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# A model for the coevolution of resistance and virulence in coupled host–parasitoid interactions

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A coevolutionary model is developed of the interaction between a host and an internal parasitoid, where the outcome of parasitism depends upon the extent to which individual hosts invest in resistance mechanisms and individual parasitoids in countermeasures (virulence). The host and parasitoid are assumed to have coupled population dynamics (of Nicholson–Bailey form) and to be composed of a series of asexual clones with different levels of resistance and virulence. Investment in resistance and virulence mechanisms is assumed to be costly. The model has two main outcomes. First, if resistance is relatively costly compared to virulence, the host may be selected not to invest in resistance mechanisms despite parasitoid investment in virulence, in effect trading off the risks of parasitism against the savings in costs. A number of cases which appear to correspond to this result have been reported. Second, for most other feasible parameter values, an arms race occurs between host and parasitoid, until effective resistance becomes so costly that the host abandons defence. This abandonment is followed by a reduction in parasitoid virulence and the cycle begins again. These cycles may explain reports of persistent additive genetic variation in resistance and virulence, and may also contribute towards population dynamic stability.

**Keywords:** coevolution; Nicholson–Bailey; parasitoid; population dynamics; resistance; virulence

## 1. INTRODUCTION

Parasitoids are often important sources of host mortality and hence are likely to have a significant effect on host population dynamics, as well as exerting strong selection pressures on the host to evolve adaptations to reduce or survive parasitoid attack (Hassell 1978; Godfray 1994). Changes in host population densities or defensive strategies will themselves influence parasitoid population dynamics, and also lead to selection on the parasitoid to counter any host defences. Thus, both the population and evolutionary dynamics of hosts and parasitoids will often be coupled, and changes in population density may influence reciprocal selection pressures and vice versa. Because of their importance in agriculture and their abundance in most terrestrial communities, the population dynamic interactions between parasitoids and their hosts have been intensively studied for nearly a century. However, it is only relatively recently that the possibility of joint evolutionary and ecological dynamics has begun to be explored. Here we describe and analyse a model of host resistance and parasitoid virulence that is appropriate for endoparasitoids that must withstand their hosts' cellular immune defences. The assumptions of the model are based on recent experimental work, chiefly with *Drosophila* and its endoparasitoids. The model aims to predict the outcome of coevolution in a host–parasitoid interaction with coupled population dynamics.

Endoparasitoids lay their eggs in the bodies of other insects, which their larvae eventually kill. After oviposition, the egg or larva often remains dormant while the host grows to full size. During this period the juvenile parasitoid is susceptible to host immunological attack, in particular cellular encapsulation, and different parasitoids have evolved a variety of adaptations to disable host defences (Salt 1970; Beckage *et al.* 1993; Strand & Pech 1995; Carton & Nappi 1997). Substantial additive genetic variation in host resistance has been found on a number of occasions (reviewed by Kraaijeveld *et al.* 1998). Evidence for genetic variation in parasitoid virulence is more mixed: it has been found in an aphid parasitoid (Henter 1995) and in isofemale line studies of *Drosophila* parasitoids (though additive and non-additive components cannot be distinguished) (Carton *et al.* 1989; Carton & Nappi 1991) but not in selection studies on *Drosophila* parasitoids (Kraaijeveld & Godfray 1999). Costs of resistance have been found—*Drosophila melanogaster* selected for higher resistance suffer from reduced larval competitive ability when food is scarce (Kraaijeveld & Godfray 1997; Fellowes *et al.* 1998a)—but there have as yet been no experiments designed to detect costs of parasitoid virulence. Though more studies are needed, the data so far are consistent with resistance and virulence being graded traits (Kraaijeveld *et al.* 1998): by this we mean that if resistance is denoted by  $x$  and virulence by  $y$  then the probability of the host's survival increases monotonically with  $x - y$ . Resistance and virulence in parasitoids is thus more like running speed in predators and prey than the

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gene-for-gene resistance mechanisms found in many plant diseases. In cross-species studies, some hosts appear never to defend themselves successfully against any of their native parasitoids (Kraaijeveld & van der Wel 1994). Finally, clines and mosaics in host resistance and parasitoid virulence have been identified at continental geographical scales (Kraaijeveld & van Alphen 1994, 1995; Kraaijeveld & Godfray 1999).

Many hosts and parasitoids have discrete synchronized generations and this has led to most host–parasitoid population models being phrased as coupled difference equations. The basic host–parasitoid population model assumes that parasitoid attack is random and that the proportion of hosts escaping parasitism in each generation is the zero term of a Poisson distribution with mean proportional to parasitoid density (the Nicholson–Bailey (1935) model). This model always shows diverging oscillations due to cycles where host overexploitation and subsequent decline is followed by a crash in parasitoid numbers, which allows the host population to recover (Hassell 1978). A stable interaction results if the proportion of hosts escaping parasitism is the zero term of a sufficiently overdispersed probability distribution, such as the negative binomial (May 1978).

A wide variety of biological processes can give rise to this type of stabilizing heterogeneity in the risk of parasitoid attack (Hassell & Pacala 1990). One such mechanism is variation in the ability of hosts to defend themselves. Bailey *et al.* (1962) implicitly, and Hassell & Anderson (1984) and Godfray & Hassell (1991) explicitly, demonstrated that sufficient, constant variation in host resistance could stabilize the Nicholson–Bailey model. However, the assumption of constant variation is problematic as the parasitoid will select strongly for high resistance, and it is therefore hard to see how sufficient non-genetic variation in encapsulation ability can be maintained. Models that allow both trait frequencies and population densities to vary are thus needed. A few models of this type have been studied (Hochberg & Holt 1995; Hochberg 1997; Doebeli 1997) and we compare our results with these studies in §4. Holt & Hochberg (1997) discuss whether coevolutionary interactions influence the success of biological control, while other models of evolutionary and population dynamics in host–parasitoid systems involving patch use and diapause strategies are described in van Baalen & Sabelis (1993) and Ringel *et al.* (1998), respectively.

