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Spatial heterogeneity in three-species, plant–parasite–hyperparasite, systems

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This paper addresses the question of how heterogeneity may evolve due to interactions between the dynamics and movement of three-species systems involving hosts, parasites and hyperparasites in homogeneous environments. The models are motivated by the spread of soil-borne parasites within plant populations, where the hyperparasite is used as a biological control agent but where patchiness in the distribution of the parasite occurs, even when environmental conditions are apparently homogeneous. However, the models are introduced in generic form as three-species reaction–diffusion systems so that they have broad applicability to a range of ecological systems. We establish necessary criteria for the occurrence of population-driven patterning via diffusion-driven instability. Sufficient conditions are obtained for restricted cases with no host movement. The criteria are similar to those for the well-documented two-species reaction–diffusion system, although more possibilities arise for spatial patterning with three species. In particular, temporally varying patterns, that may be responsible for the apparent drifting of hot-spots of disease and periodic occurrence of disease at a given location, are possible when three species interact. We propose that the criteria can be used to screen population interactions, to distinguish those that cannot cause patterning from those that may give rise to population-driven patterning. This establishes a basic dynamical 'landscape' against which other perturbations, including environmentally driven variations, can be analysed and distinguished from population-driven patterns. By applying the theory to a specific model example for host–parasite–hyperparasite interactions both with and without host movement, we show directly how the evolution of spatial pattern is related to biologically meaningful parameters. In particular, we demonstrate that when there is strong density dependence limiting host growth, the pattern is stable over time, whereas with less stable underlying host growth, the pattern varies with time.

Keywords: reaction–diffusion systems; diffusion-driven instability; host–parasite–hyperparasite interaction; spatial heterogeneity; spatio-temporal patterning

1. INTRODUCTION

Spatial heterogeneity is a characteristic feature of many ecological and epidemiological systems and has profound effects on the dynamics of invasion, growth and persistence of populations (Levin 1986). The evolution of spatial heterogeneity may be environmentally driven, when local or regional variability in driving variables imposes a spatial structure on population density. Spatial heterogeneity may also arise, however, in homogeneous environments where the evolution and persistence of the pattern is driven by the growth and movement of interacting populations (Okubo 1980; Murray 1993). The mechanisms for population-driven spatial heterogeneity were originally proposed by Turing (1952), in the context of chemical morphogenesis involving a chemical activator and an inhibitor. Turing (1952) showed that different rates of diffusion of an activator molecule and an inhibitor, together with certain nonlinear interactions in the dynamics of the activator and inhibitor, could lead to the formation of spatial pattern. This type of pattern has subsequently been explored for two-species interactions in ecological contexts by Levin (1986), Segel & Jackson (1972), Mimura & Murray (1978), and are reviewed in Okubo (1980) and Murray (1993). Criteria are now well established for the occurrence of diffusion-driven instability in two-species biological systems.

This paper addresses the question of how heterogeneity may evolve due to interactions between the dynamics and movement of three-species systems involving hosts, parasites and hyperparasites in homogeneous environments. The models are motivated specifically by the spread of soil-borne parasites of plants in the presence of hyperparasites used for biological control. The models, however, are introduced and analysed in a generic form that has much broader applicability to other three-species systems that have coupled reaction dynamics between two or more of the species, and for which diffusive movement of at least two of the species occurs. These encompass a range of applied disciplines including medical as well as botanical epidemiology, bioremediation of soil pollution and wildlife management.

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There is currently much interest in the spatio-temporal dynamics of both harmful and beneficial micro-organisms in soil (Bazin & Lynch 1994; Ritz *et al*. 1994; Gilligan 1994; Gilligan 1995). These comprise a wide range of organisms including plant pathogenic fungi, bacteria and nematodes, as well as endomycorrhizal and ectomycorrhizal fungi, growth-promoting bacteria and organisms involved in the bioremediation of polluted sites. Many of these organisms are involved in three-species interactions involving populations of plant roots, and other microbial antagonists, as well as the symbiont. Moreover, even in comparatively homogeneous environments such as an agricultural crop, the occurrence of micro-organisms is seldom uniform. The initiation of spatial heterogeneity may, in part, be accounted for by stochastic effects, for example in the arrival of inoculum at a given site, or by environmental variation in soil physical conditions. But persistence of the pattern extends long after a uniform equilibrium density of infestation might be expected to occur. We therefore examine criteria for the evolution and maintenance of spatial heterogeneity in a selected three-species interaction involving host, parasite and hyperparasite.

Specifically, we model the host plant as a population of roots, for which diffusion models have been used to describe their spread through soil (Page & Gerwitz 1974). The models also apply to the diffusive spread of plants on a plane. We also consider the occurrence of stationary plant populations with interacting dynamics with a parasite and hyperparasite. The occurrence of fungal parasites is modelled as a change in biomass. This conflates free-living inoculum in soil with inoculum on an infected host into a single variable of parasite biomass. Practically, this equates to the widely used estimates of inoculum density obtained from bioassays of soil cores (Gilligan 1983). Diffusive spread of fungal parasites occurs by hyphal growth and release of spores and other propagules into the soil, where distribution occurs through water or movement of the soil fauna. We do not consider here convectional movement of inoculum by mass flow of water. The hyperparasite is similarly modelled as a change in biomass within soil, with similar diffusive movement. We do not distinguish between hyperparasitic fungi, bacteria or other micro-organisms.

In almost all previous studies, diffusion-driven instability has been discussed and investigated for the simpler two-species system (see, for example, Murray 1993). Othmer & Scriven (1969) did address the question of three-species interactions for a very general situation. Our analysis is similar in nature to theirs, although we investigate linear stability via a different choice of parameters, provide specific necessary (and in some cases sufficient) conditions for diffusion-driven instability, and more importantly, relate the results to the ecological context which motivated our study.

The importance of this work, in light of the previous theoretical study, is both to present conditions for diffusion-driven instability in a similar way to those well-documented for two species in the biological literature (see, for example, Murray 1993), and to discuss the ecological implications of the model results. Moreover, we apply the theory developed in the paper to a specific model for plant–parasite–hyperparasite interactions, to demonstrate how the theory can be applied and the types of predictions which can be made. In fact, it is often as important to understand when pattern cannot be formed through interaction of movement and temporal dynamics as to understand when this can occur. Therefore, this type of work establishes a basic dynamical 'landscape' against which other perturbations, including environmentally driven variations, can be analysed and distinguished from a population-driven pattern.

The paper is built around the general three-species reaction diffusion system described in § 2. Standard linear stability analysis in § 3 provides the dispersion relation used to investigate the role of diffusion in destabilizing the homogeneous stable steady state. In § 4 we investigate conditions for diffusion-driven instability and then apply this theory to a model example in § 5. We conclude with a discussion regarding the importance and value of this analysis both for host–parasite–hyperparasite systems and for more general three-species interactions.

2. THE MODEL

To construct the model, we define the state variables, $H(x,t)$ is host density, $P(x,t)$ is parasite density, and $Q(x,t)$ is hyperparasite density. We make the following assumptions:

1. Growth and movement for each species are not coupled.
2. Movement is essentially at random.
3. Movement is not affected either by the presence of a second species or by the local density of the species.

Moreover, we consider the simplest scenario in which movement is restricted to one dimension (along a line), although the techniques used here can be extended to two space dimensions. We can formulate the above assumptions into a system of three nonlinear reaction diffusion equations which describe the rate of change in local densities of each species such that, the rate of change in local density is equal to the growth and decay of the population plus the random movement (diffusion). This leads to (for the host, parasite and hyperparasite, respectively)

$$\frac{\partial H}{\partial t} = A(H,P,Q) + D_H \frac{\partial^2 H}{\partial x^2}, \tag{1a}$$

$$\frac{\partial P}{\partial t} = B(H,P,Q) + D_P \frac{\partial^2 P}{\partial x^2}, \tag{1b}$$

$$\frac{\partial Q}{\partial t} = C(H,P,Q) + D_Q \frac{\partial^2 Q}{\partial x^2}, \tag{1c}$$

where A, B and C represent the intra- and interspecific dynamics for each of the host, parasite and hyperparasites, and D_H, D_P and D_Q are diffusion coefficients. This embraces the most general relationships amongst the three species, allowing for diffusive movement of all or some of the components. We derive criteria for spatial pattern due to diffusion-driven instability for these most general forms, before considering particular cases, notably when there is no movement of the host ($D_H = 0$), which is particularly appropriate in the

context of plant–parasite–hyperparasite systems. The reaction terms (A, B and C) are similarly analysed in generic form with complete coupling before considering special cases with partially uncoupled dynamics.

Intraspecific components of the reaction terms include birth, death or net growth rates. These may be simple density-independent forms, for example $A(H) = (b - d)H$, giving exponential growth or decay of the host in the absence of the parasite and hyperparasite. More usually, population growth will be density dependent. This usually involves negative inhibition, or more exceptionally synergistic cooperation, at higher densities. The former is typified by the logistic function, $A(H) = rH(1 - H/K)$, where K is the carrying capacity of the host population in the absence of other effects. Synergism may simply be represented by a term in H^2 (Segel & Jackson 1972). The generic forms for A, B and C encompass a range of interspecific effects from partial coupling where, for example, the growth of the host is affected only by the parasite, to complete coupling when the growth of each species is influenced directly by the other two. Interspecific interactions include infection terms for the transmission of infection and the conversion of biomass between host and parasite, and between parasite and hyperparasite. Examples of these terms that encompass classical functional response and numerical responses of parasites to changes in host density are given in Gubbins & Gilligan (1996) and Gilligan *et al.* (1997). Specific examples are analysed in § 5.

