J. Math. Biol. 42, 95–119 (2001)
Digital Object Identifier (DOI):
10.1007/s002850000064

Mathematical Biology

Robert Stephen Cantrell[⋆†] · Chris Cosner[⋆†] · William F. Fagan[⋆⋆†]

Brucellosis, botflies, and brainworms: the impact of edge habitats on pathogen transmission and species extinction

Received: 10 June 1999 / Revised version: 7 July 2000 /
Published online: 20 December 2000 –

Abstract. Ecological interactions between species that prefer different habitat types but come into contact in edge regions at the interfaces between habitat types are modeled via reaction-diffusion systems. The primary sort of interaction described by the models is competition mediated by pathogen transmission. The models are somewhat novel because the spatial domains for the variables describing the population densities of the interacting species overlap but do not coincide. Conditions implying coexistence of the two species or the extinction of one species are derived. The conditions involve the principal eigenvalues of elliptic operators arising from linearizations of the model system around equilibria with only one species present. The conditions for persistence or extinction are made explicit in terms of the parameters of the system and the geometry of the underlying spatial domains via estimates of the principal eigenvalues. The implications of the models with respect to conservation and refuge design are discussed.

1. Introduction

Diseases, parasites, and other transmittable pathogens (hereafter "diseases") are key components of natural ecological systems, occurring in a wide variety of plant and animal communities (e.g., Anderson and May 1986, Roelke-Parker et al. 1996, Laurence et al. 1996). The dynamics of such diseases have long been of interest in mathematical biology (e.g., Kermack and McEndrick 1927), and recent epidemiological models that incorporate spatial perspectives (e.g., Holmes 1997) continue to provide new insights.

In conservation biology, scientists are particularly concerned with the influences of diseases on the persistence or extinction of threatened species. Particular

R.S. Cantrell, C. Cosner: Department of Mathematics and Computer Science, University of Miami, Coral Gables, FL 33124, USA. e-mail: rsc@math.miami.edu

W.F. Fagan: Department of Biology, Arizona State University, P.O. Box 871501, Tempe, AZ 85287-1501, USA. e-mail: bfagan@asu.edu

⋆Research partially supported by NSF grants DMS 96-25741 and DMS 99-73017.

⋆⋆Research partially supported by start up funds from Arizona State University.

†Order of authorship is alphabetical.

Key words: Reaction-diffusion – Lotka-Votterra – Habitat edges – Principal eigenvalues – Pathogen mediated competition – Persistence – Permanence – Extinction

emphasis is often directed at understanding the roles of "reservoir species," those species that, through their great abundance and/or immunity to a disease, facilitate persistence of the disease within a region of habitat. The role of reservoir species is often a critical one, because populations of rare species (which might otherwise be too small to permit establishment of diseases on their own) may face added extinction risks via infections of various sorts spread through contact with members of reservoir populations (e.g., Begon et al. 1992, Lyles and Dobson 1993). In many cases, domestic animals may serve as disease reservoirs, and their contacts with wild species may facilitate the spread of disease into nature reserves and other areas (Roelke-Parker et al. 1996, MacDonald 1996).

Through their impacts on interspecific disease transmission, habitat edges can strongly influence species interactions, including the transmission of pathogens (Fagan et al. 1999). For example, increased edginess of forests in the Northeast US and Canada has been implicated as a key factor influencing the transmission of brainworm infections from white-tailed deer (a common species in which the effects of infection are relatively benign), to other, less common, animals such as moose, woodland caribou, and elk, where the infections can be lethal (Anderson 1972, Holmes 1996). Remnant Puerto Rican rainforests feature a similar interaction wherein parasitic botflies are transmitted to forest interior species like the endangered Puerto Rican parrot through contacts with reservoir species at forest-matrix edges (Snyder et al. 1987, Loye and Carroll 1995). Habitat edges also play a role in the transmission of human diseases. For example, in New England, ticks capable of spreading Lyme disease are common in forest-lawn edges (Duffy et al. 1994) but have low survivorship in the lawn areas themselves, which feature more variable climatic conditions (Bertrand and Wilson 1996).