## 2. MODEL

### (a) *Basic assumptions*

Assume that the host's ability to resist parasitoid attack, and the parasitoid's ability to counteract host defences, can each be described by one-dimensional variables:  $x$  (host resistance) and  $y$  (parasitoid virulence). We allow the host and parasitoid populations to consist of multiple asexual genotypes indexed by  $x$  and  $y$ . The probability that an egg of a parasitoid with genotype  $y$  is encapsulated in a host of genotype  $x$  is  $\eta(x - y)$ , where  $\eta(z)$  is a monotonically increasing function of  $z$  with  $\eta(-\infty) = 0$  and  $\eta(\infty) = 1$ . Thus the probability that the egg survives is a function of the relative difference between host resistance and parasitoid virulence. Throughout the paper we shall assume encapsulation probability to be of the form

$$\eta(z) = \frac{1}{1 + e^{-2Az}}. \quad (1)$$

When  $x = y$ , the host and parasitoid have identical chances of surviving:  $\eta(0) = 0.5$ . When  $x > y$ , the host has a better chance of surviving than the parasitoid, and the reverse is true when  $y$  is greater than  $x$ . The parameter  $A$  describes how steeply the probabilities of host and parasitoid survival change as the difference between  $x$  and  $y$  increases.

We assume that there are costs to both host resistance and parasitoid defence. The host may experience two types of costs associated with parasitoid resistance: those that are experienced irrespective of whether parasitoid attack occurs, and those that are incurred only after parasitism. The first type reflects the costs of maintaining the cellular and biochemical machinery required to mount a successful parasitoid attack, while the second includes the resources expended in actually destroying the parasitoid. Hosts that successfully defend themselves from parasitism do suffer some reduction in fitness compared with unparasitized hosts (Carton & David 1983; Fellowes *et al.* 1998b), but the difference is not great and for simplicity we shall assume that host resistance is efficient and that the costs are all of the first type. Specifically, we let the fecundity of the adult host be  $Ga(x)$  where  $G$  is the fecundity of a host that makes no investment in resistance and  $a(x)$  is a monotonically decreasing function of  $x$  with  $a(0) = 1$ . The specific function we use in simulations is  $a(x) = \exp(-c_H x)$ .

In contrast to host resistance, there is one type of cost of parasitoid virulence which is always incurred. We assume that the probability of parasitoid recruitment to the adult population is  $b(y)$ , a decreasing function of  $y$ . The specific function we use in simulations is  $b(y) = \exp(-c_P y)$ . This form of costs would occur if investment in increased parasitoid virulence decreased the probability of surviving pupation. An alternative, but more complicated, way to model the costs of virulence would be to reduce the searching efficiency or fecundity of more virulent genotypes.

### (b) *Population dynamics*

We consider hosts and parasitoids that have discrete and synchronized generations and model their population dynamics using the generalized form of the Nicholson–Bailey equations,

$$\begin{aligned} N_{t+1} &= GN_t F_t, \\ P_{t+1} &= N_t(1 - F_t). \end{aligned} \quad (2)$$

$N_t$  and  $P_t$  are the densities of hosts and parasitoids in generation  $t$ ;  $G$  is fecundity as defined above; and  $F_t$  is the probability that a host escapes parasitism in the current generation. If parasitoid search is random, and if the average number of parasitoid encounters per host is  $\beta P_t$  (where  $\beta$  is a measure of parasitoid searching efficiency), then  $F_t$  is the zero term of a Poisson distribution,  $F_t = \exp(-\beta P_t)$ , and equation (2) predicts diverging oscillations. Alternatively, as discussed in §1,  $F_t$  can be the zero term of the negative binomial distribution (May 1978; see also Chesson & Murdoch 1986), in which case,  $F_t = (1 + \beta P_t/k)^{-k}$ , where  $k$  is an index of contagion. Equation (2) now gives rise to a stable interaction for  $k < 1$  and an unstable interaction for  $k > 1$ . We have assumed throughout this paper that parasitoids have short handling times and are not egg limited.

Because we have assumed asexual reproduction, the dynamics of an ensemble of host and parasitoid genotypes can be modelled by modified sets of difference equations based on the Nicholson–Bailey model. Let  $N_t(x)$  and  $P_t(y)$  be the densities of hosts and

parasitoids with genotypes  $x$  and  $y$ , respectively, in generation  $t$ . Then

$$\begin{aligned} N_{t+1}(x) &= Ga(x)N_t(x) \left\{ F_t + (1 - F_t) \int_0^\infty \eta(x-y) Q_t(y) dy \right\}, \\ P_{t+1}(y) &= b(y) \int_0^\infty N_t(x) \{1 - \eta(x-y)\} (1 - F_t) Q_t(y) dx, \end{aligned} \quad (3)$$

where  $Q_t(y) = P_t(y)/\bar{P}_t$  is the relative frequency of parasitoids of genotype  $y$  and  $\bar{P}_t$  is the total parasitoid density in generation  $t$ . The first equation states that of the  $(1 - F_t)$  hosts of genotype  $x$  that are parasitized, a fraction  $Q_t(y)$  of the attacks are by parasitoids of genotype  $y$  and hence the hosts survive with probability  $\eta(x-y)$ . An implicit assumption here is that even if the host is encountered by more than one parasitoid, only a single egg is laid, and that no particular genotype is favoured at this stage. This assumption is not unreasonable as many parasitoids are able to detect whether a host has been previously attacked and so avoid laying further eggs (avoidance of superparasitism; van Alphen & Visser 1990; Godfray 1994). The second equation describes how those parasitoids of genotype  $y$  that survive in hosts of genotype  $x$  (with probability  $1 - \eta(x-y)$ ) go on to complete development (with probability  $b(y)$ ) and form the next generation of parasitoids.