The model (1*a–c*) is simpler in form than the system studied by Othmer & Scriven (1969) in which cross diffusion (response to gradients in the other species) was allowed. We consider the problem on some finite domain with zero flux boundary conditions, which mean that there are no external effects on the system.

Diffusion-driven instability occurs if a *stable* steady state is driven unstable by diffusion of the interacting populations (Turing 1952). Mathematically, conditions for diffusion-driven instability are calculated by slightly perturbing the stable steady state of a system and carrying out linear analysis to determine whether the small perturbations will grow or decay over time (see, for example, Murray 1993).

3. LINEAR STABILITY ANALYSIS

If it exists, the homogeneous, stable steady state of the three-species system in (1*a–c*) is given by (H^*, P^*, Q^*) where

$$A(H^*,P^*,Q^*) = B(H^*,P^*,Q^*) = C(H^*,P^*,Q^*) = 0.$$

To linearize about this steady state, we set,

$$\begin{aligned} H(x,t) &= H^* + h(x,t),\\ P(x,t) &= P^* + p(x,t),\\ Q(x,t) &= Q^* + q(x,t), \end{aligned}$$

where $|h(x,t)| \ll H^*$, $|p(x,t)| \ll P^*$ and $|q(x,t)| \ll Q^*$. By convention we choose h, p and q to have the form

$$\begin{aligned} h(x,t) &= h_0 e^{\sigma t + ikx},\\ p(x,t) &= p_0 e^{\sigma t + ikx},\\ q(x,t) &= q_0 e^{\sigma t + ikx}, \end{aligned} \tag{2}$$

where h_0, p_0 and q_0 are constants. The function e^{ikx} is periodic and bounded ($|e^{ikx}| = 1$) and k, the wavenumber, indicates the wavelength of the emergent spatial pattern (Segel 1984). The parameter σ can either be a real number (in which case spatial patterns emerging are stable over time) or a complex number, $\sigma = p + iq$ (in which case spatial patterns vary temporally). In both cases, the sign of the real part of σ (written $Re(\sigma)$) is crucially important to determine whether the pattern will grow or not. In particular if $Re(\sigma) > 0$, the linearized system grows because $|e^{\sigma t}| > 1$ and there will be spatial patterning, but if $Re(\sigma) < 0$ the perturbation decays because $|e^{\sigma t}| < 1$ and the system returns to the homogeneous steady state. Further details concerning linear stability analysis can be found, for example, in Segel (1984) and Murray (1993).

Substituting (2) into (1*a–c*) and ignoring terms higher than linear (because they are too small), we obtain the algebraic system

$$\begin{pmatrix} \sigma - A_H + D_H k^2 & -A_P & -A_Q \\ -B_H & \sigma - B_P + D_P k^2 & -B_Q \\ -C_H & -C_P & \sigma - C_Q + D_Q k^2 \end{pmatrix} \begin{pmatrix} h_0 \\ p_0 \\ q_0 \end{pmatrix} = 0, \tag{3}$$

where the subscripts H, P and Q for A, B and C represent partial derivatives

$$A_H \equiv \frac{\partial A}{\partial H},$$

evaluated at the stable steady state and so on. For non-trivial solutions ($h_0, p_0, q_0 > 0$) we require that

$$\begin{vmatrix} \sigma - A_H + D_H k^2 & -A_P & -A_Q \\ -B_H & \sigma - B_P + D_P k^2 & -B_Q \\ -C_H & -C_P & \sigma - C_Q + D_Q k^2 \end{vmatrix} = 0,$$

which collapses to the dispersion relation

$$\sigma^3 + a_1(k^2)\sigma^2 + a_2(k^2)\sigma + a_3(k^2) = 0, \tag{4}$$

where

$$a_1(k^2) = -A_H - C_Q - B_P + (D_H + D_P + D_Q)k^2, \tag{5a}$$

$$\begin{aligned} a_2(k^2) =& A_H C_Q + A_H B_P + B_P C_Q - C_P B_Q - A_P B_H \\ & - A_Q C_H - k^2(D_Q B_P + D_P C_Q + D_H C_Q \\ & + D_H B_P + D_P A_H + D_Q A_H) \\ & + k^4(D_P D_Q + D_P D_H + D_H D_Q), \end{aligned} \tag{5b}$$

$$\begin{aligned} a_3(k^2) =& - A_H B_P C_Q + A_H C_P B_Q + A_P B_H C_Q - A_P C_H B_Q \\ & - A_Q B_H C_P + C_H B_P A_Q + k^2(- D_Q A_P B_H \\ & - D_P C_H A_Q - D_H C_P B_Q + D_H B_P C_Q + D_P C_Q A_H \\ & + D_Q B_P A_H) - k^4(D_P D_H C_Q + D_H D_Q B_P \\ & + D_P D_Q A_H) + k^6 D_H D_P D_Q. \end{aligned} \tag{5c}$$

Now $Re(\sigma) < 0$ provided that

$$a_1(k^2) > 0,\ a_3(k^2) > 0,\ a_1(k^2)\,a_2(k^2) - a_3(k^2) > 0 \tag{6}$$

(Routh–Hurwitz criteria; see Murray 1993), which are therefore exactly the conditions for the steady state to be stable.

Diffusion-driven instability requires that the *stable, homogeneous* steady state is driven unstable by the interaction of the dynamics and diffusion of the species, and therefore, from the arguments given above we are interested in obtaining conditions such that, without diffusion and with diffusion, respectively

$$\begin{aligned} &Re(\sigma(k^2 = 0)) < 0, \\ &Re(\sigma(k^2 > 0)) > 0, \end{aligned} \tag{7}$$

for some $k^2 > 0$. In the well-documented two-species reaction–diffusion systems (Segel 1984; Murray 1993; Holmes *et al*. 1994), diffusion-driven instability can occur for some finite range of wavenumbers, producing stable spatial patterns (essentially independent of initial conditions) which are small disturbances away from the homogeneous, stable steady state. Extending the system to consider the interaction of three species adds to the complexity of observable patterns (Othmer & Scriven 1969).

Linear stability analysis relies on the fact that perturbations away from the stable steady state are small, and hence the initial conditions which should be used are just small deviations away from the homogeneous steady state. In § 5 where we discuss a particular ecological example, we demonstrate that the spatial patterning predicted by the linear analysis is also attained when more realistic initial conditions, far from the homogeneous steady state, are used. This may not be true in general and will depend on the nature of the dynamical interactions. However, our model example shows that linear analysis can be a useful tool to suggest outcomes for more ecologically realistic initial conditions.

4. CONDITIONS FOR DIFFUSION-DRIVEN INSTABILITY

As is the case with a two-species reaction–diffusion system (Segel 1984; Murray 1993), some general conditions can be obtained regarding the occurrence of diffusion-driven instability. For the three-species system described in this paper, we use (6) and determine whether, and under what conditions, these inequalities can be reversed. If any of the three quantities, given in (6), becomes negative then spatial patterning will be observed. In this paper, we restrict discussion to the situation where k^2 is real and positive although complex wavenumbers may produce a complex spatial structure (Othmer & Scriven 1969). We also show that a change in the sign of $a_3(k^2)$ (provided that $a_2(k^2) > 0$), produces fixed spatial patterning, whereas a change in the sign of $[a_1a_2 - a_3](k^2)$ can produce spatio-temporal patterns.

Condition 1: $a_1(k^2)$. The condition $a_1(0) > 0$ can only be satisfied if at least one of A_H, B_P or C_Q is negative (see (5*a*)). Biologically this can be interpreted as requiring at least one of the species to exhibit stable dynamics when the other two populations are held at constant densities. Provided that this is the case, $a_1(k^2)$ is a linear function of k^2 with positive coefficients, that is

$$a_1(k^2) = \alpha + \beta k^2,$$

Table 1. *Values for the coefficients b, c, d and h in the functions* $a_3(k^2)$ *and* $[a_1a_2 - a_3](k^2)$ *of the cubic (8), which are used to determine conditions for diffusion-driven instability*

	a_3	$a_1a_2 - a_3$
b	$D_HD_PD_Q$	$(D_P+D_Q)\,(D_H^2+D_PD_Q+D_HD_P+D_HD_Q)$
c	$-(D_PD_HC_Q+D_HD_QB_P+D_PD_QA_H)$	$-A_H(D_P+D_Q)(2D_H+D_P+D_Q)-B_P(D_H+D_Q)(D_H+2D_P+D_Q)-C_Q(D_H+D_P)(D_H+D_P+2D_Q)$
d	$D_H(B_PC_Q-C_PB_Q)+D_P(A_HC_Q-C_HA_Q)+D_Q(A_HB_P-A_PB_H)$	$D_H(2A_HC_Q+2A_HB_P+2B_PC_Q+C_Q^2+B_P^2-A_PB_H-A_QC_H)+D_P(2B_PA_H+2B_PC_Q+2C_QA_H+A_H^2+C_Q^2-B_HA_P-B_QC_P)+D_Q(2B_PA_H+2B_PC_Q+2C_QA_H+A_H^2+B_P^2-A_QC_H-B_QC_P)$
h	$a_3(0)$	$a_1(0)a_2(0)-a_3(0)$

where $\alpha = -A_H - C_Q - B_P$ and $\beta = D_H + D_P + D_Q$. This will always be positive if $\alpha > 0$ and therefore diffusion-driven instability cannot be obtained via $a_1(k^2)$ becoming negative.