From a different perspective, the ongoing debate regarding the disease brucellosis, American bison, and cattle ranches outside Yellowstone National Park is also a case involving edge-mediated disease transmission (e.g., Meagher and Meyer 1994, Dobson and Meagher 1996, Wilkinson 1998). In that system, individuals working under the authority of the Montana Fish, Wildlife, and Parks department are permitted to kill bison dispersing outside the park boundaries because the bison population is known to harbor Brucella abortus, a bacterium that causes brucellosis and can cause ranch cows to abort developing fetuses. On the other hand, many environmentalists are concerned that continued killing of bison at the park boundary will endanger the Yellowstone population, especially when bad wheather and/or low resource availability promote high levels of bison emigration (Turbak 1995, Woodbury 1997, Wilkinson 1998). Epidemiological analyses by Dobson and Meagher (1996) suggest that the level of bison culling required to eliminate Brucella from the Yellowstone bison population could also cause extinction of the bison population.

The brucellosis, botfly, and brainworm examples all involve reservoir species that are mostly or at least partially segregated in space from populations of other host species. Despite their spatial segregation, such reservoir species can have negative (but indirect) effects on other host species through the transmission of pathogens. Consequently, the arrangement of species interactions involved in edge-mediated pathogen transmission is in many ways similar to those of other classes of indirect effects (in particular, apparent competition mediated by a mutual predator [e.g.,

Holt and Lawton 1993, 1994]), but feature a critical spatial complication. This is perhaps most clear in the case of the brainworm in which there is some evidence for a gradual replacement of susceptible cervid species by more resistant deer populations invading from the south (Anderson 1972; see also Schmitz and Nudds 1994).

A spatially explicit model of such a host-pathogen-reservoir system would be complicated. However, investigations become more tractable if we make some reasonable simplifying assumptions about the dynamics of the three-species system. Below, we present alternative formal derivations that allow us to recast a three-species disease model as a two species competition model, representing (indirectly mediated) competition between host and reservoir species. Clearly this simplification will not capture all of the intricacies of full system's dynamics, but it greatly facilitates study of edge-mediated pathogen transmission, which appears central to the empirical examples motivating our work. Specifically, we use a system of diffusive Lotka-Volterra competition equations to represent the spatial dynamics of two species that suffer from the same disease and have some fraction of their habitats in common.

2. Formulation of the models

We are interested in scenarios where two species are affected by the same pathogen but where one species acts as a reservoir for the pathogen and transmits it to the other species. Let us denote the species which acts as a reservoir for the pathogen as species 2 and the species to which the pathogen is transmitted as species 1. We shall assume that in species 2 the effects of the pathogen are relatively benign, whereas in species 1 the effects are more severe. In particular, we assume that infected individuals of species 1 do not transmit the pathogen back to species 2 or engage in ecological interactions to any significant degree. This would be the case if the pathogen were quickly lethal to species 1, or if species 1 were closely managed and infected individuals quarantined, among other possible scenarios. Hence, we shall be concerned with the infected members of species 1 only to the extent that their removal from the population influences the persistence of species 1. However, we must account for the infected members of species 2 because they represent the source of infection in species 1 and they may engage in ecological interactions. In addition to the assumptions stated above, we shall also assume that the timescale for transmission of the pathogen is fast relative to the timescale for population dynamics in the absence of the pathogen.