### (c) Evolutionary stability

Host genotype  $x$  persists at evolutionary and population dynamic equilibrium if its long-term geometric average growth rate ( $\rho_N(x)$ ) is zero,

$$\rho_N(x) = E_t \log \left[ Ga(x) \left\{ F_t + (1 - F_t) \int_0^\infty \eta(x-y) Q_t(y) dy \right\} \right] = 0, \quad (4)$$

where  $E_t$  denotes the long-term average. Similarly, for parasitoid genotype  $y$  to persist

$$\rho_P(y) = E_t \log \left[ b(y) \int_0^\infty N_t(x) \{1 - \eta(x-y)\} (1 - F_t) / \bar{P}_t dx \right] = 0. \quad (5)$$

On the other hand, a rare mutant genotype,  $\hat{x}$  or  $\hat{y}$  will only invade if the marginal logarithmic growth rate is greater than zero,

$$\lambda_N(\hat{x}) = \lim_{N_t(\hat{x}) \rightarrow 0} \rho_N(\hat{x}) > 0, \quad (6)$$

for the host and

$$\lambda_P(\hat{y}) = \lim_{P_t(\hat{y}) \rightarrow 0} \rho_P(\hat{y}) > 0, \quad (7)$$

for the parasitoid. At equilibrium, we expect to see only hosts belonging to the set  $S_N$  where  $S_N = \{x: \rho_N(x) = 0\}$  and  $\lambda_N(x) < 0$  for  $x \notin S_N$ , and parasitoids belonging to the set  $S_P$  where  $S_P = \{y: \rho_P(y) = 0\}$  and  $\lambda_P(y) < 0$  for  $y \notin S_P$ . Note that the condition does not require that the host or parasitoid population converges to a static point equilibrium at the evolutionarily stable state—indeed, we will show below that the evolutionarily stable population often fluctuates in total density and genotype frequency.

### (d) Parameter reduction

The parameters in the model are the costs of resistance and virulence ( $c_H$  and  $c_P$ ), host fecundity ( $G$ ), the factor that scales how a difference in virulence and resistance affects host and

parasitoid survival ( $A$ ), parasitoid searching ability ( $\beta$ ) and the heterogeneity in parasitoid attack ( $k$ , the contagion parameter in the negative binomial distribution). The number of parameters can be reduced by the following rescaling:  $C_H = c_H/2A$ ;  $C_P = c_P/2A$ ;  $X = 2Ax$ ;  $Y = 2Ay$ ;  $p = P/\beta$ ;  $yn = N/\beta$ .

### (e) Numerical simulation

Only limited analytical results are possible for the full model, and its non-equilibrium behaviour was explored by simulation. We assumed that there were 51 host or parasitoid genotypes with resistance or virulence that varied in the range  $0 \leq \{X, Y\} \leq 12$ . Mutation was modelled by assuming a constant influx of all genotypes at a rate of 0.0001 per generation.

## 3. RESULTS

We begin by examining the evolutionary dynamics of resistance and virulence in situations where the underlying population dynamics without genetic variation are stable ( $k=0.8$  unless stated otherwise), returning at the end to consider if evolutionary dynamics can stabilize otherwise unstable population dynamics. Simulation showed that the model displayed two main types of persistent dynamics: (i) a stable genetic and population equilibrium where the majority of hosts do not invest in resistance and most parasitoids have a fixed level of virulence (deviations from these strategies by a minority of the population are a result of mutation); and (ii) non-equilibrium behaviour where the levels of resistance and virulence change over time, typically moving amongst a series of discrete levels. We discuss each of these in turn and also some other dynamic behaviour that is less frequently exhibited by the model.

### (a) Stable equilibrium with no host resistance

There is an asymmetry in the interaction because all parasitoids have to develop at the expense of a host, whereas some hosts will escape parasitoid attack. Thus abandoning investment in virulence is rarely advantageous to parasitoids, but conditions will arise when natural selection favours the host investing as little as possible in resistance, in effect trading-off the costs of defence against the risk of parasitoid attack.

Analysis of the model helps show the conditions under which the no-resistance evolutionarily stable strategy (ESS) occurs (details in Appendix A). First, we can calculate the optimum level of virulence for the parasitoid,  $Y^*$ , given that the host resistance trait  $X=0$ ,

$$Y^* = \log(1/C_P - 1), \quad (8)$$

(equation (A4) with  $X=0$ ). When the host resistance trait is zero, optimum parasitoid virulence is determined solely by the scaled costs of resistance ( $C_P = c_P/2A$ , recall  $A$  measures how sharply differences between the traits  $x$  and  $y$  influence the outcome of parasitism).