Conditions 2 and 3: $a_3(k^2)$, $a_1(k^2)a_2(k^2) - a_3(k^2)$. These are both cubic functions of k^2 of the form

$$y(k^2) = b(k^2e^3 + c(k^2e^2 + dk^2 + h, \tag{8}$$

with $b \geqslant 0$ and $h > 0$. If it exists, the minimum turning point for y (which is calculated from $dy/dk = 0$, $d^2y/d^2(k^2) > 0$), occurs at

$$k^2 = k_{TP}^2 = \frac{-c + [c^2 - 3bd]^{0.5}}{3b}. \tag{9}$$

Now k_{TP}^2 is real and positive if

$$d < 0 \text{ or } c < 0 \text{ (and } c^2 > 3bd), \tag{10}$$

and $y(k_{TP}^2) < 0$ if

$$2c^3 - 9bcd - 2(c^2 - 3bde^{1.5} + 27bh^2 < 0. \tag{11}$$

Combining (10) and (11) provides sufficient conditions to produce diffusion-driven instability—the coefficients b, c, d and h for $a_3(k^2)$ and $[a_1a_2 - a_3](k^2)$ are given in table 1. Substituting for these coefficients into (11) produces a complicated expression from which little insight can be gained. Therefore, we concentrate on the conditions $c < 0$ and $d < 0$ in (10) which are a necessary prerequisite for the spatial patterning in which we are interested.

By comparing the requirements for a stable, homogeneous steady state ($a_1(0) > 0, a_3(0) > 0, [a_1(0)a_2(0) - a_3(0)] > 0$), with those required to give spatial patterning (at least one of $a_1(k^2) < 0$, $a_3(k^2) < 0$, $[a_1(k_2^2(k^2) - a_3(k^2)] < 0$ for some non-zero k^2), we obtain some general *necessary* conditions on the dynamics and diffusion coefficients for the three species. These are given in table 2 and are consistent with those for two species (see Murray 1993)—in particular the rate of diffusion for at least one of the species must differ from the others. Provided that all species move (D_P, D_Q and $D_H > 0$) $a_3(k^2) \to \infty$ as $k^2 \to \infty$, $[a_1a_2 - a_3](k^2) \to \infty$ as $k^2 \to \infty$, because the coefficient b in both expressions is positive (table 2), and only a finite range of wavenumbers

Table 2. *Necessary (but not sufficient) conditions leading to diffusion-driven instability for the general three-species model, using the criteria in equation* (10)

(These conditions were obtained by comparing the expressions in table 2 with the expressions for $a_1(0)$, $a_3(0)$ and $[a_1a_2 - a_3](k^2)$ from (5*a–c*).)

	a_3	$a_1a_2 - a_3$
$c<0$	one of A_H, B_P and C_Q different sign	ditto
	at least one different diffusion coefficient	ditto
$d<0$	at least one different diffusion coefficient	$(2D_Q+D_H+D_P)B_HA_P$ $+(2D_P+D_H+D_Q)A_QC_H$ $+(2D_H+D_P+D_Q)B_QC_P<0$
	at least one of following negative $B_PC_Q-C_PB_Q$ $A_HC_Q-C_HA_Q$ $A_HB_P-A_PB_H$	

can be driven unstable. This means that the wavelength of emergent patterns can be estimated and is essentially independent of the small initial disturbance from the stable, homogeneous population levels. If one of the species does not diffuse ($D_H = 0$ say), then $b(a_3) = 0$ and it is possible to obtain an infinite range of unstable wavenumbers as we describe in § 4.

(a) *Fixed or temporally varying patterns?*

The analysis above provides us with some necessary conditions under which the stable steady state is driven unstable, but it does not describe the temporal nature of that instability. To do that we must determine whether σ is real (implying fixed spatial patterns) or complex (implying spatio-temporal patterns), and so we set $\sigma = p + iq$ and substitute into the dispersion relation obtained in (4). Comparing real and imaginary parts gives

$$p^3 - 3pq^2 + a_1(k^2)(p^2 - q^2) + a_2(k^2)p + a_3(k^2) = 0, \quad (12a)$$

$$3p^2q - q^3 + a_1(k^2)2pq + a_2(k^2)q = 0. \quad (12b)$$

From (12*b*), $q = 0$ (in which case σ is real and given by (4)) or

$$q^2 = 3p^2 + 2a_1p + a_2.$$

This expression for q^2 can be substituted into (12*a*) to give

$$\Omega(p) = 8p^3 + 8a_1p^2 + 2p(a_1^2 + a_2) + a_1a_2 - a_3 = 0. \quad (13)$$

There are two ways in which (13) can admit a positive real root:

1. If $[a_1a_2 - a_3](k^2) < 0$, $\Omega(0) < 0$, and since $\Omega \to +\infty$ as $p \to +\infty$ there must be at least one positive root.
2. Alternatively, $\Omega(p)$ may have a turning point for some p

$$p = \tilde{p} = \frac{-2a_1 + [a_1^2 - 3a_2]^{0.5}}{6} > 0,$$

such that $\Omega(\tilde{p}) < 0$. A necessary condition for this case is that $a_1^2 + a_2 < 0$ in order that $\tilde{p} > 0$.

Table 3. *Summary of the effects of coefficients from the dispersion relation* (4) *on the nature of the spatial pattern which is observed*

a_3	$a_1a_2-a_3$	a_2	comments	pattern
+	+	+	none	no pattern
+	−	+	none	spatio-temporal
−	+	+	none	temporally stable
−	−	−	$p>p_c$	spatio-temporal
−	−	−	$p<p_c$	temporally stable
+	−	−	$p>p_c$	spatio-temporal
+	−	−	$p<p_c$	temporally stable
−	+	−	$a_1^2 + a_2 > 0$	temporally stable
−	+	−	$a_1^2 + a_2 < 0, \Omega(\tilde{p}) > 0$	temporally stable
−	+	−	$a_1^2 + a_2 < 0, \Omega(\tilde{p}) < 0$ $\tilde{p} \leqslant p < p_c$	temporally stable
−	+	−	$a_1^2 + a_2 < 0, \Omega(\tilde{p}) < 0$ $p > p_c$	spatio-temporal

If $a_2(k^2) > 0$ then there is always a real value of q corresponding to a positive value of p so spatio-temporal patterns occur. If $a_2(k^2) < 0$, there is more restriction because solutions are spatio-temporal if

$$p > p_c = \frac{-a_1 + [a_1^2 - 3a_2]^{0.5}}{3} > \tilde{p},$$

and otherwise they are stable spatial structures (because q^2 is negative, q is imaginary and therefore iq is real).

These results are summarized in table 3 and are analogous to those obtained in Othmer & Scriven (1969). We show below (§5), two variations of a model which can realistically describe the ecology of a plant–parasite–hyperparasite interaction, one of which has temporally stable spatial structure and the other of which varies with time.

(b) *No host movement*

One realistic simplification which can be made in the three-species system, and which is particularly relevant to a plant system, is to assume that the host cannot move via diffusion ($D_H = 0$). This has no effect on the structure either of $a_1(k^2)$ (still linear in k^2), or of $[a_1(k^2)a_2(k^2) - a_3(k^2)]$ (still cubic in k^2). However, it reduces $a_3(k^2)$ to the quadratic function of k^2,

$$a_3(k^2) = -D_PD_QA_Hk^4 + k^2(D_P[C_QA_H - C_HA_Q] + D_Q[B_PA_H - A_PB_H]) + a_3(0), \quad (14)$$

which can be used to obtain sufficient conditions for diffusion-driven instability (by $a_3(k^2)$ becoming negative for some k^2). In particular, we vary A_H and discuss the consequences for stability of the model system. The quantity A_H is the rate of change in host density when the system is close to its steady state, and the other two

densities (parasite and hyperparasite) are fixed at their equilibrium values.

If $A_H > 0$, then $a_3(k^2) \to -\infty$ as $k^2 \to \infty$, and in this case a pattern evolves which will depend on the initial perturbation. If $A_H = 0$, an infinite range of wavenumbers can again produce diffusion-driven instability, but now we require that $D_P C_H A_Q + D_Q A_P B_H > 0$ (the coefficient of k^2 in (14) is negative). Finally if $A_H < 0$, $a_3(k^2) < 0$ only if

$$F_1 = D_P[C_Q A_H - C_H A_Q] + D_Q[B_P A_H - A_P B_H] < 0, \tag{15a}$$

and

$$F_2 = (D_P[C_Q A_H - C_H A_Q] + D_Q[B_P A_H - A_P B_H])^2 + 4 D_P D_Q A_H a_3(0) > 0. \tag{15b}$$

Intuitively these results make sense—as A_H decreases, the system becomes more stable because there is more self-regulation by the host. Therefore, the requirements on the model parameters to produce a spatial structure should get increasingly more severe. These simplified conditions can be used to determine the role of the different model parameters in producing diffusion-driven instability as we demonstrate with the biological example in § 5.

