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Modelling Density-Dependent Resistance in Insect–Pathogen Interactions

K. A. J. White[1]

Department of Mathematical Sciences, University of Bath, Bath BA2 7AY, United Kingdom

and

Kenneth Wilson

Institute of Biological Sciences, University of Stirling, Stirling FK9 4LA, United Kingdom

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We consider a mathematical model for a host–pathogen interaction where the host population is split into two categories: those susceptible to disease and those resistant to disease. Since the model was motivated by studies on insect populations, we consider a discrete-time model to reflect the discrete generations which are common among insect species. Whether an individual is born susceptible or resistant to disease depends on the local population levels at the start of each generation. In particular, we are interested in the case where the fraction of resistant individuals in the population increases as the total population increases. This may be seen as a positive feedback mechanism since disease is the only population control imposed upon the system. Moreover, it reflects recent experimental observations from noctuid moth–baculovirus interactions that pathogen resistance may increase with larval density. We find that the inclusion of a resistant class can stabilise unstable host–pathogen interactions but there is greatest regulation when the fraction born resistant is density independent. Nonetheless, inclusion of density dependence can still allow intrinsically unstable host–pathogen dynamics to be stabilised provided that this effect is sufficiently small. Moreover, inclusion of density-dependent resistance to disease allows the system to give rise to bistable dynamics in which the final outcome is dictated by the initial conditions for the model system. This has implications for the management of agricultural pests using biocontrol agents—in particular, it is suggested that the propensity for density-dependent resistance be determined prior to such a biocontrol attempt in order to be sure that this will result in the prevention of pest outbreaks, rather than their facilitation. Finally we consider how the cost of resistance to disease affects model outcomes and discover that when there is no cost to resistance, the model predicts stable periodic outbreaks of the insect population. The results are interpreted ecologically and future avenues for research to address the shortfalls in the present model system are discussed.

[1] To whom correspondence should be addressed. E-mail: kajw@maths.bath.ac.uk.

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1. INTRODUCTION

The majority of models analysing insect host–pathogen dynamics assume that the infection process is linear (see, for example, Anderson and May, 1981), in other words, that the number of new infections is linearly proportional to the density of susceptible hosts and the density of infectious agents (free-living pathogen, infectious cadavers, or infectious individuals). This contrasts with anecdotal, and some experimental, evidence (e.g., Steinhaus, 1958), which suggests that the infection process is non-linear, with the per capita risk of infection increasing at a greater than linear rate with density, due to undefined "stresses" associated with crowding. More recently, life-history theory has led to the opposite prediction, namely, that for many insect pest species we can expect phenotypic plasticity to result in a decline in susceptibility to disease with increasing population density, due to an increased investment in pathogen resistance mechanisms at high densities (Wilson and Reeson, 1998; Reeson *et al*., 1998). This is because the force of infection, and hence the risk of becoming infected, is assumed to increase with the density of conspecifics, thus favouring the investment in costly resistance mechanisms only at high densities when they are likely to be called upon. Experimental tests of this hypothesis come mainly from moth–baculovirus interactions (see Wilson and Reeson, 1998, and references cited therein). For example, Kunimi and Yamada (1990) found that when larvae of the Oriental armyworm moth were reared at a range of densities and then exposed to a known dose of nuclear polyhedrosis virus (NPV) the level of virus-induced mortality declined as larval density increased. Similar results have recently been reported by Reeson *et al.* (1998), who found that larvae of the African armyworm moth were up to eight times more resistant to NPV when reared in crowds than when reared solitarily.

While these observations stimulated the present theoretical study, we are not concerned with specific biological details concerning the noctuid moth–NPV interaction. Our purpose is to develop and analyse a model which incorporates density-dependent resistance to disease and determines how this affects the host–pathogen interactions. Moreover, this study may be of more general ecological interest for the following two reasons. First, the nature of the density-dependent resistance to disease observed by Kunimi and Yamada (1990), Reeson *et al.* (1998), and others could be considered a positive feedback mechanism. If disease is the only population regulator (as we will assume) there will be less direct host population regulation as population size increases. Hence we might project that density-dependent resistance in this form would be destabilizing. However, this is not always the case as we demonstrate below. Second, it is likely that resistance to disease has an associated cost. Thus, it is interesting to investigate how the cost of host resistance interacts with the population dynamics to alter the stability properties of the system. Again, this may have broader implications beyond our specific modelling problem.

A common feature among insect species is that they exhibit discrete population generations. With this scenario the use of continuous-time models to describe the population dynamics of a species becomes questionable unless, for example, it incorporates time dependence in appropriate demographic parameters or uses suitable timescales on which the population processes can be reasonably represented as continuous in time. Continuous-time models are, of course, of use in the cases where insect generations do overlap.

Extensive modelling carried out by Anderson and May (1981) provided a starting point for later modelling of insect populations together with their microparasitic diseases (Liu *et al*., 1987; Nisbet and Gurney, 1983; Hochberg, 1991; Dwyer and Elkington, 1993). One difficulty arising in modelling insect populations is that they typically have several life stages (e.g., eggs, different instar larvae, adults) and added to this stage structure, there is the additional complication that specific diseases may attack just one stage (often dictated by the differences in feeding behaviours). Extending the work of Anderson and May (1981), Brown (1984) distinguished between larval and adult insects although there was some non-zero probability of moving directly from the larval stage to become an adult in that model. Addressing this lack of development time, Briggs and Godfray (1995) also considered adapted models imposing a fixed development period between birth and adulthood. Several versions of the model were considered to determine how the population dynamics altered depending on the insect stage infected by disease. They also incorporated seasonal heterogeneity into host–parasite models again using delay differential equations (Briggs and Godfray, 1996).

The potential for using discrete-time models in the ecological sciences and in particular in disease-related problems has a reasonably lengthy history beginning with the Nicholson–Bailey model for host–parasitoid interactions (1935). What distinguishes a parasitoid from a pathogen is that parasitoid infection and subsequent reproduction requires the death of the host. Work by May (for example, May 1974) brought discrete-time models to the fore in theoretical ecology when he demonstrated the capacity of an apparently simple model structure (the discrete-time logistic map) to produce highly complex

nonlinear patterns including chaos. Since this time, several discrete-time models have been proposed to describe host–parasitoid interactions (see, for example, Hochberg *et al*., 1990; Beddington *et al*., 1975; Regniere, 1984; Rohani *et al*., 1994).

The discrete generational nature of the noctuid moth population which motivated the present theoretical study also motivated the choice of theoretical model as one with discrete-time structure. It differs from the more common such approach in that we are not dealing with a host–parasitoid interaction but a host–microparasite interaction in which the microparasite (pathogen) maintains itself in a free-living state within the environment.

In the next section we describe the model assumptions which were made and the actual structure of the model. We then carry out some analysis including the determination of steady-state population levels and their stability and incorporate numerical simulations of the system to demonstrate its properties. The theoretical results are interpreted in an ecological context and finally modifications to the model are described in the Discussion to address some of the shortfalls of the present model system.

2. THE MODEL

The simplistic model which we use to study the phenomenon of density-dependent resistance to disease makes the following assumptions:

1. To mimic non-overlapping generations observed in many insect populations, we use a discrete-time population model.

2. The model considers only the larval insect stage. This assumes that no major density-dependent effects arise at later stages of insect development. In particular, we define the state variables relating to larval populations as

S_i = density of susceptible larvae at the start of the ith generation

R_i = density of resistant larvae at the start of the ith generation.

3. Although resistance is determined throughout larval development in response to cues perceived as young larvae (Wilson and Reeson, 1998), for simplicity we assume that individuals are born into resistant or susceptible classes and remain within that class throughout their development. In particular, the fraction of individuals giving birth to susceptibles, $f(N)$, decreases with the total density of larvae which have survived the previous generation to reproduce. Consequently, the function $f(N)$, defined in Table I, may take the form

$$f(N) = \frac{\alpha}{1+\gamma N}, \quad \gamma = \text{constant} > 0,\ 0 < \alpha = \text{constant} < 1, \tag{1}$$

where N is the number of surviving individuals at the end of a generation.

TABLE I

Model Parameter Definitions

Parameter	
Λ_S	Number of susceptibles born per surviving individual
Λ_R	Number of resistants born per surviving individual
λ	Number of pathogen propagules produced per infected death
$f(N)$	Fraction of surviving individuals giving birth to susceptibles (density dependent)
$\sigma_S(P)$	Density-dependent survival of susceptibles
σ_R	Density-independent survival of resistants ($\sigma_S(0) > \sigma_R$)
σ_P	Density-independent survival of pathogen propagules

4. Resistance is assumed to be a phenotypically plastic response to host density. We assume that the genes governing density-dependent resistance have gone to fixation and hence we ignore host genetics here (a subsequent publication will examine the evolution of this trait).

5. Generational time is sufficiently long to allow all diseased individuals to die during a single generation.

6. When the infected individuals die, they produce free-living disease pathogen propagules which have some constant survival probability during any generation. Hence we include a third state variable

P_i = density of free-living pathogen particles at the start of the ith generation.

7. The fraction of susceptible larvae surviving to the end of their generation is a decreasing function of the density of free-living pathogen particles. Defining $\sigma_s(P_i)$ as in Table I, we typically use the form

$$\sigma_S(P_i) = \tau\left(1+\frac{aP_i}{k}\right)^{-k}, \qquad a, \tau, k = \text{constants} > 0. \tag{2}$$

This form is based on a negative binomial distribution of pathogen attacks with the parameter k an inverse measure of the degree of aggregation of such attacks (see, for example, May, 1978; Hochberg *et al.*, 1990). In our context the attack–aggregation relation reflects the distribution of free-living pathogen in the environment which will tend to be clumped since it will depend on the location of infected hosts when they die. Such aggregation tends to stabilise the population dynamics—if, however, the pathogen is not sufficiently aggregated, the dynamics are unstable and growing, unbounded oscillations are produced.