Given that we know  $Y^*$ , we can determine when  $X=0$  is stable. A rare mutant,  $\hat{X} \neq 0$  spreads if  $\lambda_N(X) > 0$ , which a little algebra shows is equivalent to

$$\max_{\hat{X} > 0} \left( \frac{e^{-C_H \hat{X}} (1 + C_P (e^{\hat{X}} - 1) G)}{1 + C_P (e^{\hat{X}} - 1)} \right) < 1. \quad (9)$$

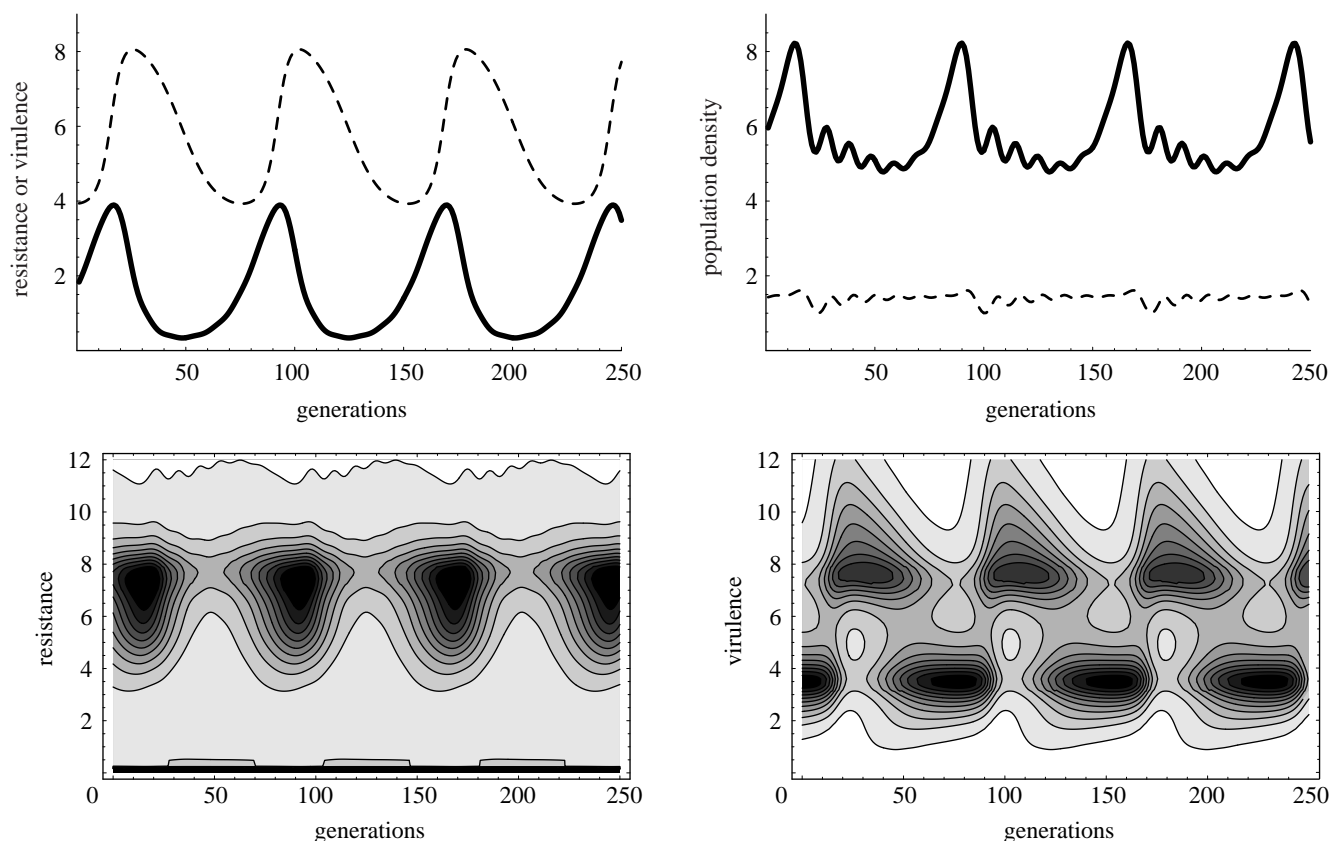


Figure 1. An example of the joint host–parasitoid population and evolutionary dynamics. (a) Average levels of host resistance ( $X$ , solid line) and parasitoid virulence ( $Y$ , broken line). (b) Population densities (host, solid line; parasitoid, broken line). Density plots of the distribution of host (c) and parasitoid (d) genotypes (dark colours are higher density, ten contours are equally spaced over the square root of frequency). In this simulation  $C_p = C_H = 0.1$ ;  $G = 1.65$ ;  $k = 0.8$ . The last 250 generations of a 1000-generation simulation are shown.

This condition is more likely to be met when  $C_H$  is large and when  $G$  is small. The dependence on  $C_p$  is more complicated, but for biologically realistic values of  $C_H$  and  $G$ , the condition is more likely to be met when  $C_p$  is small. In many circumstances, this global stability criterion can be replaced by a local stability criterion: the condition for a mutant to spread the resistance of which is just greater than zero,

$$G < 1 + \frac{C_H}{C_p}. \quad (10)$$

Here, the dependence on the three parameters is very straightforward. Finally, for a demographic equilibrium to occur with no host resistance, the probability of parasitoid attack,  $F$ , must be greater than zero. Substituting equation (8) into equation (A5), we obtain a further condition for the no-resistance ESS

$$G < 1/C_p.$$

These results have clear biological meaning. The absence of investment in defence is more likely to occur when resistance is expensive. High fecundity implies a high equilibrium probability of parasitism (in a system with coupled population dynamics) and investing in defences is more worthwhile when risks of parasitism are high. Low  $C_p$  will favour high levels of virulence

(equation (8)) and hence the host has to make greater investment in resistance for effective defence (in effect, resistance is more costly). Finally, if the costs of parasitism are too high relative to host fecundity, the parasitoid cannot regulate the host population and a population dynamic equilibrium is impossible.