(c) *Uncoupled dynamics*

To complete this section we briefly discuss the case when the dynamics are partially uncoupled. Suppose, for example, that the host does not affect the dynamics of the parasite and hyperparasite such that the dynamics are of the form

$$A(H,P,Q)\ \ B(P,Q)\ \ C(P,Q). \tag{16}$$

The dispersion relation (4) simplifies to

$$[\sigma - A_H + D_H k^2], \quad [\sigma^2 + \sigma(-B_P - C_Q + D_P k^2 + D_Q k^2) + B_P C_Q - B_Q C_P + k^2(-D_Q B_P - D_P C_Q) + D_P D_Q k^4] = 0. \tag{17}$$

In this case, diffusion-driven instability is determined by the interaction of the dynamics of the parasite (P) and the hyperparasite (Q) respectively. 'Turing' instability in two-species systems has been well-documented elsewhere and requires different diffusion coefficients and B_P and C_Q to be of opposite signs. These conditions are similar to the ones found here for the three-species interaction. The spatial dynamics of the host are unaffected by the parasite or hyperparasite and since we are perturbing about a stable steady state, this means that the host population returns to a homogeneous density distribution. Analogous results are obtained in the other two complementary situations where host and parasite dynamics do not depend on the hyperparasite density ($A(H,P)$, $B(H,P)$, $C(H,P,Q)$), and where host and hyperparasite dynamics do not depend on parasite density ($A(H,Q)$, $B(H,P,Q)$, $C(H,Q)$).

5. APPLICATION OF THE THEORY

To demonstrate the theory described above, we consider a specific model for plant–parasite–hyperparasite interactions which can exhibit both stable and temporally varying spatial patterns.

The dynamics of the plant are split into two parts. In the absence of the parasite, the plant population grows logistically with growth rate r and carrying capacity K. Parasite presence causes plant mortality at a rate of α per unit parasite. Biomass depletion or mortality of the plant results in production of the parasite at a rate b per unit plant. Interaction with the hyperparasite reduces parasite levels according to a Holling type II predation rate with μ and γ measuring the basic mortality and degree of density dependence, respectively. Finally, hyperparasite production occurs at a rate λ per parasite per hyperparasite, and the hyperparasite has a natural decay rate of d. Combining all of these gives the following forms for A, B and C:

$$A = rH\left[1 - \frac{H}{K}\right] - \alpha PH, \tag{18}$$

$$B = bPH - \frac{\mu P}{1 + \gamma P} Q, \tag{19}$$

$$C = \lambda P - dQ. \tag{20}$$

Note that as K becomes very large, the intraspecific density dependence acting on the plant population is reduced such that as $K \to \infty$, plant growth is essentially exponential. The particular form of the functions was dictated by the desire to use the simplest possible model formulation which was both a realistic (if simplistic) representation of the ecology, and at the same time able to demonstrate the types of spatial patterning described above.

(a) *Non-dimensionalization*

It is convenient to make a change of variable which reduces the number of model parameters and allows comparison between relative magnitudes of the parameters. Defining L as a typical length scale we take

$$x^+ = \frac{x}{L},\ t^+ = \frac{D_Q}{L^2} t,\ \delta_H = \frac{D_H}{D_Q},\ \delta_P = \frac{D_P}{D_Q},\ H^+ = \frac{H}{H^*},$$
$$P^+ = \frac{P}{P^*},\ Q^+ = \frac{Q}{Q^*},\ \tau = \frac{L^2 r}{D_Q},\ K^+ = \frac{K}{H^*},\ d^+ = \frac{d}{r},$$
$$\mu^+ = \frac{Q^*}{r}\mu,\ \gamma^+ = \gamma P^*,$$

where P^*, H^* and Q^* are the steady state population levels for the original dimensional system, as $K \to \infty$ (exponential plant growth). With this choice, (1a–c) becomes

$$\frac{\partial H}{\partial t} = \tau\left[H\left(1 - \frac{H}{K}\right) - PH\right] + \delta_H \frac{\partial^2 H}{\partial x^2}, \tag{21a}$$

$$\frac{\partial P}{\partial t} = \tau\mu\left[\frac{PH}{1+\gamma} - \frac{PQ}{1+\gamma P}\right] + \delta_P \frac{\partial^2 P}{\partial x^2}, \tag{21b}$$

$$\frac{\partial Q}{\partial t} = \tau d(P - Q) + \frac{\partial^2 Q}{\partial x^2}, \tag{21c}$$

dropping the superscript for notational simplicity.

(b) *Model analysis*

The model system (21a–c) has a homogeneous steady state

Table 4. *Coefficients which play a role in determining conditions for diffusion-driven instability, for the model system* (21*a–c*) *where* $\phi = P^*/(1+\gamma P^*)$

	no diffusion: coefficients for a stable steady state
$a_1(0)$	$\tau\left[\frac{(1+\gamma)\phi}{K}+d-\mu\gamma\phi^2\right]$
$a_1a_2-a_3(0)$	$\tau^3\left[\frac{(1+\gamma)\phi}{K}+d-\mu\gamma\phi^2\right]\left[\frac{d(1+\gamma)\phi}{K}-\frac{(1+\gamma)\mu\gamma\phi^3}{K}+\frac{d\mu\phi}{(1+\gamma P^*)}+\mu P^*\phi\right]-d\tau^3\mu P^*\phi\left[1+\frac{1+\gamma}{K(1+\gamma P^*)^2}\right]$
$a_3(0)$	$d\tau^3\mu\phi P^*\left[1+\frac{1+\gamma}{K(1+\gamma P^*)^2}\right]$
	diffusion: coefficients with a role in spatial patterning
$c(a_3)$	$\tau\left[\delta_P\delta_H d-\delta_H\mu\gamma\phi^2+\delta_P\frac{(1+\gamma)\phi}{K}\right]$
$c(a_1a_2-a_3)$	$\tau\left[(\delta_H+\delta_P)(\delta_H+\delta_P+1)d-(\delta_H+1)(\delta_H+2\delta_P+1)\mu\gamma\phi^2+(\delta_P+1)(2\delta_H+\delta_P+1)\frac{(1+\gamma)\phi}{K}\right]$
$d(a_3)$	$\tau^2\left[\delta_H\frac{d\mu\phi}{(1+\gamma P^*)}+\delta_P\frac{d(1+\gamma)\phi}{K}+\left(\mu P^*\phi-\frac{\mu\gamma(1+\gamma)\phi^3}{K}\right)\right]$
$d(a_1a_2-a_3)$	$2\tau^2(\delta_H+\delta_P+1)\phi\left[\frac{d(1+\gamma)}{K}-\frac{(1+\gamma)\mu\phi^2}{K}-d\mu\gamma\phi\right]+\tau^2(\delta_H+\delta_P)(d^2+\mu P^*\phi)$
	$+(1+\delta_H)(\mu^2\gamma^2\phi^4)+\tau^2(1+\delta_P)\left[d\mu\phi+\frac{(1+\gamma)^2\phi^2}{K^2}\right]$

$$P^*=\frac{-\mathcal{J}+(\mathcal{J}^2+4K^2\gamma e^{0.5}}{2K\gamma},\ Q^*=P^*,\ H^*=\frac{(1+\gamma)P^*}{1+\gamma P^*}, \tag{22}$$

where $\mathcal{J}=1+\gamma+K(1+\gamma)$ and the Jacobian matrix is

$$\begin{pmatrix} A_H & A_P & A_Q \\ B_H & B_P & B_Q \\ C_H & C_P & C_Q \end{pmatrix}=\tau\begin{pmatrix} \frac{-(1+\gamma)P^*}{K(1+\gamma P^*)} & \frac{-(1+\gamma)P^*}{1+\gamma P^*} & 0 \\ \frac{\mu P^*}{1+\gamma} & \frac{\mu\gamma P^{*2}}{(1+\gamma P^* e^2} & \frac{-\mu P^*}{1+\gamma P^*} \\ 0 & d & -d \end{pmatrix}. \tag{23}$$

Note that A_H and C_Q are negative, while B_P is positive. The coefficients c and d of the cubic functions $a_3(k^2)$ and $[a_1a_2-a_3](k^2)$ together with the expressions for $a_1(0)$, $a_3(0)$ and $[a_1a_2-a_3](0)$ are given in table 4 with

$$\phi=\frac{P^*}{1+\gamma P^*},$$

from which the following observations can be made:

1. $a_1(0)>0$, if d (the ratio of plant growth to hyperparasite death rate) is sufficiently large, and/or μ (a measure of hyperparasite-induced parasite mortality to plant growth rate) is sufficiently small.
2. $a_3(0)$ is always positive.
3. $c(a_3)<0$ requires that δ_P is smaller than 1, and/or δ_H (conditions on the relative diffusion rates).
4. $c(a_1a_2-a_3)<0$ could occur if $\delta_P<\delta_H$.
5. $d(a_3)>0$ for $\gamma\leqslant 1$ is a minimum requirement. The parameter γ is related to the degree of density dependence in hyperparasite-induced parasite mortality.
6. $d(a_1a_2-a_3)<0$ will require large γ.

These observations are not exhaustive, and may not be sufficient on their own, but they serve to illustrate the ways in which the theory can be related back to the model parameters. In particular, it is striking to note that conditions on c in both cases are related to movement parameters, and those on d are associated with the dynamical interactions. Moreover, these observations agree with the conditions given in table 2. With reference to these, it is apparent, using (23), that the necessary conditions for $c(a_3)<0$, $c(a_1a_2-a_3)<0$ and $d(a_1a_2-a_3)<0$ can be satisfied, but that $d(a_3)$ is always positive if $\gamma\leqslant 1$.

In the case of no host movement ($\delta_H=0$), $a_3(k^2)$ reduces to a quadratic function and more specific criteria can be obtained as we show in § 5(d) below.