8. Finally we assume that there is some cost associated with resistance to disease which has the effect of reducing larval survival for the resistant class in the absence of disease. The differential survival arising from costs associated with host resistance to disease is incorporated into the model by assuming that

$$\sigma_S(0) = \tau > \sigma_R$$

(see Table I for definition of parameters). Such costs have been demonstrated in a number of interactions between insects and their pathogens and parasites (see, for example, Fuxa and Richter, 1989; Boots and Begon, 1993; Kraaijveld and Godfray, 1997). Survival of susceptibles may fall below that of the resistant class at some non-trivial population density (i.e., $\sigma_S(P_i > 0) < \sigma_R$) due to the disease.

These assumptions give rise to the model system (parameters given in Table I)

$$S_{i+1} = \Lambda_S f(N_i)\, N_i \tag{3a}$$

$$R_{i+1} = \lambda_R (1 - f(N_i))\, N_i \tag{3b}$$

$$P_{i+1} = \sigma_P P_i + \lambda(\sigma_s(0) - \sigma_s(P_i))\, S_i, \tag{3c}$$

where

$$N_i = (\sigma_s(P_i)\, S_i + \sigma_R R_i)$$

is the total number of individuals which survive to the end of the ith generation.

3. MODEL OUTCOMES

We present results in three cases; in the first we assume no resistance to disease, in the second we assume that there is no disease but two host compartments, and finally we consider the complete model system and investigate its behaviours. Details of the reduced models, steady-state levels, and stability criteria in each case are given in the appendix.

3.1. Case 1: No Resistant Class

With $f(N_i) = 1$, we obtain the non-trivial steady state $(\tilde{S}, \tilde{P})$ given implicitly by the relations

$$\sigma_S(\tilde{P}) = \frac{1}{\Lambda_S}, \qquad \tilde{S} = \frac{\tilde{P}\Lambda_S(1-\sigma_P)}{\lambda(\sigma_S(0)\,\Lambda_S - 1)}, \qquad \sigma_S(0)\,\Lambda_S > 1. \tag{4}$$

Since $\sigma_S(0) \leqslant 1$, we must take $\Lambda_S > 1$ so that, in the absence of parasitism, the host population grows in successive generations without bound.

Stability requires that host survival is a decreasing function of the pathogen population at the steady state bounded such that reduction in survival does not alter too severely from a change in pathogen numbers close to the steady state.

Considering the specific functional form given in (2) we obtain the explicit steady state

$$\tilde{S} = \frac{\tilde{P}\Lambda_S(1-\sigma_P)}{\lambda(\sigma_S\Lambda_S - 1)}, \qquad \tilde{P} = \frac{k}{a}\left[(\Lambda_S\tau)^{1/k} - 1\right] \tag{5}$$

and the single stability condition

$$k < \frac{\tau\Lambda_S - 1}{\Lambda_S\tau(1 - (\Lambda_S\tau)^{-1/k})} = Q(k; \tau, \Lambda_S) \tag{6}$$

which requires that $k < 1$ (see Fig. 1). Figure 1 also shows numerical solutions to the time-dependent problem in the two cases $k < 1$ and $k > 1$. In other words, in the absence of a resistant class, the host–pathogen interaction is stable only if pathogen aggregation is sufficiently great ($k < 1$).

3.2. Case 2: No Pathogen

If $P_0 = 0$ then $P_i = 0$ for all subsequent generations and there is a single non-trivial steady state given by

$$f(\tilde{N}) = \frac{1 - \sigma_R\Lambda_R}{\sigma_S(0)\,\Lambda_S - \sigma_R\Lambda_R}. \tag{7}$$

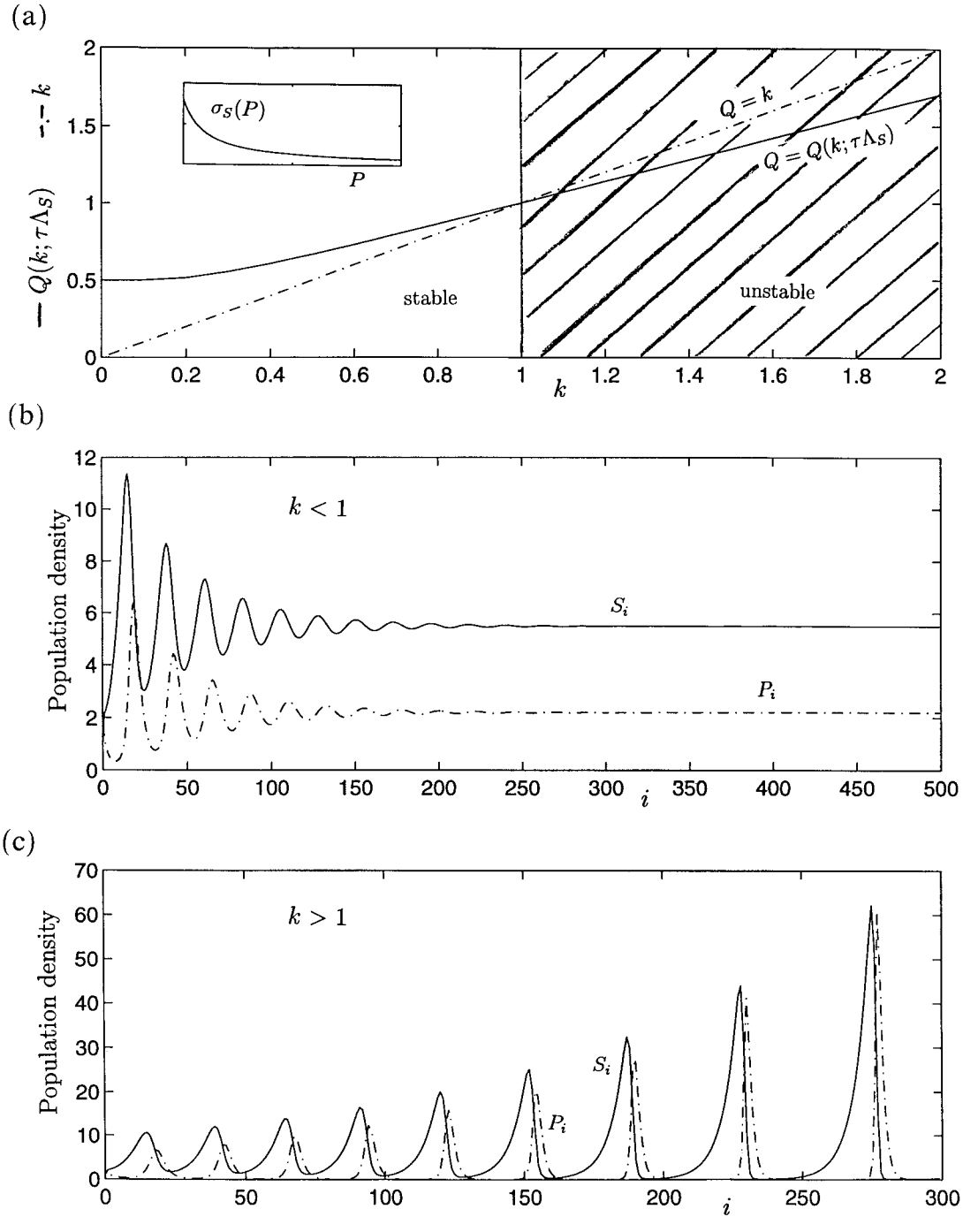

FIG. 1. (a) Graphical solution of (6). The solid line is $Q(k;\tau\Lambda_S)$, and the dash-dotted line is k. For this example we used $\tau\Lambda_S = 2$ but in all cases, for $k < 1$ the inequality (6) holds, for $k = 1$, $Q(1;\tau\Lambda_S) = 1$ and for $k > 1$ the inequality is violated. The inset shows a typical form for $\sigma_S(P)$. In (b) and (c) we show numerical simulations of the model system (8a) and (8b) using model parameters $\Lambda_S = 1.5$, $\lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, and $a = 0.1$ and initial conditions $S_0 = 1$ and $P_0 = 2$. The solid line gives S_i (number of susceptible hosts) and the dashed-dotted line gives P_i (number of pathogen particles). In (b) $k = 0.5$ and in (c) $k = 1.5$, demonstrating for an arbitrary set of model parameters that the non-trivial steady state is stable for values of k less than one and unstable otherwise when $f(N) = 1$.

If either $f(N) = \text{constant}$ or $\sigma_S(0)\,\Lambda_S = \sigma_R \Lambda_R$, there is no such steady state and the population will either grow or decay exponentially. In other words, in the absence of pathogen, the population dynamics are unstable either when the fraction of individuals giving birth to susceptibles is density independent or if there is no difference in survival and fecundities of the susceptible and resistant classes.

When a non-trivial steady state does exist, the monotonic decreasing nature of the function $f(N)$ means that there is a single non-trivial steady state (see Fig. 2); moreover this steady state is stable only if the susceptible hosts have unbounded growth and the resistant hosts decay in the absence of interaction between the two subpopulations.

3.3. Case 3: The Complete System

We investigate the complete model system using specific forms for the functions $f(N)$ and $\sigma_S(P)$.