To obtain models which are sufficiently tractable as to allow the analysis of spatial effects we shall start with a nonspatial model for two competing populations which both are affected by a pathogen and show how either the nature of the questions we want to ask or the assumptions we want to make about our system allow us to reduce the models to simpler forms. Let S_i denote the density of individuals of species i which are uninfected by the pathogen. We shall assume that such individuals are susceptible to the pathogen. Let I_i denote the density of individuals of species i which are infected by the pathogen, and let $P_i = S_i + I_i$. A reasonably general but simple model which embodies some of the assumptions stated above is

$$\begin{aligned}
\frac{dS_1}{dt} &= [A_1 - B_{11}S_1 - B_{12}P_2]S_1 - C_{11}I_1S_1 - C_{12}I_2S_1 \\
\frac{dI_1}{dt} &= (C_1I_1 + C_2I_2)S_1 - M_1I_1 \\
\frac{dS_2}{dt} &= A_2S_2 + A_3I_2 - [B_{21}S_1 + B_{22}P_2]S_2 - C_{22}I_2S_2 \\
\frac{dI_2}{dt} &= C_{22}I_2S_2 - [B_{21}S_1 + B_{22}P_2]I_2 - M_2I_2
\end{aligned} \tag{2.1}$$

(See for example Anderson and May 1986, Begon et al. 1992.) In (2.1) all of the coefficients are assumed to be constants except possibly the coefficients C_{i2}, which may be constant or may have the form $C_{i2} = c_{i2}/P_2$ depending on detailed assumptions about the way the pathogen is transmitted among the population that harbors it. The term M_1I_1 in the second equation should be viewed simply as mortality. The term M_2I_2 in the fourth equation may include components of mortality and recovery. The term A_3I_2 in the third equation could reflect recovery from the pathogen or could reflect the birth of healthy offspring from infected individuals. The model assumes that infected adults do not produce infected offspring; that is, in epidemiological terminology, there is no vertical transmission of the pathogen. The system (2.1) can be viewed as a Lotka-Volterra competition model augmented with a standard type of epidemic model for the pathogen; see (Murray 1993) for discussions of population and epidemic models, (Kermack and McKendric 1927) for epidemic models, and (Anderson and May 1986) for both together.

2.1. First reduction: the viewpoint of persistence

We are primarily interested in predictions of persistence and extinction of populations, so we shall examine our models from the viewpoint of persistence theory, as discussed in (Hutson and Schmitt 1992, Cantrell et al. 1993a,b, 1996, Cosner 1994). Roughly speaking, the theoretical prediction of persistence for a system such as (2.1) is based on the invasibility of the system by populations at low densities. In other words, persistence theory provides a formal mathematical version of the idea that invasibility implies coexistence. In applying persistence theory to (2.1), the determination of persistence of species 1 would be based on the stability or instability with respect to invasion by species 1 of steady states where species 1 is not present. Notice that I_1 must be zero in any steady state of (2.1) where S_1 is zero, and that I_1 cannot increase when S_1 is small. (All the positive terms in dI_1/dt have S_1 as a factor.) Thus, at those steady states which are relevant to determining the persistence of species 1, which are precisely those with $S_1 = 0$, we also have $I_1 = 0$ and hence the presence or absence of an explicit I_1 term in the model is irrelevant. Another way to understand this point is to consider the simpler epidemic model which would occur if I_2 and S_2 were held fixed. The model for S_1 and I_1 would then take the form

$$\begin{aligned}
\frac{dS_1}{dt} &= (A - BS_1)S_1 - CS_1I_1 - DS_1 \\
\frac{dI_1}{dt} &= CS_1I_1 + DS_1 - M_1I_1
\end{aligned} \tag{2.2}$$

with A, B, C, D depending on the coefficients of (2.1) and the set values of S_2 and I_2. A phase plane analysis of (2.2) shows that the model predicts persistence of S_1 if and only if $A - D > 0$. The term $-CS_1I_1$ is irrelevant. (The reason for this is that I_1 is a state variable which becomes small if S_1 becomes small.) For a discussion of how to perform a phase plane analysis see (Murray 1993). The same conclusion about (2.2) also follows from formal persistence theory. See (Hutson and Schmitt 1992.) Because the terms involving I_1 do not affect the prediction of persistence of species 1 we will drop them from further consideration. (The presence or absence of the I_1 terms may affect the predicted equilibrium density of species 1 but not the prediction of persistence.)