#### (b) Cycles in resistance and virulence

Cycles in both the population and frequency domains were found for a wide range of parameter values. Two examples are shown in figures 1 and 2. In figure 1, hosts oscillate between no resistance and a mixture of no resistance and moderately high level of resistance, while parasitoids oscillate between medium and high levels of virulence. In figure 2, both host resistance and parasitoid virulence increase in a more continuous manner over time, until a level is reached when the host abandons investment in resistance; this is followed by a sharp reduction in parasitoid virulence and the cycle begins again. For other parameters, intermediate behaviours are shown; for example hosts and parasitoids switch between three or more discrete levels of resistance and parasitism.

Analytical treatment of the full model is impossible but its dynamics can be understood by making a ‘monomorphic approximation’. Assume that the host population is monomorphic for resistance and calculate the optimum parasitoid virulence; similarly assume that the parasitoid

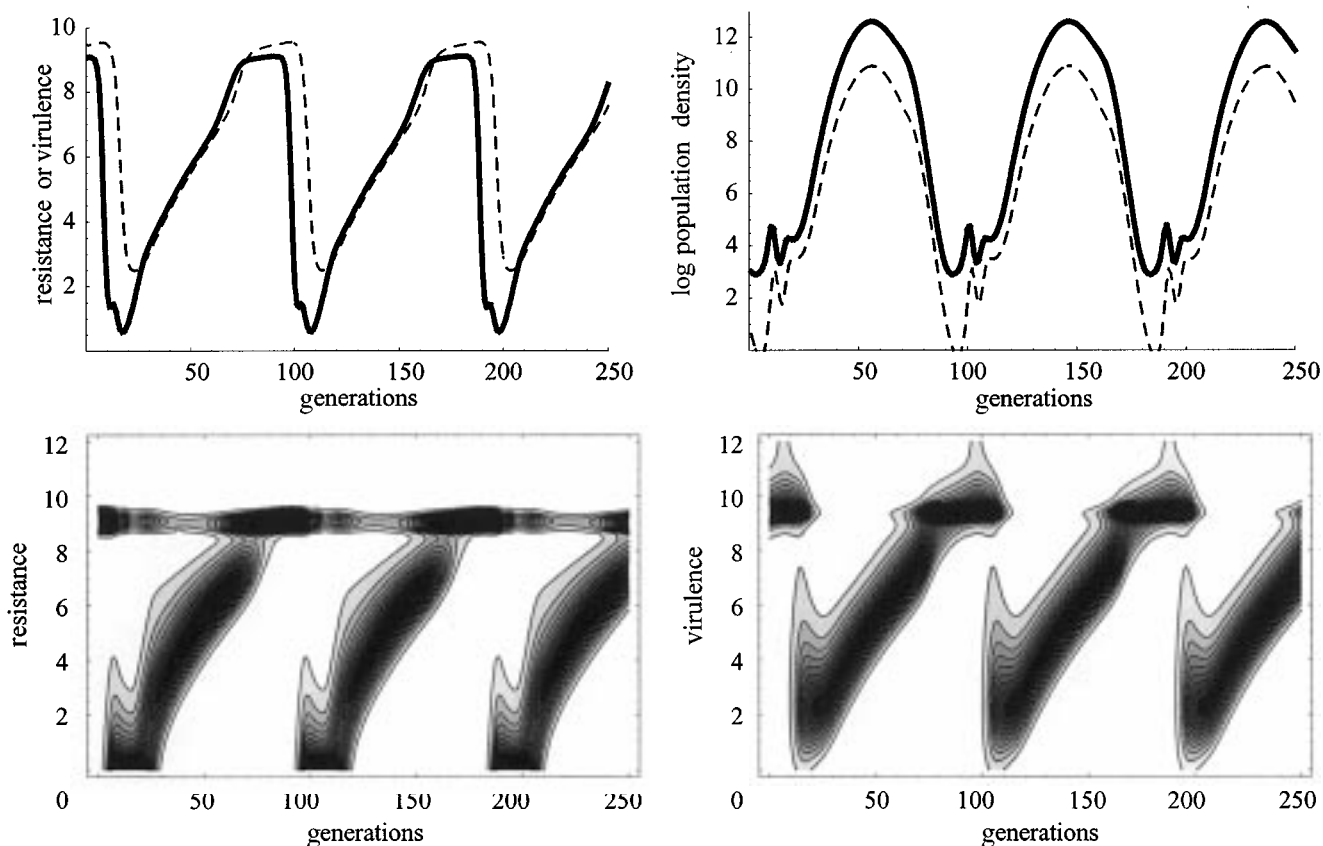


Figure 2. As figure 1 but now resistance and virulence are more costly, and fecundity higher:  $C_p = C_H = 0.3$ ;  $G = 5$ .

population is monomorphic for virulence and calculate the optimum host resistance. By plotting both functions in virulence–resistance space, the evolutionary dynamics can be predicted. The results closely match the dynamics of the full polymorphic system.