(c) *Numerical simulations*

The conditions obtained from the coefficients were used to find parameter domains in which spatial patterning (both stable and temporally varying) would be observed. With the value of K relatively small, the density dependent constraints on the host are large and in this case $a_3(k^2)$ becomes negative (figure 1), suggesting that the spatial patterning is stable over time (σ is real). This is verified in two model simulations which are shown in figures 2 and 3. The difference between these two simulations is not in

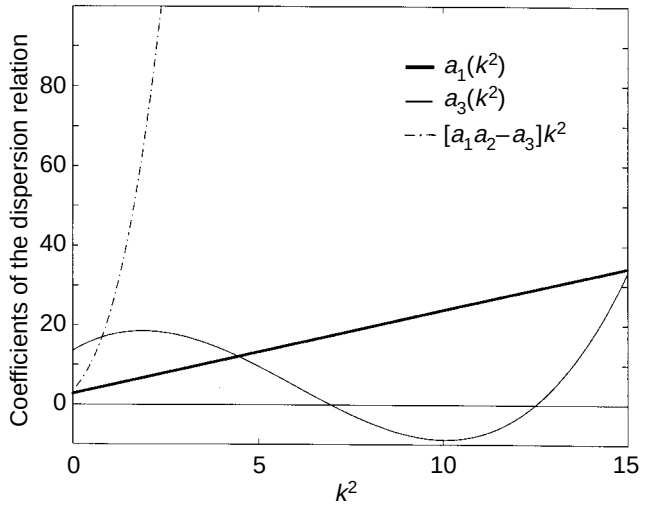

Figure 1. Coefficients of the dispersion relation (4) for the dimensionless system (21*a–c*). For diffusion-driven instability to arise, at least one of the coefficients (in this case $a_3(k^2)$) must be negative for some range of k^2. The model parameters used here are: $K = 10$, $\tau = 1$, $\mu = 15$, $d = 5$, $\gamma = 4$, $D_H = 1$ and $D_P = 0.1$. The instability arises because $c(a_3)$ is negative (whilst $[c^2 - 3bd](a_3) > 0$).

parameter values but in initial distributions. In figure 2, the homogeneous steady state was subjected to a small random perturbation to give initial conditions consistent with the linear analysis carried out above. In figure 3, ecologically realistic initial conditions were used to represent a point source of parasite inoculum in a host and hyperparasite system exhibiting small random fluctuations about the homogeneous steady state. In both cases (figures 2 and 3), the model system settles to a time-independent spatially heterogeneous distribution with the same wavelength. The translation of the spatial pattern between figures 2 and 3 is caused by the differences in initial disturbance. The important point is that the wavelength in the two patterns is essentially the same, indicating that the pattern predicted by the linear theory is an attractor for a wide range of initial conditions. For large values of K, host growth is essentially exponential (in the absence of parasite induced mortality), $[a_1a_2 - a_3](k^2)$ is negative for some values of k^2 (figure 4) and the resulting pattern, as shown in figure 5 for initial conditions consistent with the application of linear theory, varies with time. In figure 6 we show the model solution for the initial conditions used in figure 3. Again, we obtain the same spatio-temporal pattern observed in figure 5 and conclude, in this case, that the model solution predicted by linear theory is a good indicator for the behaviour of the solution with more general initial conditions.

These results are intuitively pleasing, not only because the behaviours predicted by the linear theory extend to more realistic initial conditions, but also because the less stable underlying host growth patterns correspond to a less stable spatial structure.

(d) *No host movement—sufficient conditions for patterning*

One simplifying assumption which is particularly appropriate in the context of a plant–parasite–hyperparasite system is that the plant (host) does not move, and hence, $D_H = 0$.

As described in §4(b), this reduces $a_3(k^2)$ to a quadratic function but leaves $[a_1a_2 - a_3](k^2)$ as a cubic. Therefore we consider sufficient conditions for diffusion-driven instability via a sign change in $a_3(k^2)$ only.

If $K \to \infty$, $A_H \to 0$ and $a_3(k^2)$ is the linear function,

$$a_3(k^2) = -A_P B_H k^2 + a_3(0),$$

which is a positive function for all $k^2 \geqslant 0$ because A_P and B_H are of opposite sign; therefore spatial patterning will not arise due to a change in the sign of $a_3(k^2)$, although it still could via a change in $[a_1a_2 - a_3](k^2)$.

With $K \ll \infty$, A_H is negative and the conditions $F_1 < 0$, $F_2 > 0$ given in equations (15*a*)–(15*b*) are

$$F_1 = \tau^2\left[\delta_P\frac{d(1+\gamma)\phi}{K} - \frac{(1+\gamma)\mu\gamma\phi^3}{K} + \mu\phi P^*\right] < 0, \quad (24a)$$

$$F_2 = \tau^4\left(\left[\delta_P\frac{d(1+\gamma)\phi}{K} - \frac{(1+\gamma)\mu\gamma\phi^3}{K} + \mu\phi P^*\right]^2 - \frac{4\delta_P d\mu(1+\gamma)P^*\phi^2}{K}\left[1 + \frac{1+\gamma}{K(1+\gamma P^* e^2}\right]\right) > 0. \quad (24b)$$

These inequalities were used as critical functions to determine the role of different parameter values (all of which satisfy conditions for the homogeneous steady state to be stable in the absence of diffusion), in diffusion-driven stability. In figure 7 we show the inequalities (24*a–c*) from which it can be seen that diffusion-driven instability (via $a_3(k^2)$ becoming negative) is more likely to occur if

1. K, the dimensionless carrying capacity is small (figure 7*a*).
2. μ, the dimensionless maximum hyperparasite-induced parasite mortality is large (figure 7*b*).
3. γ, the dimensionless measure of density dependence in parasite mortality is small (figure 7*c*).
4. δ_P, the ratio of the parasite to hyperparasite diffusion rates is comparatively small (figure 7*d*).
5. d, the hyperparasite mortality rate is small (figure 7*e*).

The parameter τ plays no part in this process since it is a factor of F_1 and hence if F_1 is negative for some parameter combination, altering τ will not change its sign.

6. DISCUSSION

We have developed and tested a theoretical framework for the analysis of population-driven spatial pattern in three-species systems, involving diffusion-driven instability. Criteria are derived that identify certain broad classes of population behaviour, in which fixed (figures 2 and 3) or temporally varying (figures 5 and 6) spatial pattern amongst the species can or cannot occur by diffusion-driven instability in otherwise homogeneous environments. This establishes a basic dynamical 'landscape' against which other perturbations, including environmentally driven or stochastic variations, can be analysed and distinguished from a population-driven pattern.

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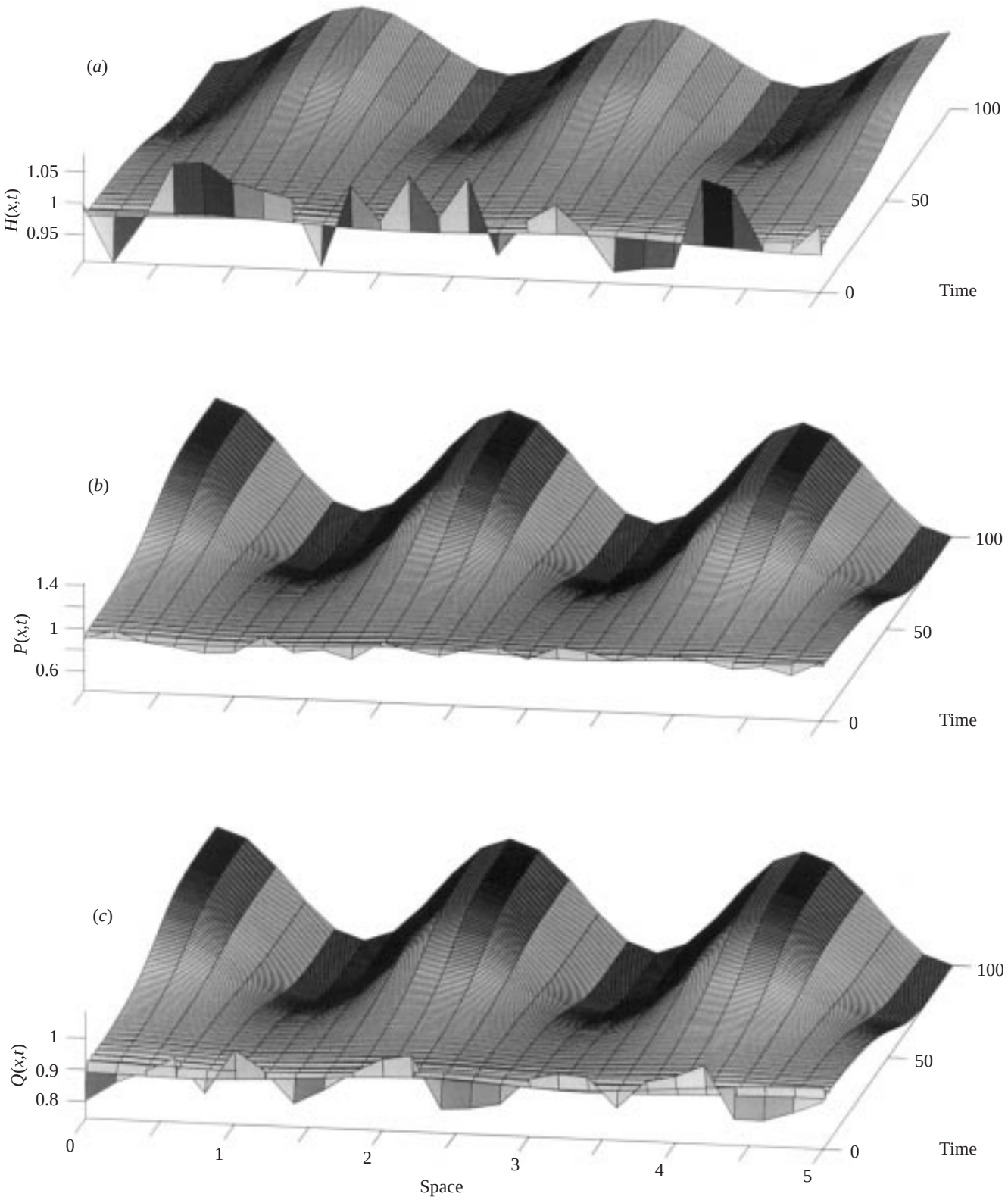

Figure 2. Model solution using the parameters given in figure 1. The initial condition for each species was given by small, random fluctuations about its stable, homogeneous steady state level. The spatial pattern which is produced by diffusion-driven instability is stable over time and has an approximate dimensionless wavelength of two units. (*a*) Host; (*b*) parasite; (*c*) hyperparasite.