2.2. Second reduction: biological hypotheses

If we consider only the terms of (2.1) that do not involve I_1 we get

$$\begin{aligned}\frac{dS_1}{dt} &= [A_1 - B_{11}S_1 - B_{12}P_2]S_1 - C_{12}I_2S_1\\ \frac{dS_2}{dt} &= A_2S_2 + A_3I_2 - [B_{21}S_1 + B_{22}P_2]S_2 - C_{22}I_2S_2\\ \frac{dI_2}{dt} &= C_{22}I_2S_2 - [B_{21}S_1 + B_{22}P_2]I_2 - M_2I_2.\end{aligned} \tag{2.3}$$

We shall always assume that the dynamics of the pathogen are fast relative to the rest of the system, so that we can use a pseudo-equilibrium hypothesis to assume that I_2 tracks S_2 and S_1. This makes sense in terms of the coefficients if C_{22} is large. The detailed form of the resulting system depends on the specific assumptions built into the model. We shall discuss two scenarios which lead to a Lotka-Volterra competition model. They differ in details but both embody the assumptions that the effects of the pathogen on species 2 are mild.

Scenario 1. In this scenario we assume that the pathogen has no ecological impact on species 2, and that the population of species 2 is homogeneously mixed, so that the chance of encountering an infected individual is proportional to the number of infected individuals. Then C_{22} is a constant, and because infected and uninfected individuals have the same vital rates,

$$\begin{aligned}A_2 &= (\text{birth rate of uninfected}) - (\text{death rate of uninfected})\\ &= (\text{birth rate of infected}) - (\text{death rate of infected})\\ &= A_3 - M_2.\end{aligned}$$

In that case we may add the last two equations in (2.3) to obtain for $P_2 = S_2 + I_2$ the equation

$$\frac{dP_2}{dt} = A_2P_2 - [B_{21}S_1 + B_{22}P_2]P_2 \tag{2.4}$$

The pseudo equilibrium I_2^* is found by setting $\dfrac{dI_2}{dt} = 0$, using $S_2 = P_2 - I_2$ in the equilibrium equation

$$C_{22}S_2 - B_{21}S_1 + B_{22}P_2 - M_2 = 0$$

and solving for I_2 to get

$$I_2^* = [1 - (B_{22}/C_{22})]P_2 - (B_{21}/C_{22})S_1.$$

We are assuming that C_{22} is large so that the coefficient of P_2 is positive. Substituting into the equation for S_1 yields

$$\begin{aligned}\frac{dS_1}{dt} &= A_1S_1 - [B_{11}S_1 + B_{12}P_2]S_1 - C_{11}([1 - (B_{22}/C_{22})]P_2 - (B_{21}/C_{22})S_1)S_1 \\ &= [A_1 - [B_{11} - (C_{11}B_{21}/C_{22})]S_1 - [B_{12} + C_{11}(1 - (B_{22}/C_{22}))]P_2]S_1. \end{aligned} \tag{2.5}$$

Together the equations (2.4) and (2.5) give a Lotka-Volterra competition system for S_1 and P_2 provided C_{22} is large and $C_{11}B_{21}/C_{22}$ is small. One way that $C_{11}B_{12}/C_{22}$ could be small is if B_{12} is small, which would mean the competitive impact of species 1 on species 2 is small. In that case the main contribution involving C_{11} to equation (2.5) is the term $-C_{11}P_2S_1$.