Figure 3a shows the monomorphic approximation for the same parameters as figure 1. The derivation of the various curves is given in Appendix A. Line A is the optimum parasitoid virulence as a function of host resistance. For the functions used here, optimum virulence is simply proportional to host resistance. Line B is optimum host resistance as a function of parasitoid virulence. This function is more complicated and consists of a locally stable optimum (solid part of B) linked by a fitness minimum (dotted part of B) with the part of the  $y$ -axis above the intersection with the fitness minimum which represents the region where the no-resistance ESS is locally (or possibly globally) stable. The solid part of line B is divided into two by line C. Below C, no demographic equilibrium is possible (because  $F^*$ , equilibrium parasitism,  $< 0$ ) and optimum resistance is calculated for an exponentially increasing host population. Above C, an equilibrium is possible and the optimum resistance is calculated under equilibrium conditions. The host resistance function is thus cusp-shaped, with an area of bistability where the behaviour of the system is influenced by the relative magnitude of the two fitness peaks, and the types of mutation available to the population.

Begin with the situation where hosts do not invest in resistance ( $X = 0$ ). The intercept of line A with the  $y$ -axis on figure 3a gives the optimum parasitoid virulence (point  $a$ ). Because point  $a$  lies to the left of the fitness-

minima part of line B, this state of the system is locally stable. However, the fitness equilibrium corresponding to the solid part of line B is greater and when a suitable mutant genotype arises, host resistance increases and the system moves to point  $b$ . In doing this, the system leaves the region of parameter space where a demographic equilibrium is possible and host population densities increase. The optimum parasitoid response to this increased host resistance is higher virulence and the system moves to point  $c$ . Now, however, parasitoid virulence is so high that hosts are selected to abandon resistance and the system moves to point  $d$ . With host resistance now zero, parasitoids are selected to reduce investment in virulence and the system returns to point  $a$ . The monomorphic approximation thus predicts resistance should oscillate between zero and the level represented by points  $b$  and  $c$ , while virulence should oscillate between the levels of points  $a$  and  $b$ , and  $c$  and  $d$ . The full model shows almost exactly this behaviour, though the host population does not have time to move completely from resistance levels of 0 to  $b/c$  before the parasitoid response favours no resistance again.

Figure 3b shows the equivalent monomorphic approximation for the parameters in figure 2. The pattern is similar, except now resistance and virulence move between four levels. In figure 2, the first three levels are not as discrete as in the previous example. The reason for this is that the state of the system when host resistance is zero is not locally stable (note the position of the fitness minima) and hence a range of mutants invade simultaneously. The third host resistance level is locally stable (the fitness minima lies to the right of the ESS) and hence there is a discrete jump between levels three and four.

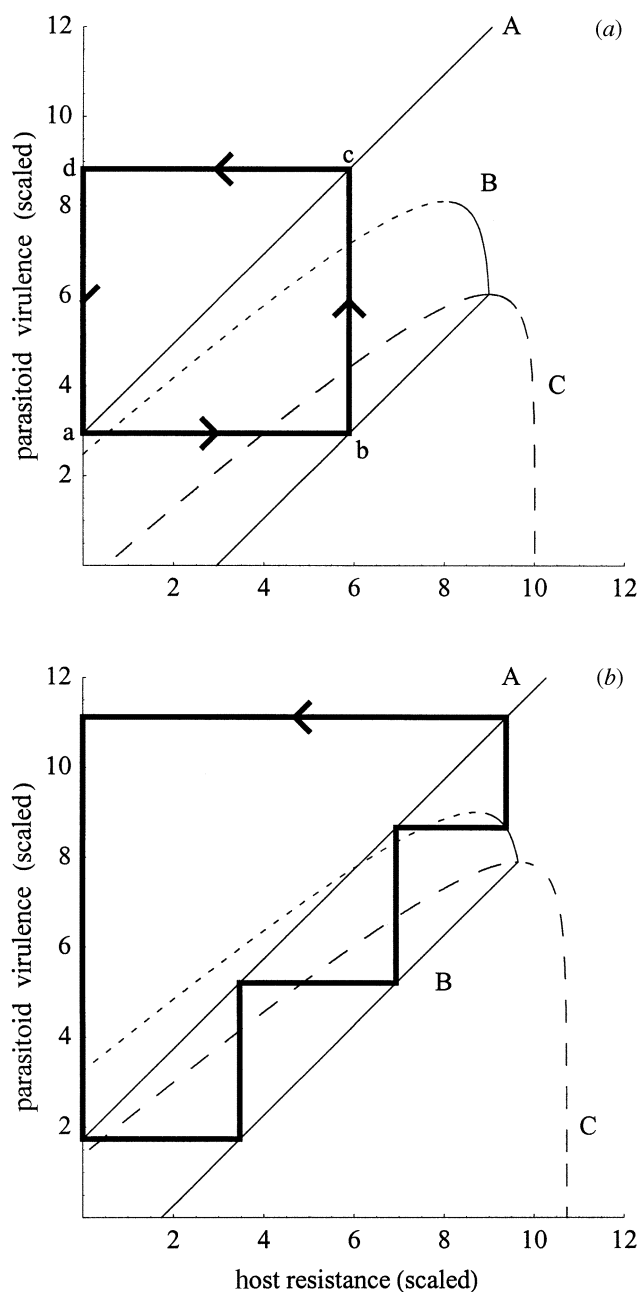


Figure 3. Monomorphic approximations to the simulations displayed in figure 1*a* and figure 2*b*. The axes of the graphs are scaled host resistance ( $X$ ) and parasitoid virulence ( $Y$ ). As explained more fully in the text, line A represents optimum parasitoid virulence as a function of host resistance and line B optimum host resistance as a function of parasitoid virulence. Line B consists of two sections: the solid part in the interior of the graph, linked by a dashed line representing a fitness minima with the part of the  $y$ -axis above the intersection with the minima. Above line C a demographic equilibrium exists but below it there is none, which affects the calculation of the optimum host response. Assuming the population is always monomorphic, the evolutionary trajectory through resistance–virulence space can be predicted (heavy lines with arrows indicate the direction of evolution; in (a) in the sequence a, b, c, d).