The models are introduced in generic form so that they have broad applicability to a range of interacting populations. These are initially analysed with complete coupling amongst all three species. Subsequently, restricted classes of interaction are considered, where there is no diffusion of one species and when the coupling amongst populations is reduced. The models are motivated for host–parasite–hyperparasite systems, which comprise two overlapping sets of activators and inhibitors (the host and parasite, and the parasite and hyperparasite, respectively). Amongst hosts, parasites and hyperparasites, spatial patterning can lead to hot-spots of disease, where

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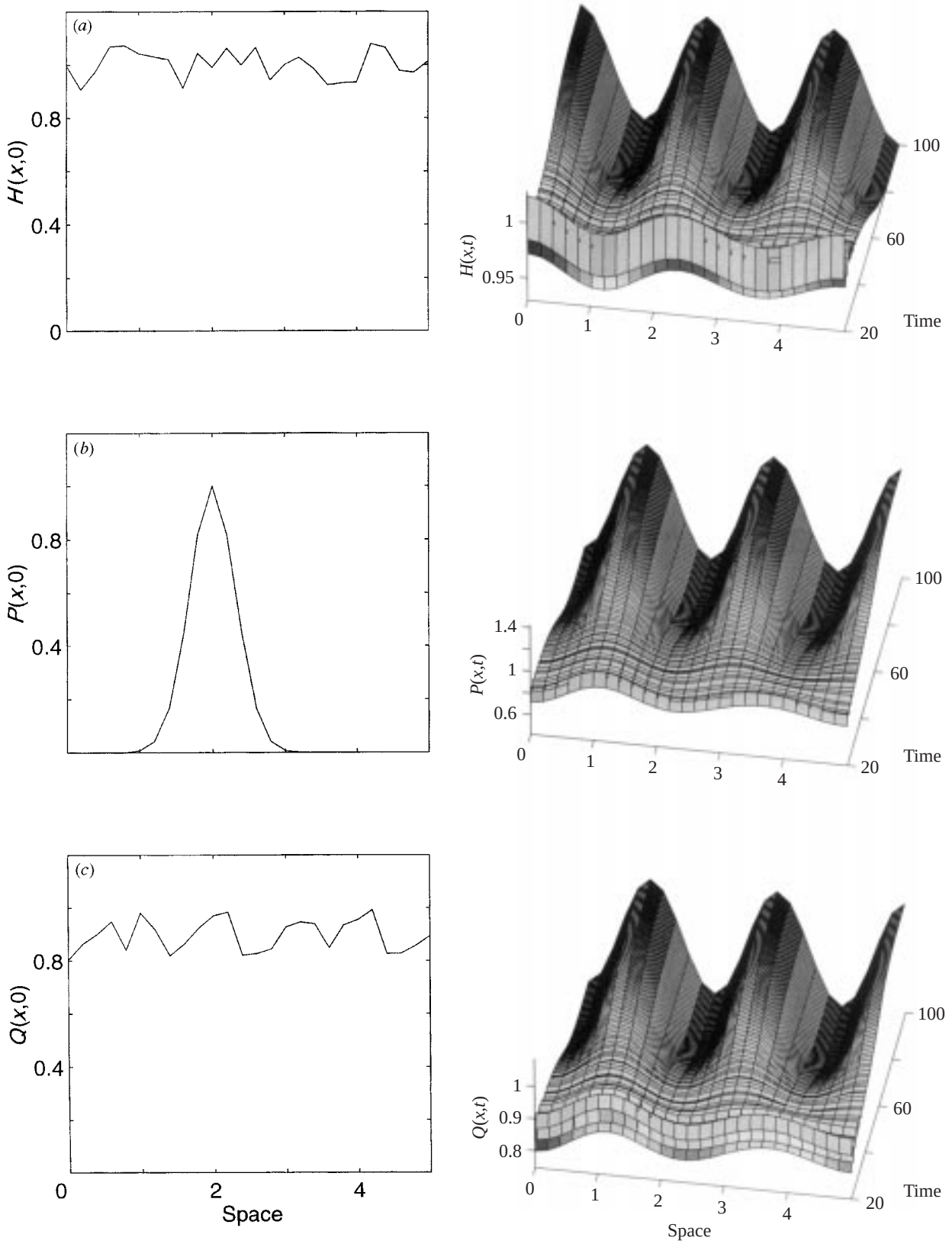

Figure 3. Model solution using the parameters given in figure 1. The initial condition for the host and hyperparasite was as for figure 2. The initial condition for the parasite was given by the expression $P(x,0) = \exp[-5(x-2)^2]$. On the left-hand side we show these initial conditions, and on the right-hand side the stable spatial pattern which is attained. The solution was split in this way because of large transient population levels, which meant that the amplitude of the final distributions was not easy to see if the complete time series was shown in a single figure. The final spatial distribution of the three species matches that given in figure 2, subject to translation with the same dimensionless wavelength (approximately two units). (*a*) Host; (*b*) parasite; (*c*) hyperparasite.

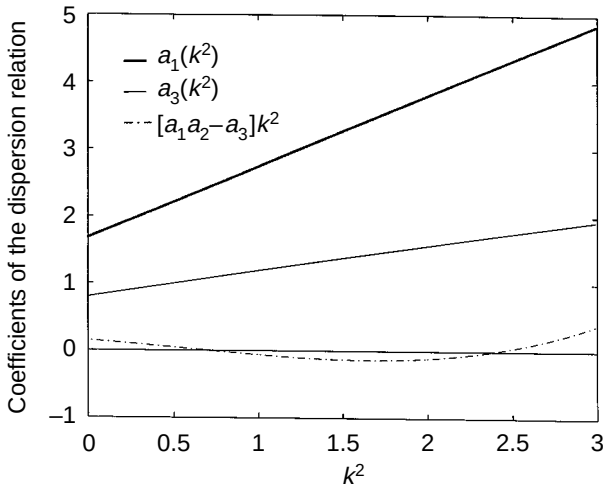

Figure 4. Coefficients of the dispersion relation (4) for the dimensionless system (21*a–c*). For diffusion-driven instability to arise, at least one of the coefficients (in this case $a_1(k^2)a_2(k^2) - a_3(k^2)$) must be negative for some range of k^2. Model parameters used here are: $K = 500$, $\tau = 1$, $\mu = 2$, $d = 2$, $\gamma = 4$, $D_H = 0.04$ and $D_P = 0.02$. Both $c(a_1a_2 - a_3)$ and $d(a_1a_2 - a_3)$ are negative with $[c^2 - 3bd](a_1a_2 - a_3) > 0$.

inoculum density of a pathogenic parasite and its hyperparasite increases and the host decreases (cf. figures 2–3 and 5–6). We distinguish between necessary conditions, given in table 2 for the generic models, and sufficient conditions, derived in §§4(b) and 5(d) for restricted cases with no host movement. Necessary conditions imply only that patterning may occur: failure to satisfy the necessary conditions in table 2, however, means that patterning due to diffusion-driven instability will not occur. Sufficient conditions infer that patterning will occur. We propose that the necessary criteria in table 2 can be used to screen hypothetical population interactions, to distinguish interactions that cannnot cause patterning from those that may give rise to population-driven patterning. Table 3 may then be used to identify the nature of the spatial pattern, if it is likely to occur.

Diffusion-driven instability requires a stable equilibrium amongst the species in the absence of movement (Turing 1952). Moreover, spatial patterning in population density cannot arise in three-species systems, without coupled reaction dynamics between two or more of the species and diffusive movement of at least two of the species. In common with two-species interactions, sufficient criteria impose quite restrictive conditions on the occurrence of diffusion-driven instability. They require that diffusion is faster in the inhibitor (cf. the hyperparasite), than in the activator (cf. the parasite) species. They also require that at least two of the three possible pairs of partial derivatives (A_H, B_P, C_Q), for the community (May 1974) or Jacobian matrix, differ in sign (cf. equation (23)). Here the partial derivatives define the rate of change of the population close to the equilibrium density. The requirement that two out of the possible three of the (intraspecific) diagonal elements of the community matrix differ, is less restrictive, however, than for two-species systems, where both must differ (Okubo 1980). This raises an interesting question for future work on whether this result compounds for a large system: that is, if a large number of species interact, are small differences in their respective diffusion coefficients sufficient to give rise to spatial patterning via diffusion-driven instability?