Scenario 2. In this scenario we assume that infected individuals of species 2 do not give birth while infected but may recover from the infection. We also assume that any individual in species 2 will contact only some fixed number of other individuals per unit time, whatever the population may be, so that the contact rate between susceptible and infected individuals now depends on the fraction of the population which is infected rather than the total number of infected individuals. Hence, in this scenario, $C_{22} = c_{22}/P_2$ and we have $A_2 = R$ = recovery rate of infected individuals, $A_2 = a_2 - d_2$ where a_2 = birth rate of uninfected individuals of species 2; d_2 = death rate of infected or uninfected individuals of species 2 so $M_2 = R + d_2$. Thus, we have

$$\begin{aligned}\frac{dS_2}{dt} &= (a_2 - d_2)S_2 + RI_2 - [B_{11}S_1 + B_{22}P_2]S_2 - \frac{c_{22}I_2S_2}{P_2} \\ \frac{dI_2}{dt} &= \frac{c_{22}I_2S_2}{P_2} - [B_{11}S_1 + B_{22}P_2]I_2 - RI_2 - d_2I_2.\end{aligned}$$

Adding these equations yields

$$\frac{dP_2}{dt} = a_2S_2 - d_2P_2 - [B_{11}S_1 + B_{22}P_2]P_2 \tag{2.6}$$

In this scenario the pseudo-equilibrium is $I_2^* = (P_2/c_{22})[c_{22} - R - d_2 - B_{21}S_1 - B_{22}P_2]$. Using $S_2 = P_2 - I_2^*$ gives $S_2 = (P/c_{22})[R + d_2 + B_{21}S_1 + B_{22}P_2]$. Using that form in (2.6) yields

$$\frac{dP_2}{dt} = \left[\left(\frac{a_2}{c_{22}} - 1\right)d_2 + \frac{R}{c_{22}}\right]P_2 - \left(1 - \frac{a_2}{c_{22}}\right)[B_{21}S_1 + B_{22}P_2]P_2. \tag{2.7}$$

This will make sense as part of a Lotka-Volterra system for P_2 and S_1 provided a_2/c_{22} is small and R/c_{22} is large relative to d_2. Again, these assumptions are consistent with the hypotheses that the dynamics of the pathogen are rapid and its

effects on species 2 are mild. If we assume the same sort of contact function for species 1 as for species 2, the equation for S_1 has $C_{11} = c_{11}/P_2$ so that we have

$$\begin{aligned}\frac{dS_1}{dt} &= [A_1 - c_{11} + (c_{11}R/c_{22}) + d_2(c_{11}/c_{22})]S_1 \\ &\quad -([B_{11} - (c_{11}/c_{22})B_{21}]S_1 + [(B_{12} - (c_{11}/c_{22})B_{22})]P_2)S_1. \quad (2.8)\end{aligned}$$

In the cases that interest us c_{11} should be relatively large. For (2.8) to make sense that requires some other assumptions, for example that R is large, c_{11}/c_{22} is close to 1, B_{12} is relatively large and B_{21} is relatively small. These are still consistent with our general assumptions about the systems we are modeling.

2.3. Remarks on hypotheses

We can reasonably reduce the system (2.1) to a Lotka-Volterra competition system for S_1 and P_2, at least for purposes of studying the persistence of S_1, under the detailed hypotheses of the scenarios described above. Related but different hypotheses might lead to competition models with a slightly different structure. The key assumptions appear to be:

1. the effects of the pathogen on species 2 are mild;
2. the rates of transmission and (if recovery is possible) recovery from infection are large relative to the ecological birth and death rates and competition coefficients in the system; and
3. in the absence of the pathogen the competitive impact of species 1 on species 2 is relatively weak.

Further discussion of models for species interactions where transmission of a pathogen between species is a factor are given in (Begon et al. 1992, Lyles and Dobson 1993).

2.4. Scaling and spatial modeling

For our treatment of spatial effects we begin with a Lotka-Volterra competition system of the sort which appears in equations (2.4), (2.5) or (2.7), (2.8). We rescale the variables representing the densities of uninfected individuals of species 1 and all individuals of species 2 so that their carrying capacities are equal to 1, so that in regions where both species are present the rescaled variables s_1 and p_2 satisfy the Lotka-Volterra competition model

$$\begin{aligned}\frac{ds_1}{dt} &= (r_1 - s_1 - \alpha_{12}p_2)s_1 \\ \frac{dp_2}{dt} &= (r_2 - p_2 - \alpha_{21}s_1)p_2.\end{aligned} \quad (2.9)$$

In (2.9) the competition terms account for both ordinary competition and the effects of pathogen transmission. The interactions are quantified by the coefficients α_{ij}.