3.3.1. *Case* 3a: *Density-Independent Resistance*; *i.e.*, $f(N) = \textit{Constant}$. With $f(N) = \alpha = \text{constant}$ and taking

$$\sigma_S(\tilde{P}) = \tau \left[1 + \frac{a\tilde{P}}{k} \right]^{-k}$$

the model gives rise to a single non-trivial steady state

$$\tilde{P} = \frac{k}{a} \left[\left(\frac{\tau \alpha \Lambda_S}{1 - (1-\alpha)\,\sigma_R \Lambda_R} \right)^{1/k} - 1 \right]$$

provided that

$$0 < 1 - (1-\alpha)\,\sigma_R \Lambda_R < \alpha \tau \Lambda_S.$$

The stability criteria derived in Appendix A.3 were investigated graphically using the coefficients given in (19a)–(19c) evaluated for our particular choice of $\sigma_S(P)$ and $f(N)$. Pathogen aggregation in host attack is associated with the stability of host–pathogen interactions (May, 1978; Hochberg *et al.*, 1990; Hassell and

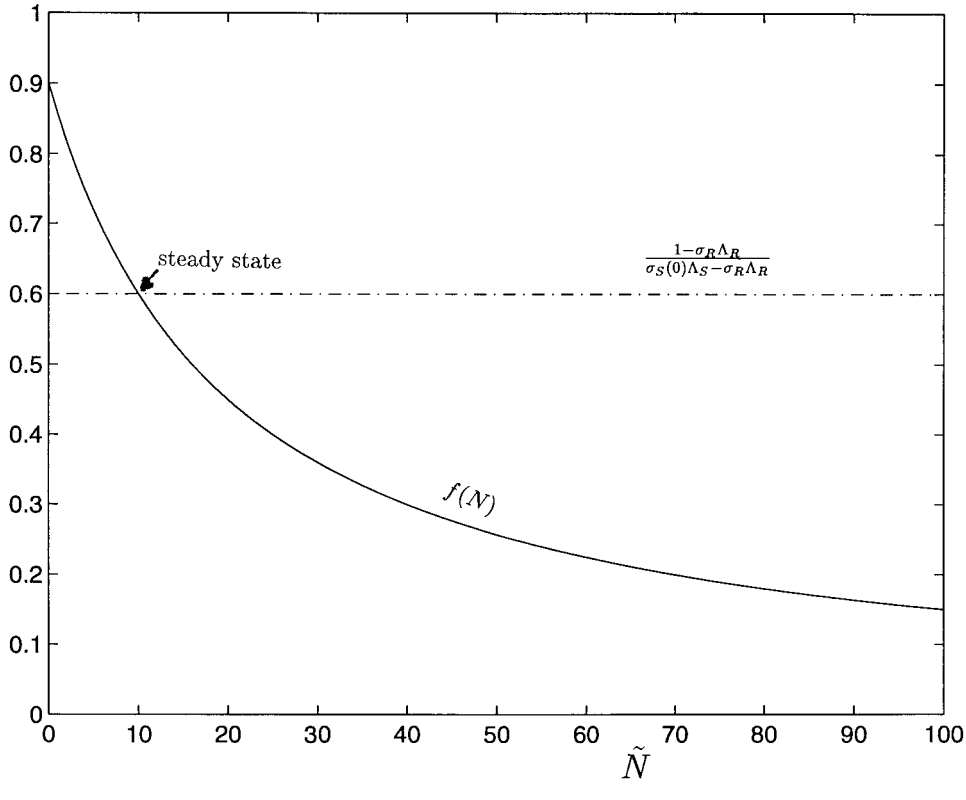

FIG. 2. Graphical solution to (7) when $\alpha = 0.9$ and $\gamma = 0.05$, indicating a single non-trivial steady state. The solid curve is the lhs of (7) and the dash-dotted line, the rhs of (7).

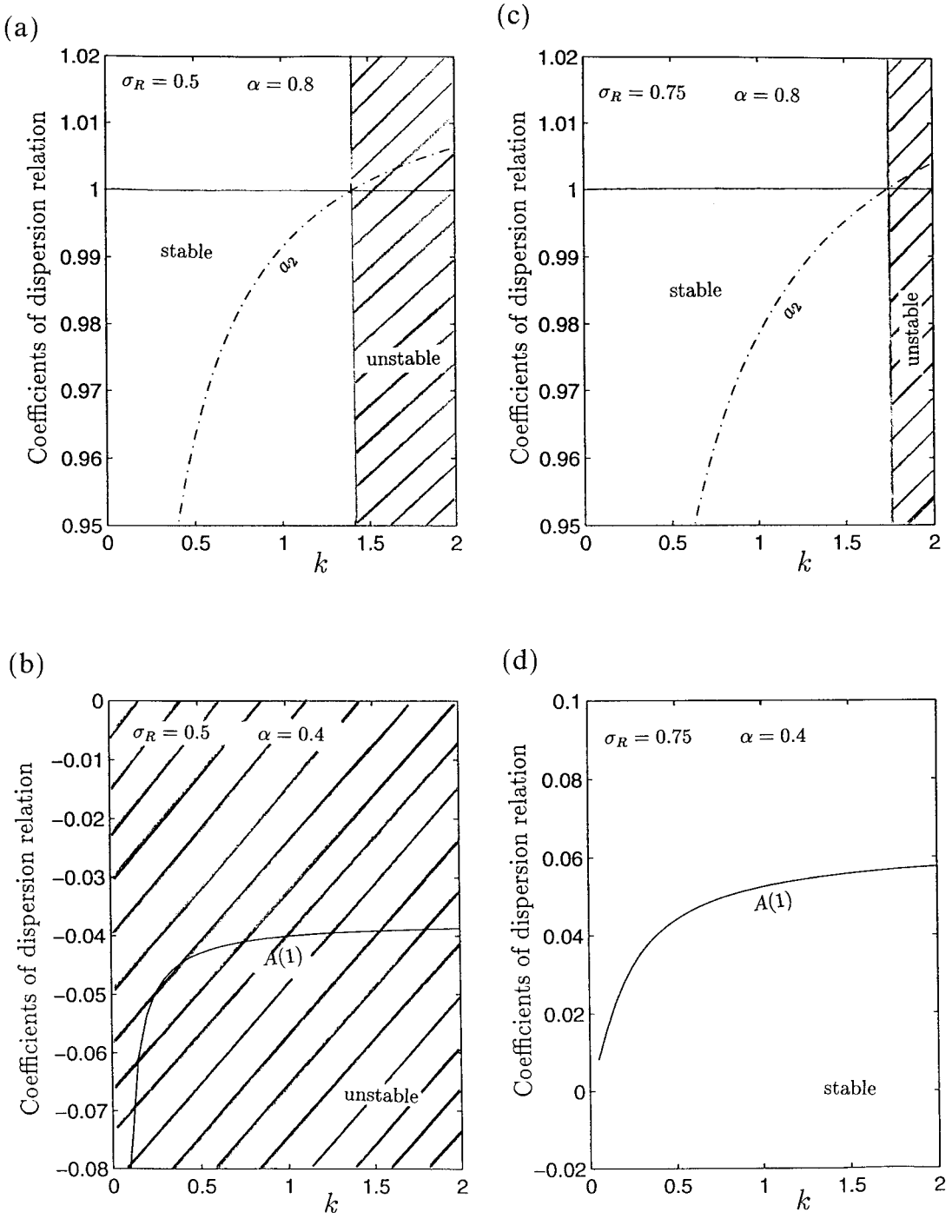

FIG. 3. Stability criteria given by the Jury conditions (outlined in Appendix A.3) when $f(N) = \alpha = \text{constant}$ as the parameter k, an inverse measure of parasite aggregation, is varied. In the shaded regions, the steady state is unstable and in all other regions, the steady state is stable. We show only those criteria which alter the stability of the system when k varies. The other criteria in each case still satisfy their respective inequality given in A.3. Where it appears, the solid line is the expression $A(1) = (1 + a_1 + a_2)$ and the dash-dotted line is a_2. Stability requires that $A(1) = (1 + a_1 + a_2) > 0$, $-A(-1) = (1 - a_1 + a_2) > 0$, and $|a_2| < 1$. Note that $-A(-1) > 0$ was always found to hold for these parameter combinations. The model parameters common to all calculations were $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, and $a = 0.1$. In (a) and (b) $\sigma_R = 0.5$ and in (c) and (d) $\sigma_R = 0.75$. In (a) and (c) $\alpha = 0.8$ and in (b) and (d) $\alpha = 0.4$. For the parameters used in (d), none of the stability criteria were violated and so the population dynamics will settle to a non-trivial stable steady state.