**(c) Other coevolutionary outcomes**

Cycles in resistance and virulence are the dominant behaviour shown by the model for low to moderate costs of resistance and virulence, outside the region of parameter space where hosts are selected not to invest in

resistance. Especially when costs are very high, other evolutionary results are possible, though often these lead to unstable population dynamics. (i) When both costs are very high (in our model  $C_p > 0.5$  and  $C_H > 0.5$ ) neither investment in resistance nor virulence is favoured ( $X=Y=0$ ). (ii) When  $C_p$  is very high and  $C_H$  intermediate, a no virulence but positive resistance ESS can occur, although typically the parasitoid is unable to regulate the host population, which increases exponentially. (iii) For a small range of parameter values (in our model near the region where  $C_H+C_p=1$ ) an internal ESS with both virulence and resistance greater than zero is a locally stable ESS. We have obtained this ESS in simulations, but typically the basin of attraction of the ESS is small and the system normally displays coevolutionary cycles.

**(d) Population dynamics**

Do the coevolutionary dynamics shown here contribute to population stability and can they stabilize the Nicholson–Bailey model? This question was not the main focus of our study but we briefly summarize the results of our simulations. The ESS in which the host abandons investment in resistance requires stable population dynamics (in our model  $k < 1$ ) for it to be observed. This is expected since at the ESS there is no heterogeneity in risk of parasitism. Persistent coevolutionary cycles can be observed for  $k > 1$  though for our standard assumptions about mutation the system was not persistent as  $k \rightarrow \infty$  (the Nicholson–Bailey limit).

**4. DISCUSSION**

Host–parasitoid interactions are attractive systems, both theoretically and experimentally, to study coevolution between species with coupled population dynamics. The aim of our work has been to explore the coevolutionary dynamics between the host immune response to endoparasitoid attack and the countermeasures of the parasitoid, an area that has been the subject of intense experimental study in the last ten years. Our main findings are that coevolution in coupled host–parasitoid systems can lead to two main types of dynamic behaviour. First, a stable ESS occurs in which the host does not invest in resistance but the parasitoid invests in virulence mechanisms. This ESS is found when costs of resistance are relatively high and fecundity relatively low. In these circumstances, the host is selected to risk parasitoid mortality rather than pay the heavy costs of effective defence. At this ESS, both the host and parasitoid are genetically monomorphic and hence the evolutionary dynamics do not contribute towards population dynamic stability. The second behaviour is coevolutionary cycles, where the host and parasitoid engage in an arms race until effective resistance becomes so costly that the host is selected to abandon resistance, followed by a reduction in parasitoid virulence, so resetting the cycle. These coevolutionary cycles can contribute to population stability.

Several studies have examined the potential for coevolutionary and population dynamic interactions between parasitoids and their hosts, while a larger number of workers have examined equivalent interactions between predators and prey, and pathogens and hosts. Seger (1992) has provided a useful classification of these types of

coevolutionary model. First, models can be distinguished by their genetic assumptions; specifically by whether they assume (i) a fixed level of genetic variation, with population interactions determined by trait means, or (ii) arbitrary levels of genetic variation with interactions specified between individual genotypes. Models of the first type are typically analysed using quantitative genetic techniques (Lande 1976). Hochberg & Holt's (1995) model of the evolution of a refuge in a host–parasitoid context is of this form and earlier verbal models by Pimentel (1961) embody this type of assumption. Evolutionary cycles can arise if reciprocal selection leads to higher resistance and virulence, and if as a consequence parasitoid populations then decline, selection for costly resistance is relaxed. Studies of predator–prey coevolution within a quantitative genetic (Rosenzweig *et al.* 1987; Abrams 1992; Saloniemi 1993; Matsuda & Abrams 1994; and see especially Abrams & Matsuda 1997) or related framework (Dieckmann *et al.* 1995) show that other types of evolutionary cycles are possible for different assumptions about numerical and functional responses.

Models that describe interactions between specific resource and consumer genotypes can be classified further by whether the interactions are matched or graded. In the former case, the fitness of a consumer genotype is a function of how well it matches the resource genotype. Gene-for-gene mechanisms are of this type as are, in the terminology of this paper, host–parasitoid interactions where the probability of host survival is a function of  $|x - y|$ . In a graded interaction, resource and consumer genotypes can be ranked along a single axis and higher values increase the probability of survival. Running speed in a predator–prey interaction is an example of this type of interaction, as are host–parasitoid interactions where the probability of host survival is a function of  $x - y$ . The population genetics of matching genotypes (particularly gene-for-gene interactions) have been intensively investigated in the host–pathogen literature (Anderson & May 1982; May & Anderson 1983; Frank 1993; 1994a; Haraguchi & Sasaki 1996; Ebert & Hamilton 1996; and reviewed by Seger 1992), but graded interactions have received less attention (Seger 1992; Frank 1994b). The only graded host–parasitoid model that we know of is Hochberg's (1997), though non-equilibrium behaviour in this model has yet to be studied.

