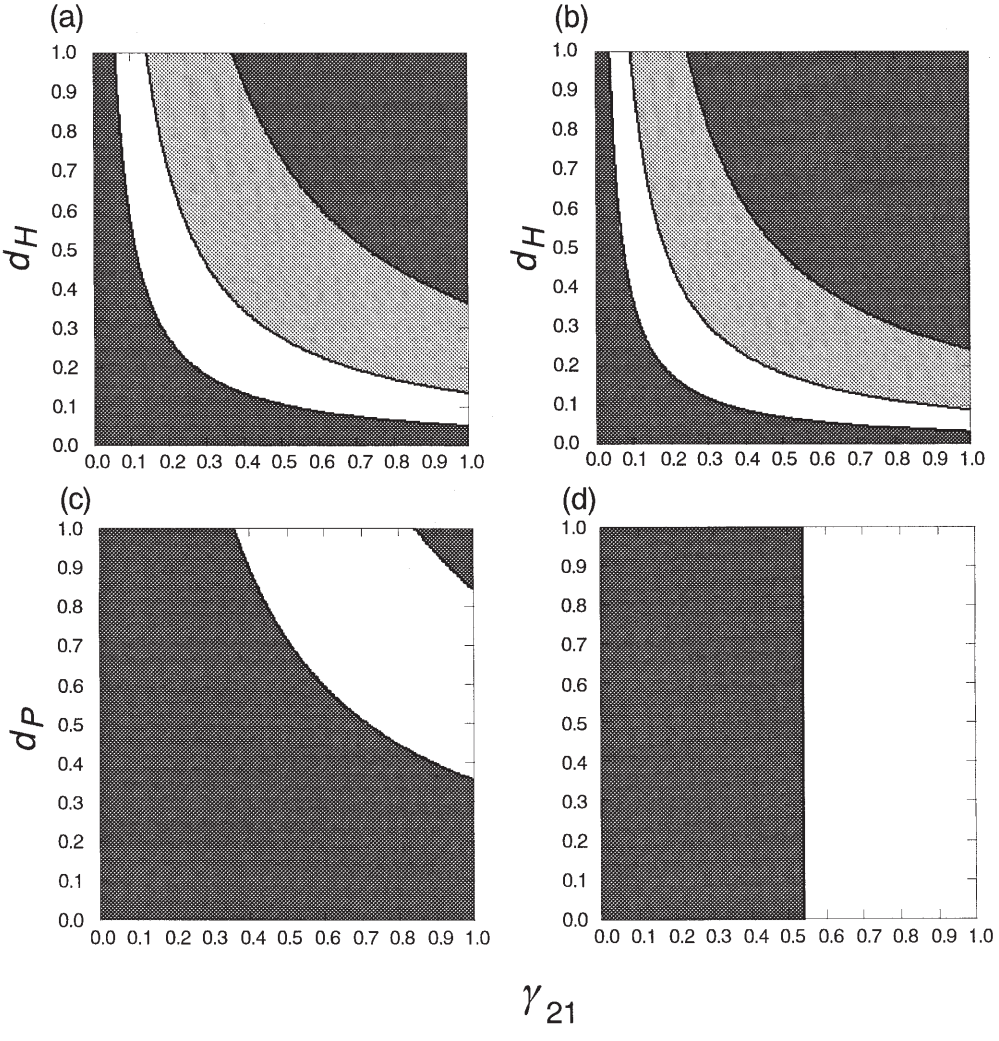

Fig. 8a–d. The effect of a positive correlation between virulence and competitive ability in the parasitoid, showing how this can influence the ecological dynamics of the monomorphic interactions of (**a**) resident host–resident parasitoid, (**b**) resident host–mutant parasitoid, (**c**) mutant host–mutant parasitoid, and (**d**) mutant host–resident parasitoid. *White area*, stable persistence; *shaded area*, persistence with unstable dynamics; *black area*, extinction

has demonstrated that genetic variation in hosts and parasitoids can promote the ecological persistence of the interaction. This persistence arises as particular host phenotypes suffer disproportionally high levels of attack. As persistence is promoted when host phenotypes shift to regions of parasitoid-free space, then this begs the question as to whether parasitoids are able to evolve different virulence strategies to match these host shifts.

This result presents an interesting dimension to the problem of biological control and evolution, initially raised by Holt and Hochberg (1997). If hosts are able to evolve resistance against attack from released natural enemies but the parasitoids do not show sufficient variability to counter these changes, then adequate control may be lost and the economic problem reoccurs. This in turn suggests that more complex release strategies may be required for adequate control. Recently it has been empirically demonstrated that resistance to attack against one parasitoid does not ensure ability to resist attack from a second parasitoid (Fellowes et al. 1998). It may be, therefore, that multiple release strategies of different parasitoids are advantageous to long-term pest control.

However generated, it is the variation in host susceptibility to attack that promotes the ecological persistence of otherwise unstable host–parasitoid interactions (Bailey et al. 1962; Chesson and Murdoch 1986; Pacala et al. 1990; Hassell et al. 1991). This variability in attack brought about by differences in host physiology (Kraaijeveld and Godfray 1997; Kraaijeveld et al. 1998), spatial (Hassell 1978; Bonsall and Hassell, this issue), or temporal (Godfray et al. 1994) distributions can have profound effects on the persistence, evolution, and coevolution of host–parasitoid assemblages.

Although considerable progress has been made in the physiology and genetics of host resistance (Fellowes et al. 1998; Kraaijeveld et al. 1998), little progress has been developed in understanding parasitoid virulence (but see Strand and Pech 1995). It is clear that an understanding of the evolution of virulence must be coupled with an understanding of the population dynamics of host–parasitoid interaction. Clearly, more experimental and theoretical work remains to be done to meet the challenge of determining how coevolutionary forces shape the assemblages, population dynamics, individual behaviors, and genetics of host–parasitoid interactions.

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Appendix

Fitnesses of the two host phenotypes in the absence of parasitoid are equivalent to the population multiplicative rate and

$$w_{H_1} = \frac{H_{1,t+1}}{H_{1,t}} = \gamma \cdot \exp\left(-\beta_1 \cdot H_{1,t} - \beta_2 \cdot H_{2,t}\right)$$
$$w_{H_2} = \frac{H_{2,t+1}}{H_{2,t}} = \gamma \cdot \exp\left(-\beta_2 \cdot H_{2,t}\right) \quad \text{(A1)}$$

From ESS requirement (Eq. 14), we find $H_1^* = H_2^* = 0$ and no positive solutions for H_1^* and H_2^*. Because $w_{H_1} < w_{H_2}$ always holds for positive densities, the competitively superior phenotype 2 outcompetes the other in a host-only population.