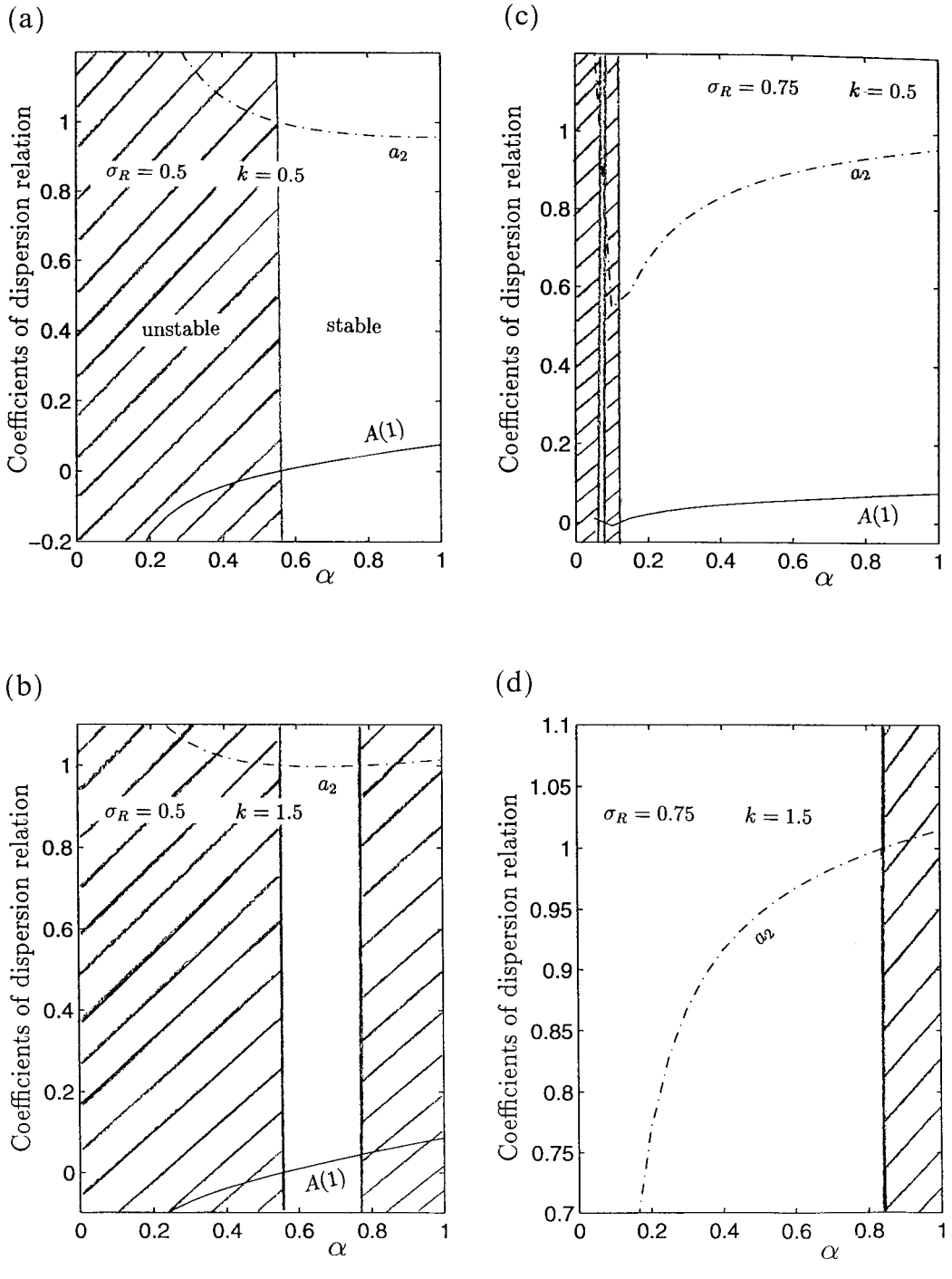

FIG. 4. Stability criteria given by the Jury conditions (outlined in Appendix A.3) when $f(N) = \alpha =$ constant as the parameter α is varied. As above, the steady state is unstable in shaded regions and otherwise it is stable. We show only those criteria which alter the stability of the system when α varies. The other criteria in each case still satisfy their respective inequality given in A.3. Where it appears, the solid line is the expression $A(1) = (1 + a_1 + a_2)$ and the dash-dotted line is a_2. Stability requires that $A(1) = (1 + a_1 + a_2) > 0$, $-A(-1) = (1 - a_1 + a_2) > 0$, and $|a_2| < 1$. Note that $-A(-1) > 0$ was always found to hold for these parameter combinations. The model parameters common to all calculations were $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, and $a = 0.1$. In (a) and (b) $\sigma_R = 0.5$ and in (c) and (d) $\sigma_R = 0.75$. In (a) and (c) $k = 0.5$ and in (b) and (d), $k = 1.5$.

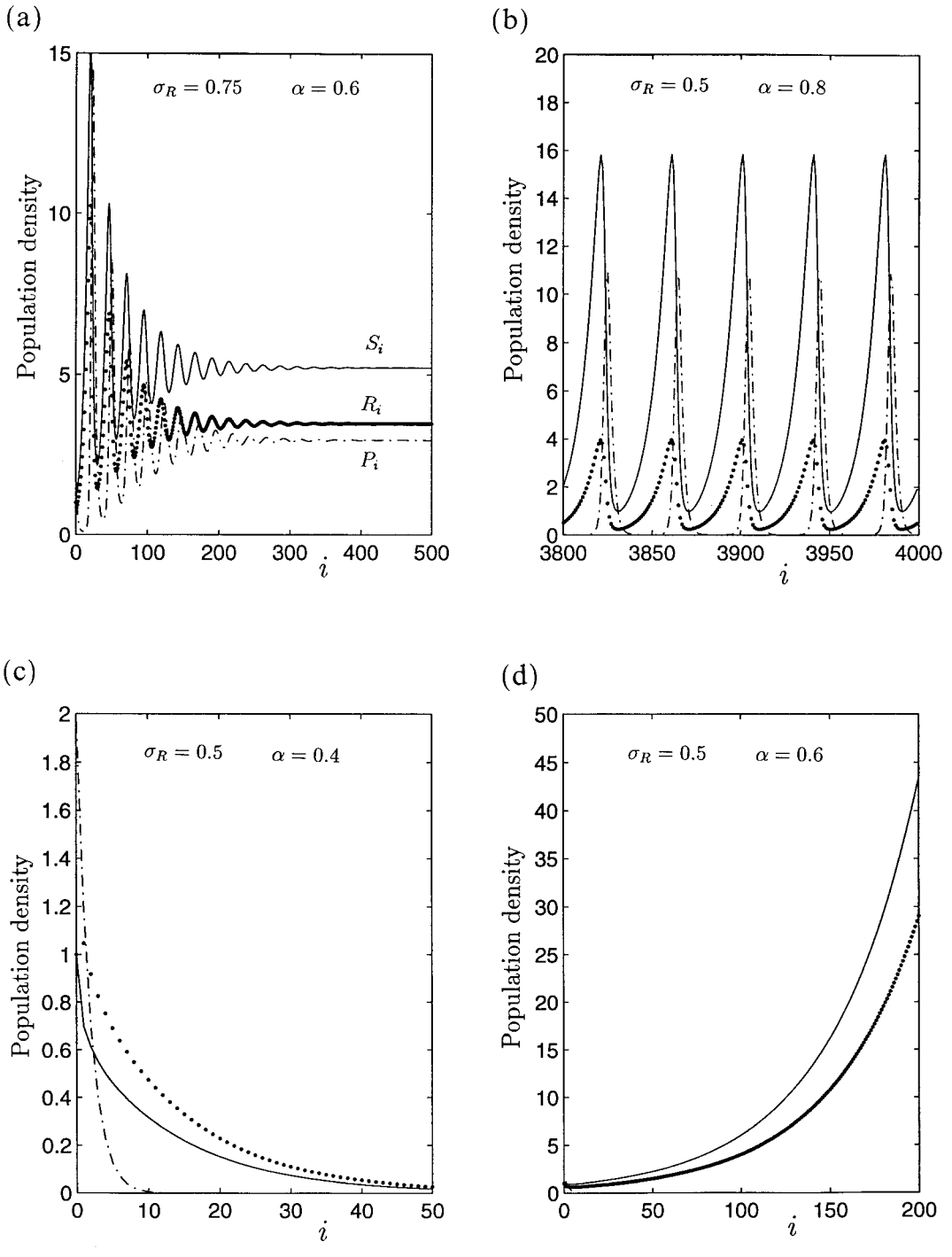

FIG. 5. Numerical model solutions to (3a)–(3c) with $f(N) = \alpha$ = constant. Parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, $a = 0.1$, and $k = 1.5$. In (a) $\alpha = 0.6$ and $\sigma_R = 0.75$, in (b) $\alpha = 0.8$ and $\sigma_R = 0.5$, in (c) $\alpha = 0.4$ and $\sigma_R = 0.5$, and in (d) $\alpha = 0.6$ and $\sigma_R = 0.5$. The solid line gives the susceptible population levels S_i, the dotted line gives the resistant population levels R_i, and the dash-dotted line gives the parasite population levels P_i.

May, 1974) and was shown to play an important role here in the absence of a resistant class (Case 1). Hence in Fig. 3 we varied the parameter k, an inverse measure of aggregation, in four cases to see how stability of the non-trivial steady state depended on aggregation. Two points arise from this. First, the dynamics are stable for $k > 1$ which was *not* the case with no resistant class (see Fig. 1) and second, there is some non-trivial relation between α (the density-independent fraction of surviving individuals giving birth to susceptibles) and k to ensure stability since decreasing α when $\sigma_R \Lambda_R \leqslant 1$ destabilizes the non-trivial steady state whereas decreasing α when $\sigma_R \Lambda_R \geqslant 1$ stabilizes the system.

In Fig. 4 we continued to investigate the linear stability criteria detailed in A.3 varying α for two values of k ($k = 0.5 < 1$ and $k = 1.5 > 1$). With $k = 0.5$ (which would produce stable dynamics in the absence of a resistant class), the model system is stable provided that α is sufficiently large in all cases. The picture is somewhat different when $k = 1.5$ (unstable dynamics in the absence of a resistant class). Here if $\sigma_R \Lambda_R < 1$, the non-trivial steady state is stable for some intermediate values of α but if $\sigma_R \Lambda_R \geqslant 1$, the steady state is stable provided that α is sufficiently small.