Of previous host–parasitoid models, our work is most closely related to Doebeli's (1997), but it differs in that while we assumed graded interactions between host and parasitoid genotypes, Doebeli modelled matching interactions. Both models can predict evolutionary cycles, though the underlying biological mechanisms are very different. In Doebeli's basic model, without costs, parasitoids 'chase' hosts towards one end of the linear trait space, the hosts then 'jumping', by mutation and recombination, over the parasitoid which then chases the host to the opposite end of the trait space. Adding costs does not destroy the cycles but makes them asymmetrical (the host and parasitoid spending less time in costly regions of parameter space). Thus, in this case, hosts seek areas of trait space currently free from the risk of parasitism (enemy-free space) while in our case an arms race is followed by host 'surrender'. A further difference between the two models is that, with a graded interaction, an ESS is possible at which

hosts do not invest in a resistance mechanism. Interestingly, in a model of graded host–parasite evolution with very different population dynamic assumptions, Frank (1994b) found regions of parameters in which hosts did not invest in defence, as well as regions of persistent cycles that we suspect share a common biological mechanism with ours. A third difference between our model and Doebeli's is that we assumed clonal genetics while Doebeli explicitly modelled a diploid sexual system. Doebeli's approach is more realistic, though analytical simplifications are far harder to obtain. We found that the frequency and presence of evolutionary cycles depended quite critically on mutation rates, indicating that a more explicit representation of the generation of genetic variation will be needed to make the model more realistic. The genetic cycles in Doebeli's model make a greater contribution to population stability than those in ours, an effect that is due to the higher rate at which genetic variation is generated through sex and recombination, and possibly also to the greater propensity for evolutionary cycles with matching rather than graded interactions.

Our models are very simple, and comparisons with experimental data must be made with caution, but some of our results do match observed patterns in nature. First, there are many host–parasitoid systems in which the host appears to have no resistance mechanisms to its main parasitoid, while the parasitoid invests in overcoming host defences (as judged by its performance on other species of hosts). The best studied example is *Drosophila subobscura*, a northern European species that invariably succumbs to its main parasitoid *Asobara tabida* (Kraaijeveld & van der Wel 1994). Clearly these cases may arise because no resistance mechanisms are possible for the host, but this seems unlikely as closely related species show effective resistance, and we suggest that these are examples of no-resistance ESSs. Second, our prediction of coevolutionary cycles provides an explanation for the maintenance of additive genetic variation in resistance and virulence, as was found by Henter & Via (1995) and Henter (1995) in a study of pea aphid, *Acyrtosiphon pisum*, and its parasitoid *Aphidius ervi*. However, other experimental results (Kraaijeveld & Godfray 1997; Kraaijeveld *et al.* 1998; Fellowes *et al.* 1999) suggest areas where further modelling is required, for example the incorporation of costs of resistance that vary with host density, and the addition of other species of host or parasitoid.

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## APPENDIX A

### (a) *Monomorphic approximations of the full model*

Suppose both host and parasitoids are monomorphic with traits  $X$  and  $Y$ , respectively, and that a stable demographic equilibrium exists. The marginal host and parasitoid ESSs are found by considering the spread of rare mutants,  $\hat{X}$  and  $\hat{Y}$ , respectively,

$$\frac{\partial \lambda_N(\hat{X}, X)}{\partial \hat{X}} \Big|_{\hat{X}=X} = \frac{a'(X)}{a(X)} + \frac{(1-F)\eta'(X-Y)}{F + (1-F)\eta(X-Y)}, \quad (\text{A1a})$$

$$\frac{\partial \lambda_p(\hat{Y}, Y)}{\partial \hat{Y}} \Big|_{\hat{Y}=Y} = \frac{b'(Y)}{b(Y)} + \frac{\eta'(X-Y)}{\eta(X-Y)}, \quad (\text{A1b})$$

where primes represent derivatives. Second-order conditions (not shown) need to be examined to check for evolutionary stability. Substituting for the functions  $a(\cdot)$ ,  $b(\cdot)$  and  $\eta(\cdot)$ , and making use of the fact that at demographic equilibrium

$$F = \frac{1 - Ga(X)\eta(X-Y)}{Ga(X)(1 - \eta(X-Y))}, \quad (\text{A2})$$

we obtain an implicit expression for optimum host resistance,  $X^*$ , as a function of  $Y$

$$Y = X^* + \log \left[ \frac{G \exp(-C_H X^*) - 1}{C_H} - 1 \right], \quad (\text{A3})$$

and an explicit expression for optimum parasitoid resistance,  $Y^*$  as a function of  $X$

$$Y^* = X + \log \left[ \frac{1}{C_P} - 1 \right]. \quad (\text{A4})$$

An internal equilibrium is only possible if  $F > 0$  which, from equation (A2), requires  $Ga(X)\eta(X-Y) > 1$ . Substituting for  $a(\cdot)$  and  $\eta(\cdot)$  we obtain the condition

$$Y > X + \log[Ga(X) - 1]. \quad (\text{A5})$$

(The further condition  $F < 1$  is normally guaranteed if  $G > 1$ .) Where the above does not hold, host and parasitoid population densities expand exponentially, and as in these circumstances all hosts, asymptotically, are exposed to (though not all killed by) parasitoids, the ESS host resistance is given by equations (A1) with  $F \rightarrow 0$ . Again, substituting for our functions we obtain the optimum host resistance when no demographic equilibrium exists:

$$X^* = Y + \log \left[ \frac{1}{C_H} - 1 \right]. \quad (\text{A6})$$

In figure 3, equation (A4) is line A; equation (A5) is line C; and equations (A3) and (A6) define line B in and out of the region of demographic stability, respectively.

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