Three-species systems also admit the possibility of temporally varying spatial patterns (Othmer & Scriven 1969), which cannot occur in two-species systems. These spatio-temporal oscillations, arising from diffusion-driven instability, can be distinguished from temporal oscillations which arise in the absence of movement, by inspecting the stability of the dynamical interaction in the spatially homogeneous case. Again, it would be interesting to investigate the effect of random movement on a system of three or more species in which their dynamical interactions alone exhibit temporal oscillations. The oscillations are of practical importance because they can lead to the apparent drifting of hot-spots of disease and periodic occurrence of disease at a given location, even under homogeneous agricultural conditions. They may also give rise to drifting in the location of beneficial micro-organisms in bioremediation and biological control.

The requirement for a markedly faster rate of diffusion of the inhibitor compared with an activator further restricts the occurrence of this form of population-driven pattern. Parasites may diffuse faster than their hosts, when they are spread by random movement of a vector such as an aphid or nematode, or when they are facultative parasites with a saprophytic lifestyle, as with many fungal parasites. Data are scarce for the movement of parasitic or hyperparasitic biological control agents, but here, nematodes are likely to move faster than bacteria and fungi.

Experimental testing of the hypotheses for spatial patterning in three-species interactions requires spatially homogeneous environmental conditions in order to distinguish between population-driven and environmentally driven pattern. Homogenous conditions can be obtained in certain natural and agricultural environments, where, for example, soil physical properties are uniform at the scale of interest and water is not limiting. Immediate experimental progress, however, is likely to come from the use of microcosm experiments, in which environmental conditions are strictly controlled and treatments can be replicated. Three-species interactions involving plant or invertebrate hosts with bacterial, fungal or nematode parasites and hyperparasites are ideally suited for experimentation in microcosms: for example, epidemics involving a host (radish), a fungal parasite (*Rhizoctonia solani*), and a biological control agent in the form of a fungal hyperparasite (*Trichoderma viride*), can be completed within 15–20 days in replicated microcosms (Kleczkowski *et al*. 1996, 1997; Bailey & Gilligan 1997). Densities of many microbial parasites and hyperparasites can be assayed by molecular methods (Dewey *et al*. 1997). The radish microcosm is well suited to the analysis of movement of the parasite and hyperparasite with a static host population. Similar systems can be devised for other combinations of fungal, bacterial or nematode parasites, or hyperparasites, while a change of host to a grass or cereal yields a dynamically changing root population. Experimental testing, using microcosms is not restricted to plant disease: it can also be applied to aerial pathogens, to invertebrate hosts and

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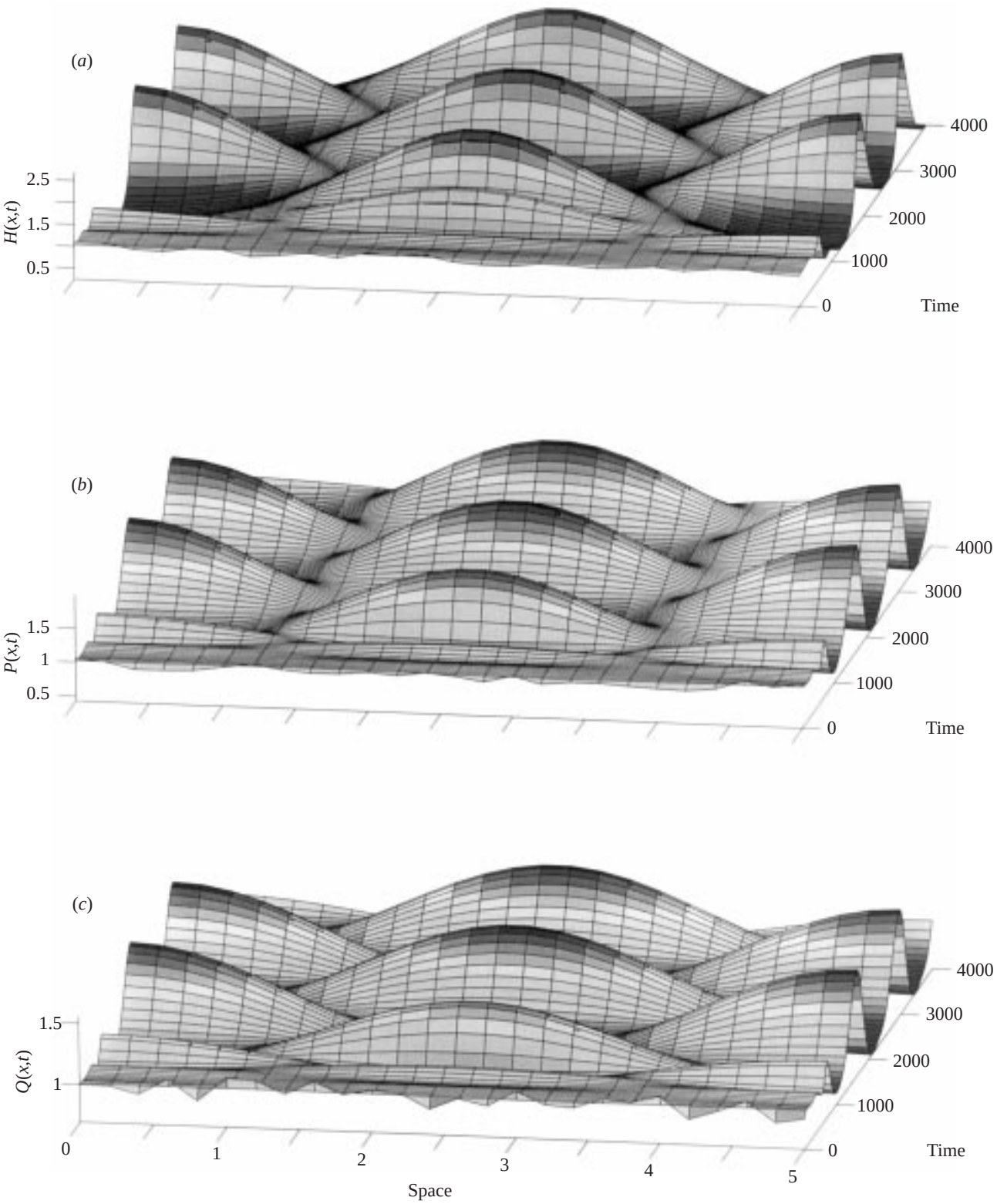

Figure 5. Model solution using the parameters given in figure 4. The initial condition for each species was given by small random fluctuations about its stable, homogeneous steady state level. The spatial pattern which is produced by diffusion-driven instability varies temporally, and has a dimensionless spatial wavelength approximately equal to five units and dimensionless temporal wavelength of around 1200 units. (*a*) Host; (*b*) parasite; (*c*) hyperparasite.

to interacting species of beneficial and harmful micro-organisms in soils, foods and other stored products. We propose that once the reaction terms for interacting species are identified and tested, and preliminary estimates are derived for rates of diffusion, the necessary conditions in table 2 can be used to separate those interactions where population-driven patterning is not possible by diffusion-driven instability, from those where it may occur. Subsequent microcosm experiments may then be used to test the hypotheses, to revise models and to assist in devising sampling protocols for efficient characterization of spatial pattern.

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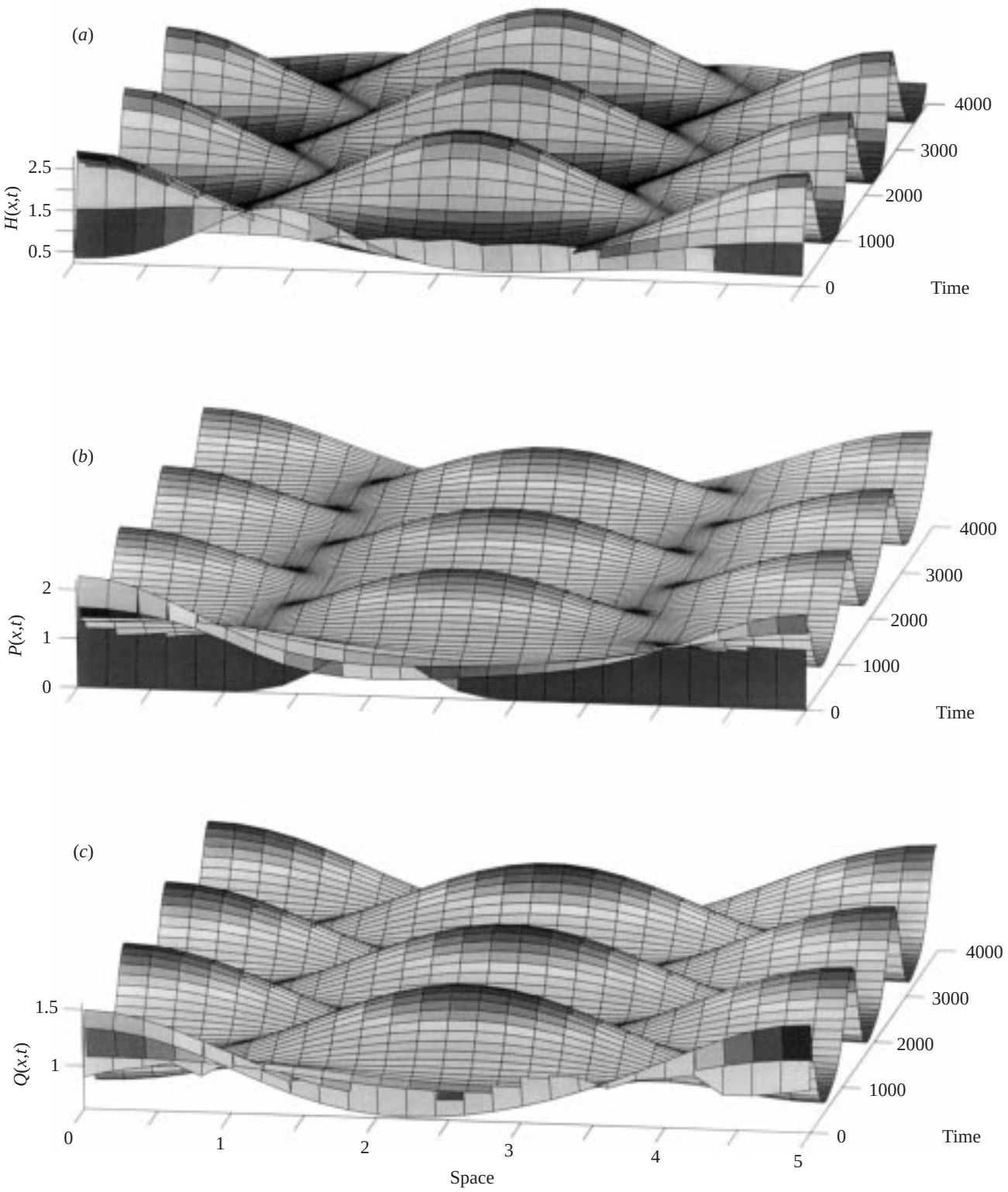