Combining the outcomes from Figs. 3 and 4 leads to the two general observations:

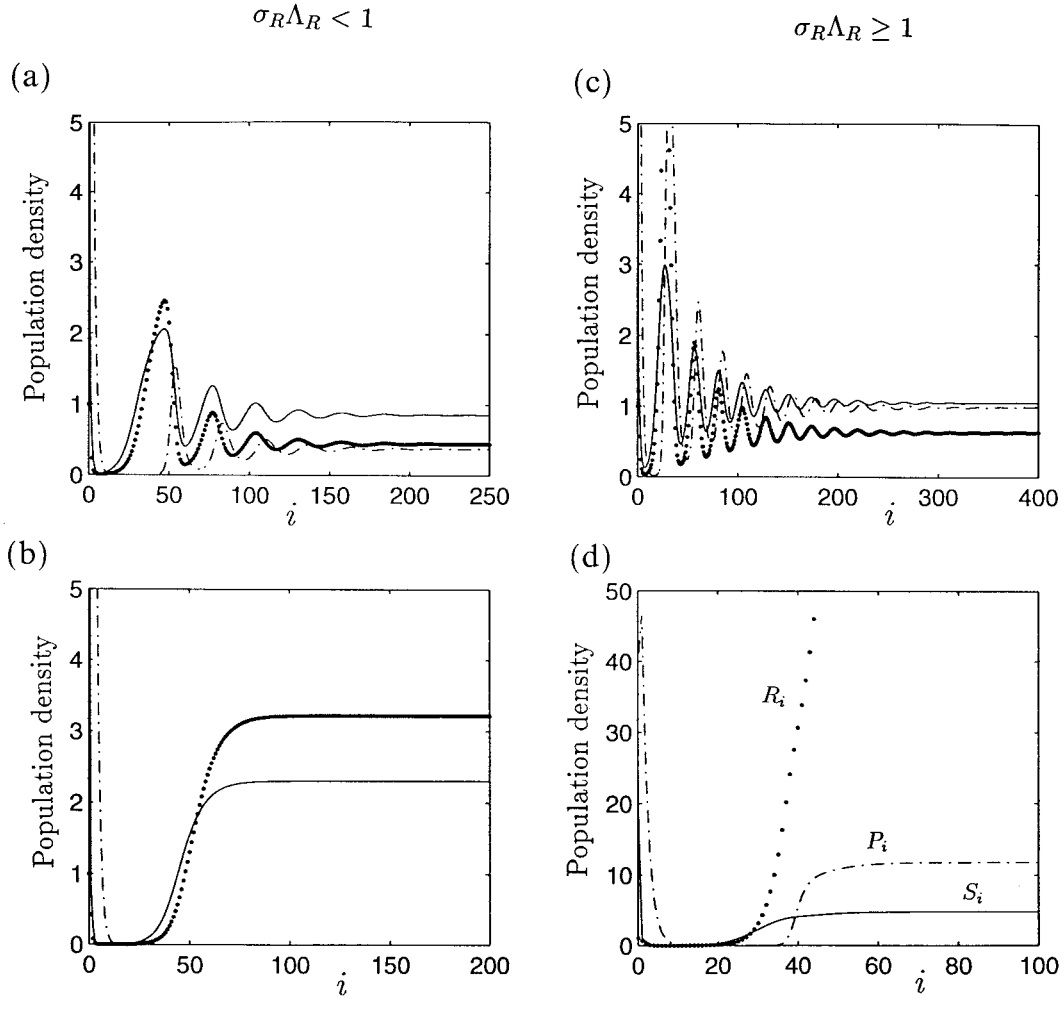

FIG. 6. Numerical model solutions for the complete model system when $f(N)$ is density dependent. In (a) and (b) we have $\sigma_R \Lambda_R < 1$ and in (c) and (d) we have $\sigma_R \Lambda_R \geqslant 1$. The equation $\Pi(\tilde{P}) = 0$ (described in the Appendix) has two solutions, indicating that the system is bistable so that model behaviours may depend on initial conditions. The particular model parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.9$, $a = 0.5$, $k = 1.5$, $\alpha = 0.8$, and $\gamma = 0.25$. In (a) and (b) we have $\sigma_R = 0.5$ and in (c) and (d) we have $\sigma_R = 0.8$. In (a) and (c) initial conditions were $S_0 = 10$, $R_0 = 1$, and $P_0 = 10$ and in (b) and (d) initial conditions were $S_0 = 20$, $R_0 = 1$, and $P_0 = 40$. The solid line gives the susceptible population levels S_i, the dotted line gives the resistant population levels R_i, and the dash-dotted line gives the parasite population levels P_i.

1. Inclusion of a resistant class tends to stabilise host–pathogen interactions (model case 1 is now stable for $k > 1$).

2. Parasitism can have a non-trivial effect on the stability of a two-host system (model case 2 requires that $f(N)$ is *not constant* in order to obtain a non-trivial steady state).

Motivated by the linear stability analysis shown in Figs. 3 and 4, the numerical simulations in Fig. 5 demonstrate the types of behaviour which the model system (3a)–(3c) can exhibit. In particular, we see that both stable non-trivial steady-state solutions (Fig. 5a) and stable oscillations (Fig. 5b) are possible with $f(N) = \alpha = \text{constant}$. Comparing parameter values in these two cases suggests that in Fig. 5a, the host susceptible–resistant dynamics dominate whereas in Fig. 5b, the susceptible host–pathogen dynamics dominate. Viewed in this way, the model solutions are to be expected since the host–pathogen dynamics (with $k > 1$) are unstable with growing oscillations; inclusion of resistance stabilises this and hence either stable oscillations arise if the resistant class is a relatively weak component in the population dynamics or there is a stable steady state if the resistant class has a strong influence on the dynamics.

If, however, a large fraction of the population is born resistant but the resistant population has a relatively small survival rate, the system collapses either to the trivial steady state (Fig. 5c) or to unbounded growth for the host and decay of the pathogen population (Fig. 5d).

3.3.2. *Case b*: *Density-Dependent Resistance*, $f(N) = \alpha/(1 + \gamma N)$. With $f(N) = \alpha/(1 + \gamma N)$ and taking

$$\sigma_S(P) = \tau \left[1 + \frac{aP}{k} \right]^{-k}$$

we see that two non-trivial steady states are possible for a given set of model parameters (Fig. 6) in both cases $\sigma_R \Lambda_R < 1$ (net decay of the resistant class) and $\sigma_R \Lambda_R \geqslant 1$ (net growth of the resistant class). That is, the existence of the steady states does not depend on the fecundity properties of the resistant class. Depending on the initial conditions, the model system either approaches a non-trivial steady state or collapses to

1. a non-trivial steady state for S and R with the pathogen driven to extinction ($\sigma_R \Lambda_R < 1$) or

2. a non-trivial steady state for S and P but with an exponentially growing resistant class ($\sigma_R \Lambda_R \geqslant 1$).

Both of these outcomes are consistent with the behaviours of the underlying two-class interactions but what is of interest is the bistable nature of the model system.

To investigate further, we compared the model results in which $f(N) = \alpha = \text{constant}$ to the present density-dependent scenario. Our findings are shown in Fig. 7 for an arbitrary set of model parameters and again for the two cases $\sigma_R \Lambda_R < 1$ and $\sigma_R \Lambda_R \geqslant 1$. With $\sigma_R \Lambda_R < 1$, we consider the effect of density-dependent resistance on stable oscillatory behaviour (Fig. 7a) and see that for relatively low levels of density dependence (Fig. 7b) the oscillations are damped and a stable steady state results but that as the density dependence increases the non-trivial steady state collapses (Fig. 7c) to a system for the host only (pathogen driven to extinction).

When density-dependent resistance was imposed on a non-trivial steady state (Fig. 7d) with $\sigma_R \Lambda_R \geqslant 1$, stability was maintained for low levels of density dependence (Fig. 7e) but again collapsed as the density dependence was increased (Fig. 7f) to non-trivial susceptible host and pathogen levels but an exponentially growing resistant class.

At first these results may seem somewhat counter-intuitive since the density dependence $f(N)$ in some sense represents a positive feedback mechanism reducing regulation of the host as host population levels increase. However, this must also be coupled to the underlying host–pathogen interaction which would exhibit unstable oscillations in the absence of a resistant class. The inclusion of density dependence in $f(N)$ means that a smaller fraction of hosts become susceptible to the disease and hence the oscillatory behaviour will play a less dominant role and the dynamics can be stabilized. However, if the strength of the density dependence is too great, then either the pathogen will not be maintained (Fig. 7c) or the resistant class will be able to grow without bound (Fig. 7f). These two alternatives arise for reasons discussed in the previous section.

Finally we investigated whether imposing a cost to resistance ($\sigma_S(0) > \sigma_R$) affects the population dynamics. With $\alpha < 1$, a fraction $(1 - \alpha)$ of surviving individuals give birth to resistant individuals; if α is sufficiently small, assuming no cost to resistance ($\sigma_S(0) = \sigma_R$) will *destabilise* the non-trivial steady state (Figs. 8a and 8b). However, with larger α (Fig. 8c), assuming no cost to resistance does not affect the stability of the non-trivial steady state (cf. Figs.8a and 8c).