We now turn to the spatial aspects of the model. We assume that there are two bounded open planar habitat regions, Ω_1 and Ω_2, with species i inhabiting only

Ω_i, but that the intersection $\Omega_E = \Omega_1 \cap \Omega_2$ represents an edge region or zone of overlap where both species may be present. (Note that this formulation includes many different possible geometric configurations for Ω_1 and Ω_2, ranging from the case where Ω_1 and Ω_2 have only a small fraction of their edge regions in common to the case in which one of the habitat patches is completely surrounded by the other.) To model dispersal we use simple diffusion and denote the diffusion rate of the ith species by D_i. We allow the usual types of boundary conditions, where some fraction (perhaps all or none) of the individuals that encounter the boundary of their domain die and the remainder return to the domain.

Let $u_i(x,t)$ be the density of species i on Ω_i. Because the species interact only in the overlap region Ω_E and because u_i is not defined on Ω_j $(j \neq i)$ except in Ω_E, we need to introduce an expression that is equal to u_i on Ω_E and equal to zero on $\Omega_j \backslash \Omega_E$ into the equation for u_j to describe the interaction. To that end, we define

$$U_i(u_i(x,t)) = \begin{cases} u_i(x,t) & \text{on} \quad \Omega_E \\ 0 & \text{on} \quad \Omega_j \backslash \Omega_E,\ j \neq i. \end{cases}$$

We can now formulate the model. We use the standard mathematical notation $\Delta = \partial^2/\partial x^2 + \partial^2/\partial y^2$ for the Laplace operator. We denote normal derivatives on $\partial\Omega_i$ by $\partial/\partial\eta$. The model is

$$\begin{aligned} \frac{\partial u_i}{\partial t} &= D_i \Delta u_i + (r_i - u_i - \alpha_{ij} U_j(u_j))u_i \quad \text{on } \Omega_i \times (0,\infty) \\ \beta_i(x)u_i + (1-\beta_i(x))\frac{\partial u_i}{\partial \eta} &= 0 \qquad \text{on } \partial\Omega_i \times (0,\infty), \qquad (2.10) \\ i &= 1,2, \quad j \neq i. \end{aligned}$$

The term $\beta_i(x)$ is assumed to take values in the interval $[0,1]$. It represents the fraction of individuals which die upon encountering the boundary. Thus, $\beta_i \equiv 1$ denotes a lethal boundary (Dirichlet condition) while $\beta_i \equiv 0$ denotes a reflecting boundary (Neumann or no-flux condition). We shall always assume that Ω_i is bounded for $i = 1, 2$.

In what follows we shall analyze how the size of the edge region Ω_E influences the predictions of the model (2.11). Note that the model (2.11) is symmetric with respect to i and j in its general form, so the characterization of the different roles played by the two species will depend on our choices of the parameter values.

3. Mathematical preliminaries

Our purpose in this section is two-fold. First, we construct a mathematical framework suitable for the analysis of the model we have formulated in Section 2. The elements of the framework are well-known from the theories of dynamical systems and partial differential equations. However, the assumption that the two species being modelled inhabit distinct but overlapping domains lends a certain novelty to the manner in which the elements of the framework are assembled. Once the framework is constructed, we gather particular known results regarding single-species reaction-diffusion equations and the spectra of elliptic operators that we will need to implement the analysis in Section 4 which to the best or our knowledge is original.

3.1. The model as a semidynamical system

We must verify that our model is well posed and that it can be viewed as a semidynamical system on an appropriate space, so that we can apply general abstract results on persistence. Our model can be treated via minor extensions or modifications of standard results on parabolic systems, specifically as discussed in (Henry 1981, Hutson and Schmitt 1992, Pazy 1983) so we shall only sketch the necessary arguments.