Figure 6. Model solution using the parameters given in figure 4. The initial condition for the host and hyperparasite was as for figure 5. The initial condition for the parasite was given by the expression $P(x,0) = \exp[-5(x-2)^2]$. The stable spatio-temporal patterning which is produced, matches, with translation, that shown in figure 5 to give an oscillatory spatial pattern, with a dimensionless spatial wavelength approximately equal to five units and dimensionless temporal wavelength of approximately 1200 units. (*a*) Host; (*b*) parasite; (*c*) hyperparasite.

We derived sufficient conditions for spatial patterning of a relatively simple system with restricted dynamics (18–20), without host movement. The model is characterized by a logistic function for host growth that in the limit includes exponential growth as a special case. There is a simple mass-action contact rate between the host and parasite, with a simple functional response for the contact rate between the parasite and hyperparasite. Non-dimensionalization assisted the analysis of the dynamics by reducing the numbers of parameters, although it is neither unique (other transformations are, of course, possible), nor is it essential to demonstrate spatial

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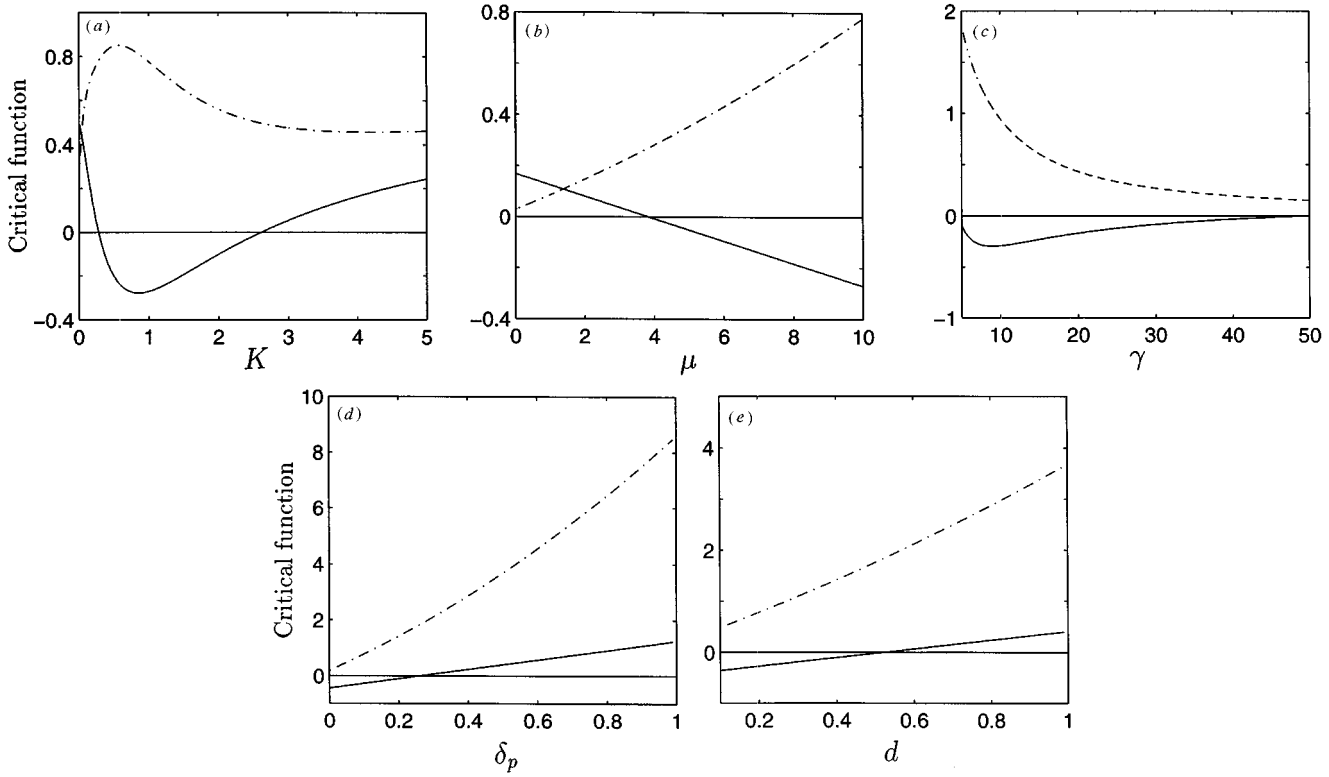

Figure 7. Evaluation of the inequalities (24*a–b*) to show how diffusion-driven instabilities depend on the various model parameters. In (*a*) $\tau = 5$, $\mu = 10$, $d = 0.2$, $\gamma = 12$, and $D_P = 0.1$; (*b*) $\tau = 5$, $K = 1$, $d = 0.2$, $\gamma = 12$, and $D_P = 0.1$; (*c*) $\tau = 5$, $K = 1$, $\mu = 10$, $d = 0.2$, and $D_P = 0.1$; (*d*) $\tau = 5$, $K = 1$, $\mu = 10$, $d = 0.2$, and $\gamma = 12$; and (*e*) $\tau = 5$, $K = 1$, $\mu = 10$, $\gamma = 12$, and $D_P = 0.1$. The solid line represents F_1 and the dashed line F_2. For diffusion-driven instability, $F_1 < 0$ and $F_2 > 0$.

patterning. However, the simpler the model, the easier it is to determine how the solution is affected by the parameters. Inspection of figure 7 shows how changing one parameter, while holding the others constant, can switch the behaviour from homogeneous to heterogeneous pattern, due to the occurrence of diffusion-driven instability. The model can easily be extended to consider alternative forms of functional response for the parasite–hyperparasite interaction (Gubbins & Gilligan 1996, 1997), and for the parasite–host interactions (Mimura & Murray 1978; Gilligan *et al*. 1997). We note, in particular, the intermediate generic models used by Mimura & Murray (1978), to generalize the forms of density dependence in intraspecific terms, that enabled them to elaborate the models introduced by Segel & Jackson (1972) for two-species interactions. We do not pursue this here, having presented a generic form that embraced inter- and intraspecific terms.

The dynamics of host growth affect the temporal behaviour of spatial patterning. Thus, when there is strong density dependence limiting host growth, the pattern is stable over time (figures 2 and 3), whereas with less stable underlying host growth (i.e. with negligible intraspecific density dependence), the pattern varies with time (figures 5 and 6). We also derived and tested a set of conditions on the parameters for the reduced model with no host movement (cf. figure 7). These criteria can be translated into testable biological hypotheses, for example, that μ, the dimensionless parameter for the maximum hyperparasite-induced parasite mortality, is greater than a certain critical value (cf. $\mu > 4$ in figure 7*b*). The case of a static host, with no diffusion, can be further simplified by uncoupling host dynamics so that the parasite and hyperparasite essentially interact in a homogeneous host density. Then the criteria for diffusion-driven instability revert to the two-species interaction between the parasite and hyperparasite.

The results given here for three-species interactions provide a basis for the detection, analysis and prediction of population-driven patterning due to diffusion-driven instability. They extend and qualify the work of Othmer & Scriven (1969) in an ecological context. Although the models are motivated specifically for the spread of soil-borne parasites of plants, they encompass a range of applied disciplines that involve microbial or invertebrate parasites and antagonists. The range includes medical epidemiology, wildlife management and bioremediation of soil pollution, as well as aerial epidemics of plants in both natural and agricultural communities. Both the generic models and the specific models are based on assumptions common to many reaction–diffusion models: that growth and movement can be separated into a reaction term and a simple diffusion term; that parameters are constant over time and space; and that there are zero flux boundary conditions, so that there is no external influence on the system. Of these assumptions, the temporal and spatial constancy of parameters is likely to attract most attention in ecological modelling. Periodic forcing has been widely used in temporal models of epidemic and ecological processes to simulate seasonal changes in birth, death and transmission parameters (Anderson & May 1981; London & Yorke 1973; Schwartz

1985). Sherratt (1995) has analysed the effects of temporally varying diffusion parameters in certain models, while Maini *et al*. (1992) and Shigesada *et al*. (1987) have made some progress in the analysis of spatially varying diffusion parameters. Future challenges lie in linking these approaches and in testing the models against experimental data, for which soil-borne microbial systems, amongst others (Gilligan 1994, 1995; Kleczkowski *et al*. 1996; Gubbins & Gilligan 1996, 1997) provide a rich source.

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