When $\alpha = 1$, all insects are born susceptible in the absence of any density-dependent resistance. In this case, assuming a cost to resistance in the density-dependent case can stabilise the dynamics from those with no density dependence (Figs. 9a and 9b); removing the cost

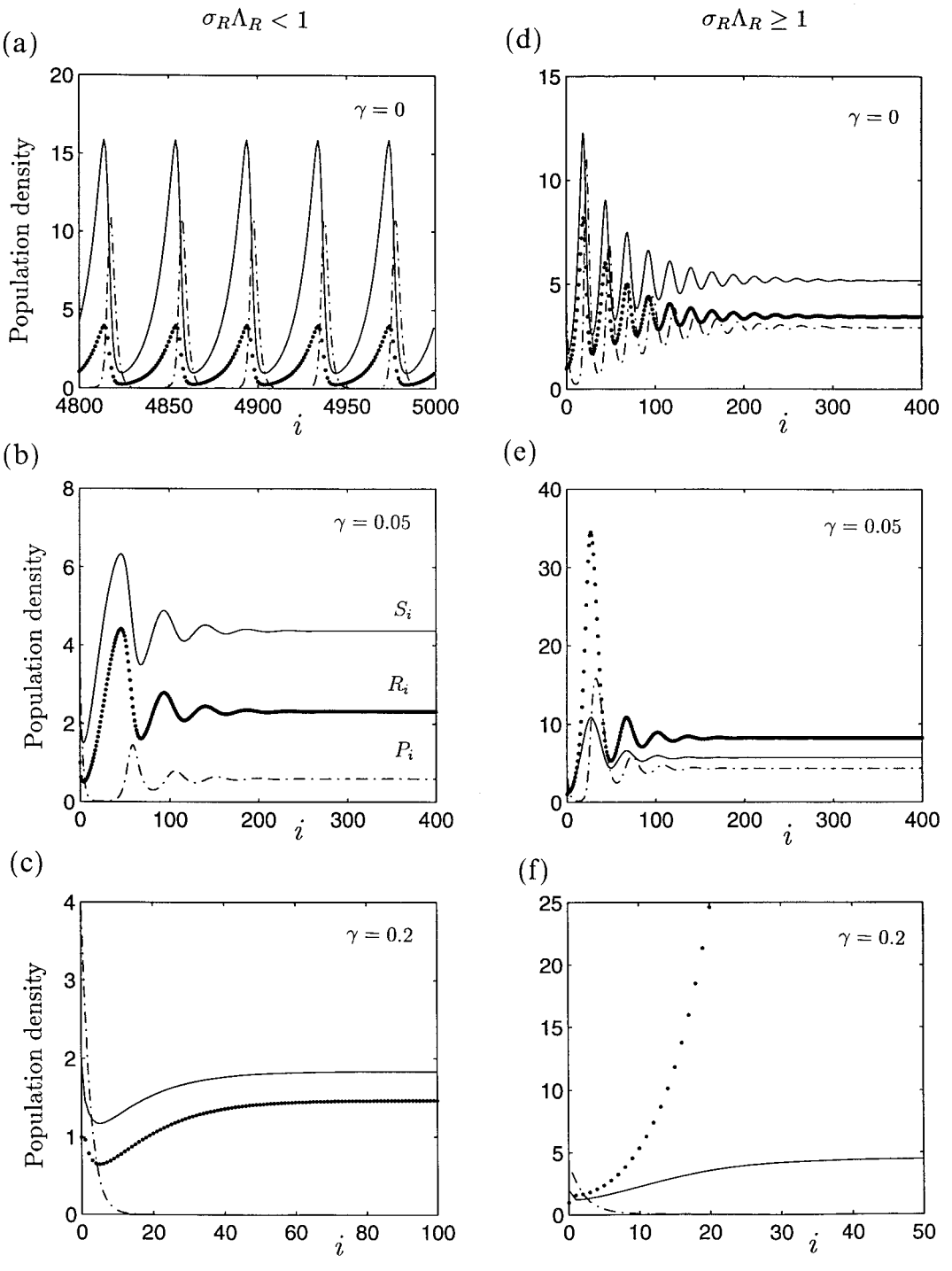

FIG. 7. Comparison between density-independent and density-dependent response to disease In (a)–(c) $\sigma_R \Lambda_R < 1$ and in (d)–(f) $\sigma_R \Lambda_R \geq 1$. Moreover, in (a) and (d), $\gamma = 0$ which means that a constant (density-independent) fraction of newborns are resistant to disease; in the remaining figures there is a varying amount of density dependence. In all figures, the solid line gives the susceptible population levels S_i, the dotted line gives the resistant population levels R_i, and the dash-dotted line gives the parasite population levels P_i. Model parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, $a = 0.1$, and $k = 1.5$. In (a)–(c), $\alpha = 0.8$ and $\sigma_R = 0.5$ and in (d)–(f), $\alpha = 0.6$ and $\sigma_R = 0.75$. Finally in (b) and (e), $\gamma = 0.05$ and in (c) and (f), $\gamma = 0.2$.

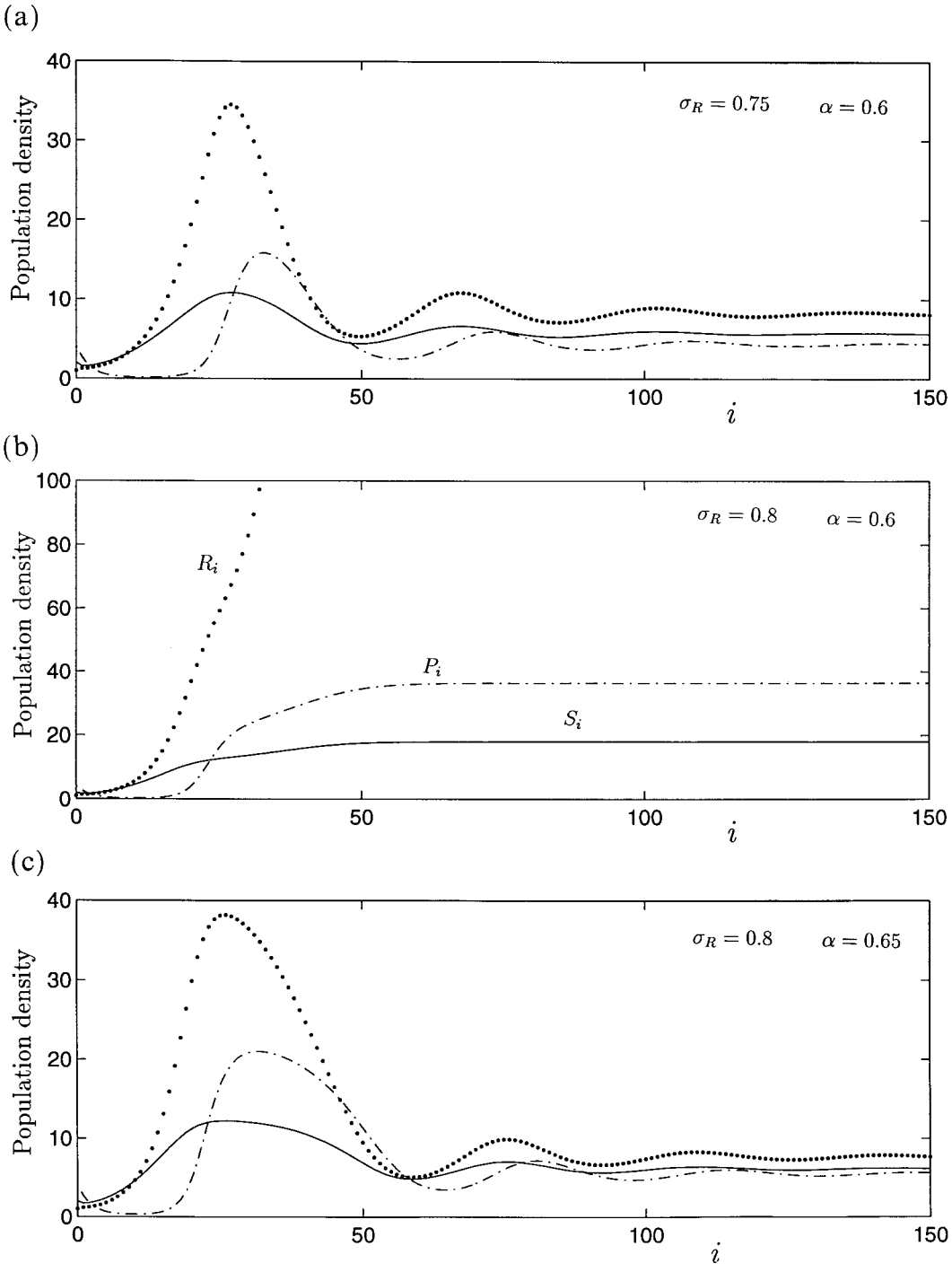

FIG. 8. Numerical model solutions for the complete density-dependent system (3a)–(3c) to investigate how fixing a cost to resistance affects model dynamics. Parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, $a = 0.1$, $\gamma = 0.05$, and $k = 1.5$ and in each case we used the initial conditions $S(0) = 2$, $R(0) = 1$, and $P(0) = 4$. In (a) $\alpha = 0.6$ and $\sigma_R = 0.75$, in (b) $\alpha = 0.6$ and $\sigma_R = 0.8$, and in (c) $\alpha = 0.65$ and $\sigma_R = 0.8$. In all figures, the solid line gives the susceptible population levels S_i, the dotted line gives the resistant population levels R_i, and the dash-dotted line gives the parasite population levels P_i.

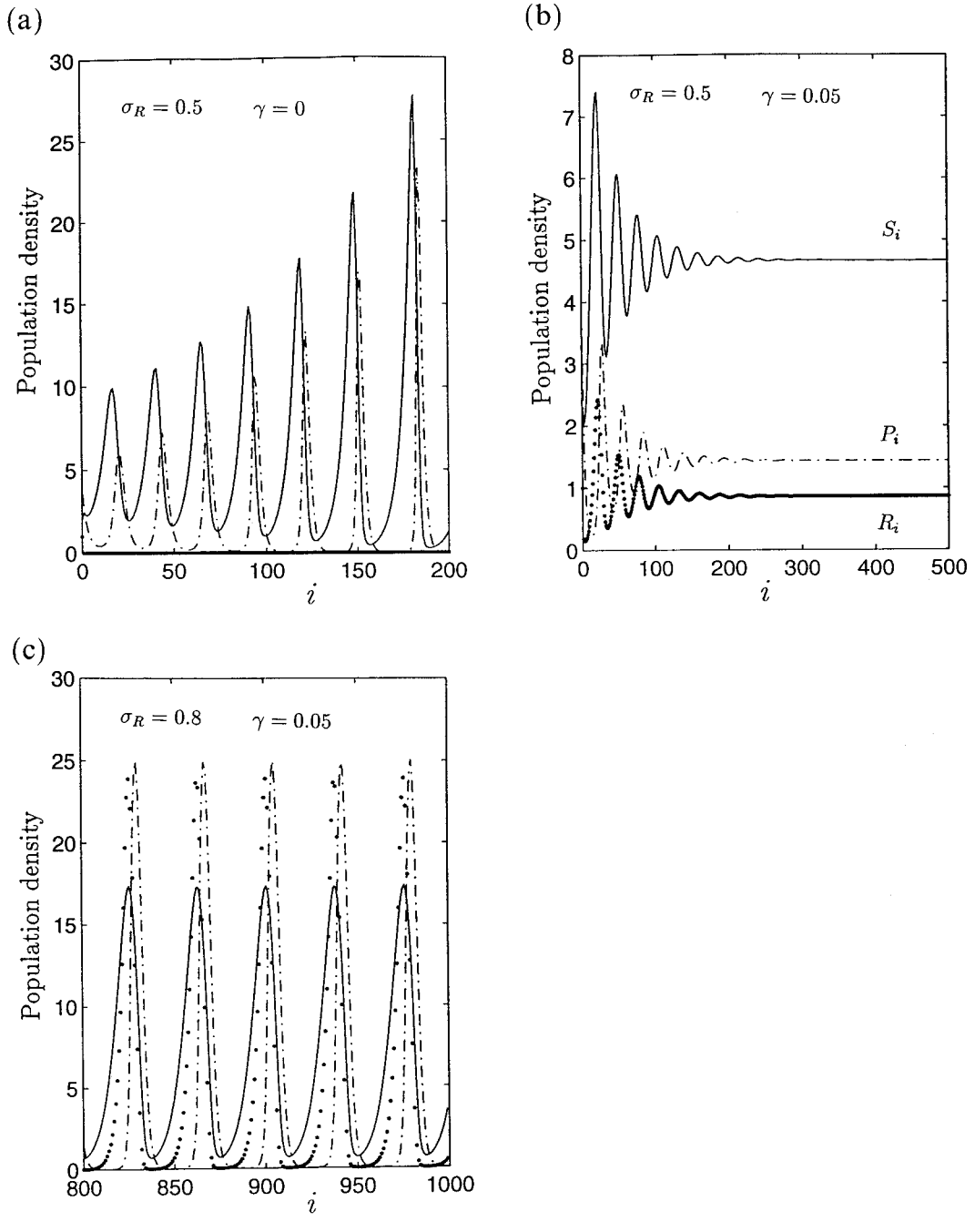

FIG. 9. Numerical model solutions for the complete system (3a)–(3c) with $\alpha = 1$. This means that in the absence of any density dependence ($\gamma = 0$), all insects are susceptible to disease. Parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5$, $\sigma_P = 0.5$, $\tau = 0.8$, $a = 0.1$, and $k = 1.5$ and in each case, we used the initial conditions $S_0 = 2$, $R_0 = 1$, and $P_0 = 4$. The solid line gives the susceptible population levels S_i, the dotted line gives the resistant population levels R_i, and the dash-dotted line gives the parasite population levels P_i. In (a) $\gamma = 0$ and $\sigma_R = 0.5$, in (b) $\gamma = 0.05$ and $\sigma_R = 0.5$, and in (c) $\gamma = 0.05$ and $\sigma_R = 0.8$.

of resistance then reduces this stability and gives rise to stable oscillations (Fig. 9c).

Considering the model solutions shown in Figs. 8 and 9, it is apparent that the most stable scenario arises when there is a cost to resistance and a constant fraction of individuals are born resistant to disease in the absence of any density dependence.

4. DISCUSSION

A cornerstone of modern epidemiology is the so-called mass-action assumption (Anderson and May 1981; de Jong *et al*., 1995), which posits that the horizontal transmission of a pathogen is linearly dependent on the densities of susceptible and infectious hosts. However, evidence from a number of empirical studies suggests that although pathogen transmission efficiency usually does increase with host and pathogen density, it often appears to do so at a decelerating rate (e.g., Dwyer and Elkinton, 1993). A number of workers have modelled this non-linearity in the transmission process in a phenomenological way, for example, by assuming that transmission depends on some power of host or pathogen density (e.g., Liu *et al*., 1987; Hochberg, 1991). Others have assumed that the decline in transmission efficiency is due to reduced pathogen uptake at high densities as a result of the spatial clumping of pathogen (Briggs and Godfray, 1995). Only recently have attempts been made to examine this phenomenon using more mechanistic models. For example, Dwyer *et al*. (1997) provide experimental data and theoretical models which appear to explain the non-linearity in the transmission process in terms of heterogeneities in pathogen resistance. However, this work concentrates on processes occurring over a single epidemic and consequently it is difficult to compare the modelling and its outcomes with our model structure and analysis. The other models mentioned here do consider the dynamics over a longer period of time but assume that demographic processes occur continuously in time. Again, this makes direct comparison of results difficult but we do observe some similar dynamics in our model. These include bistability (as seen in Liu *et al*., 1987), multigenerational host–pathogen cycles (Liu *et al*., 1987; Briggs and Godfray, 1995), and single-host generation cycles (Briggs and Godfray, 1995). In all cases, these models assume that susceptibility to pathogen attack varies between individuals but is unrelated to the density of susceptible hosts.

In contrast, the models presented here assume that pathogen resistance is density dependent, an observation for which there is some experimental data (Kunimi and Yamada, 1990; Goulson and Cory 1995; Reeson *et al*., 1998) and which is predicted on theoretical grounds. Wilson and Reeson (1998) have argued that if resistance to pathogens is costly to maintain (see Fuxa and Richter, 1989; Boots and Begon, 1993; Kraaijeveld and Godfray, 1997), then we can expect organisms to be under strong selection to invoke resistance mechanisms only when they are likely to be required Thus, because individuals are more likely to encounter pathogens like baculoviruses as population density increases, we can expect insects to invest most resources in pathogen resistance mechanisms at high population densities. In other words, the fraction of resistant individuals in the population will increase with the density of conspecifics, as modelled here. The purpose of the present paper was not to directly address issues raised by Wilson and Reeson (1998) but to investigate how a density-dependent response to disease resistance in the way suggested by the hypothesis could affect host–pathogen dynamics. We have not attempted to include host genetics in these models, but assume that the genes for density-dependent prophylactic resistance have gone to fixation. Future work will examine the evolution of this phenomenon and gene frequency dynamics. Here we concentrate on the dynamics of a genetically monomorphic host population.

With $f(N)$ monotonically decreasing, we assume that there is a decline in susceptibility to disease with increasing population density. This form of density dependence motivated our study with the alternative form $f(N) =$ constant providing a baseline model for comparison of results. However, as we described in the Introduction, there is evidence from other systems (mainly anecdotal) suggesting that susceptibility may increase with population density. This would lead to a function $f(N)$ which was monotonic increasing. Based on the model solutions presented here and the apparently negative feedback which such a choice of $f(N)$ might represent, we would speculate that if $f(N)$ was monotonic increasing it would act, in many cases, as a stabilizing influence on the host–pathogen dynamics. A further alternative would be a density-dependent response to disease in which $f(N)$ decreased for small N (reflecting increased investment in resistance) but increased for large N as population stress dominated. The latter is certainly worthy of some detailed investigation and will contribute to further exploration of this model system. We envisage that such a non-monotonic structure could significantly alter the model behaviours, possibly giving rise to multiple non-trivial and stable steady states.

The choice of a discrete-time model was a natural one given the generational nature of many insect species

including noctuid moths and it follows in the tradition of many other insect models. Moreover, in a parallel unpublished study (Gudelj and White, 1998), a continuous time model produced similar model outcomes.

The main findings of this study are as follows:

1. The inclusion of a resistant class stabilizes host–pathogen interactions despite disease acting as the population regulator. This stabilizing effect arises because fewer host individuals are born susceptible to disease. In turn this reduces the impact of disease on the host population and hence reduces the effect of an inherently unstable interaction when parasite aggregation among the host population is low.

2. The most stable scenario arises when a constant fraction of newborns are resistant to disease. This contrasts with the no pathogen scenario (Case 2) when $f(N) = \alpha$ produces unstable dynamics. Thus the three-way interaction is crucial to produce and maintain stable dynamics.

3. With density-dependent resistance to disease, cost to resistance tends to stabilize population dynamics if they would otherwise be unstable (oscillatory or unbounded).

4. Density-dependent resistance to disease gives rise to a model system in which the final outcome depends on the initial conditions. This bistability has important consequences, in particular where a pathogen is introduced into an insect host population to control its growth. Such introductions may arise if the insect is a crop pest and a virus is being used as a biocontrol agent. From the model analysis, it is clear that the final outcome is sensitive to initial conditions and hence sufficient information must be available to make the correct decisions about whether or not to introduce the pathogen.

With the particular model formulation presented here, insect outbreaks with density-dependent resistance arise only if there is no cost to disease resistance (Fig. 9). In reality, however, insect outbreaks may be more common, indicating some shortfalls in the model. One obvious reason for this is that we consider only the larval insect population where disease is manifested. This assumes that no major density or time lag effects occur during the other insect stages (adult, egg, etc). A second reason may relate to the lack of spatial component in our model formulation. In particular, density-dependent relocation (Dingle, 1996) may explain more local variations in larval densities. Both of these aspects warrant investigation and will form a basis from which we will amend the existing model structure.

However, even in this simple form, the model provides important clues as to the consequences of density-dependent resistance to disease, some of which, at first sight, seem rather counterintuitive.

APPENDIX

Linear Stability Analysis

A.1. Case 1

With $f(N_i) = 1$, the model system (3a)–(3c) reduces to the simple host–pathogen model

$$S_{i+1} = \Lambda_S \sigma_s(P_i)\, S_i \tag{8a}$$

$$P_{i+1} = \sigma_P P_i + \lambda(\sigma_s(0) - \sigma_s(P_i))\, S_i, \tag{8b}$$

variations of which have been well studied elsewhere (Nicholson and Bailey, 1935; Hochberg *et al.*, 1990).

Setting $S_{i+1} = S_i = \tilde{S}$ and $P_{i+1} = P_i = \tilde{P}$ we obtain steady-state values for the model system (8a) and (8b)

$$\tilde{S} = \tilde{P} = 0$$

and

$$\sigma_S(\tilde{P}) = \frac{1}{\Lambda_S}, \qquad \tilde{S} = \frac{\tilde{P}\Lambda_S(1-\sigma_P)}{\lambda(\sigma_S(0)\,\Lambda_S - 1)}.$$

Linearising about the steady state gives the Jacobian matrix for the non-trivial case

$$J = \begin{pmatrix} 1 & \Lambda_S \tilde{S} \dfrac{d\sigma_S}{dP} \\ \lambda\left(\sigma_S(0) - \dfrac{1}{\Lambda_S}\right) & \sigma_P - \lambda \tilde{S} \dfrac{d\sigma_S}{dP} \end{pmatrix} \tag{9}$$

with characteristic equation $\chi^2 - (\operatorname{tr} J)\,\chi + \det J = 0$. Jury conditions (see, for example, Murray, 1993; Lewis, 1977) which ensure that $|\chi| < 1$ and hence linear stability of the steady state are

$$1 - \operatorname{tr} J + \det J > 0 \tag{10a}$$

$$1 + \operatorname{tr} J + \det J > 0 \tag{10b}$$

$$1 - \det J > 0, \tag{10c}$$

where

$$\text{tr } J = 1 + \sigma_P - \lambda \tilde{S} \frac{d\sigma_S}{dP}, \quad \det J = \sigma_P - \lambda \Lambda_S \sigma_S(0)\, \tilde{S} \frac{d\sigma_S}{dP}.$$

Condition (a) requires that $d\sigma_S/dP < 0$ in which case (b) is always satisfied ($\sigma_S(0)\, \Lambda_S > 1$ is required for the non-trivial steady state to exist). Condition (c) imposes a lower bound on the derivative, namely

$$\frac{1 - \Lambda_S \sigma_S(0)}{\Lambda_S^2 \sigma_S(0)\, \tilde{P}} < \left. \frac{d\sigma_S}{dP} \right|_{(\tilde{S}, \tilde{P})}. \tag{11}$$

A.2. Case 2

In this case, the model system reduces to the one-dimensional non-linear map

$$\begin{aligned} N_{i+1} &= \sigma_S(0)\, \Lambda_S f(N_i)\, N_i + \sigma_R \Lambda_R (1 - f(N_i))\, N_i \\ &= G(N_i), \end{aligned} \tag{12}$$

where

$$N_i = \sigma_S(0)\, S_i + \sigma_R R_i.$$

This has steady-state values given by $\tilde{N} = G(\tilde{N})$ which are stable provided that $|G'(\tilde{N})| < 1$. In this case we have

$$\tilde{N} = 0 \qquad \text{or} \quad f(\tilde{N}) = \frac{1 - \sigma_R \Lambda_R}{\sigma_S(0)\, \Lambda_S - \sigma_R \Lambda_R}$$

and then the non-trivial steady state is stable provided that

$$|1 + \tilde{N} f'(\tilde{N})(\sigma_S(0)\, \Lambda_S - \sigma_R \Lambda_R)| < 1$$

or equivalently

$$-2 < \tilde{N} f'(\tilde{N})(\sigma_S(0)\, \Lambda_S - \sigma_R \Lambda_R) < 0. \tag{13}$$

Due to the monotonic decreasing nature of $f(N)$, this leads to the conditions:

1. If $\sigma_S(0)\, \Lambda_S \geqslant 1$ and $\sigma_R \Lambda_R < 1$, the steady state is stable provided that

$$\tilde{N} f'(\tilde{N}) > \frac{-2}{\sigma_S(0)\, \Lambda_S - \sigma_R \Lambda_R}$$

and otherwise stability is lost via a pitchfork bifurcation (oscillations).

2. If $\sigma_S(0)\, \Lambda_S \leqslant 1$ and $\sigma_R \Lambda_R > 1$, the steady state is never stable.

A.3. Case 3

The complete model system has non-trivial steady states obtained from the implicit relation

$$f\left[\frac{\Omega_2(\tilde{P})}{\Omega_1(\tilde{P})}\right] - \Omega_1(\tilde{P}) = 0 \tag{14a}$$

$$\Omega_1(\tilde{P}) = \frac{1 - \sigma_R \Lambda_R}{\Lambda_R \sigma_S(\tilde{P}) - \sigma_R \Lambda_R} \tag{14b}$$

$$\Omega_2(\tilde{P}) = \frac{\tilde{P}(1 - \sigma_P)}{\lambda \Lambda_S(\sigma_S(0) - \sigma_S(\tilde{P}))}. \tag{14c}$$

Since $0 < f(\tilde{N}) < 1$, the non-trivial steady state will arise only if $0 < \Omega_1(\tilde{P}) < 1$ (see (14a)). This requires that either

1. $\Lambda_S \sigma_S(\tilde{P}) > 1$ and $\sigma_R \Lambda_R \leqslant 1$ or
2. $\Lambda_S \sigma_S(\tilde{P}) \leqslant 1$ and $\sigma_R \Lambda_R > 1$.

The values for the susceptible, resistant, and total host levels at steady state are then calculated to be

$$\tilde{S} = \Lambda_S \Omega_2(\tilde{P}) \tag{15a}$$

$$\tilde{R} = \Lambda_2[\,1 - \Omega_1(\tilde{P})\,] \frac{\Omega_2(\tilde{P})}{\Omega_1(\tilde{P})} \tag{15b}$$

$$\tilde{N} = \frac{\Omega_2(\tilde{P})}{\Omega_1(\tilde{P})}, \tag{15c}$$

all of which can be found once $\tilde{P}$ is known.

If $f(N) = \alpha = \text{constant}$, (14a)–(14c) collapses to give

$$\sigma_S(\tilde{P}) = \frac{1 - (1 - \alpha)\, \sigma_R \Lambda_R}{\alpha \Lambda_S}$$

and since $0 < \sigma_S(\tilde{P}) < 1$, we require that

$$0 < 1 - \sigma_R \Lambda_R + \alpha \sigma_R \Lambda_R < \alpha \Lambda_S$$

for the non-trivial steady state to exist.

If $f(N) = \alpha/(1 + \gamma N)$, the non-trivial steady state (14a)–(14c) collapses to the non-linear implicit relation for $\sigma_S(P)$

$$\begin{aligned} \Pi(P) = &-\lambda \Lambda_S(\sigma_S(0) - \sigma_S(P)) \\ &\times [\,\alpha \Lambda_S \sigma_S(P) + (1 - \alpha)\, \sigma_R \Lambda_R - 1\,] \\ &+ \gamma P(1 - \sigma_P)(\Lambda_S \sigma_S(P) - \sigma_R \Lambda_R) = 0. \end{aligned} \tag{16}$$

The Jacobian for the non-trivial steady state is given as

$$J=\begin{bmatrix} \sigma_P-\lambda\sigma'_S(P)S & \lambda(\sigma_S(0)-\sigma_S(P)) & 0 \\ \Lambda_S\sigma'_S(P)\,S\eta(N) & \Lambda_S\sigma_S(P)\,\eta(N) & \Lambda_S\sigma_R\eta(N) \\ \Lambda_R\sigma'_S(P)\,S(1-\eta(N)) & \Lambda_R\sigma_S(P)(1-\eta(N)) & \Lambda_R\sigma_R(1-\eta(N)) \end{bmatrix}, \tag{17}$$

where

$$\eta(N)=f(N)+Nf'(N).$$

The characteristic equation is a cubic function of the form

$$A(\chi)=\chi^3+a_1\chi^2+a_2\chi+a_3=0, \tag{18}$$

where the coefficients a_i are given by the relations

$$a_1=-\sigma_P+\lambda\sigma'_S(P)\,S-\Lambda_R\sigma_R(1-\eta(N)) -\Lambda_S\sigma_S(P)\,\eta(N) \tag{19a}$$

$$a_2=-\lambda(\sigma_S(0)-\sigma_S(P))\,\Lambda_S\sigma'_S(P)\,S\eta(N) +(\sigma_P-\lambda\sigma'_S(P)\,S)(\Lambda_R\sigma_R(1-\eta(N)) +\Lambda_S\sigma_S(P)\,\eta(N)) \tag{19b}$$

$$a_3=0. \tag{19c}$$

Since $a_3=0$, the Jury conditions for linear stability ($|\chi|<1$) are given by

$$A(1)=1+a_1+a_2>0$$

$$-A(-1)=1-a_1+a_2>0$$

$$1>|a_2|.$$

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