The essential steps in the analysis are to establish that the differential operators in (2.11) generate an analytic semigroup on an appropriate space, and then to verify that the nonlinear terms have the appropriate mapping properties on that space. Let A_i be the Laplace operator Δ on Ω_i. Choose p large enough that the Sobolev space $W^{2,p}(\Omega_i)$ embeds in the space $C^{1,v}(\overline{\Omega}_i)$ of functions with Hölder continuous first derivatives on Ω_i. Define the domain of A_i as $\mathrm{dom}A_i = \{u \in W^{2,p}(\Omega_i) : \beta_i(x)u + (1-\beta_i(x))\partial u/\partial\eta = 0 \text{ on } \partial\Omega_i\}$.

Lemma 3.1. *Assume that $\partial\Omega_i$ is smooth and that $\beta_i(x)$ is smooth on each connected component of $\partial\Omega_i$. Then A_i generates an analytic semigroup on $L^p(\Omega_i)$.*

Discussion. Since $\partial\Omega_i$ and β_i are smooth, it follows from standard elliptic *a priori* estimates (Agmon et al. 1959, Friedman 1976) that for all $u \in \mathrm{dom}A_i$ we have $\|u\|_{W^{2,p}(\Omega_i)} \le C(\|A_i u\|_{L^p(\Omega_i)} + \|u\|_{L^p(\Omega_i)})$ where C does not depend on u. $\mathrm{dom}A_i$ is dense in $L^p(\Omega_i)$. It then follows as in (Friedman 1976, Part 1, Section 18) that the operator $A_i - \lambda I$ satisfies inequalities implying that A_i generates an analytic semigroup as in (Friedman 1976, Part 2, Section 2).

Since A_i generates an analytic semigroup, we can define fractional powers A_i^α of A_i and use them to define norms on subspaces of $L^p(\Omega_i)$ by $\|u\|_\alpha = \|A_i^\alpha u\|_{L^p(\Omega_i)}$. (If we have pure Neumann boundary conditions, i.e. in the case where $\beta_i(x) \equiv 0$ on $\partial\Omega_i$, then A_i is not invertible so we must use $\|u\|_\alpha = \|A_i^\alpha u\|_{L^p(\Omega_i)} + \|u\|_{L^p(\Omega_i)}$). The subspaces are defined by $X_i^\alpha = \{u \in L^p(\Omega_i) : \|A_i^\alpha u\|_{L^p(\Omega_i)} < \infty\}$. If we take α sufficiently close to 1, then X_i^α embeds in $C^{1,v}(\overline{\Omega}_i)$. (See Henry 1981, Pazy 1983).

We can now define a space on which our model can be interpreted as a semidynamical system. Let $X = X_1^\alpha \times X_2^\alpha$ where $\alpha \in (0,1)$ is close enough to 1 that X_i^α embeds in $C^{1,v}(\overline{\Omega}_i)$. Define $A : X \to L^p(\Omega_1) \times L^p(\Omega_2)$ by $A : (u_1, u_2) \mapsto (A_1u_1, A_2u_2)$. Since A_i generates an analytic semigroup on $L^p(\Omega_i)$, A generates an analytic semigroup on $L^p(\Omega_1) \times L^p(\Omega_2)$ which can be represented by letting the semigroups generated by A_1 and A_2 act componentwise on (u_1, u_2). Define $U_i(u_i)$ on Ω_j, $j \ne i$, by

$$U_i = \begin{cases} u_i & \text{in} \quad \Omega_E \\ 0 & \text{on} \quad \Omega_j \backslash \Omega_E,\ j \ne i. \end{cases}$$

The map U_i defines a bounded linear operator from $L^q(\Omega_i)$ to $L^q(\Omega_j)$ for any $q \in [1,\infty]$. Define $F : X \to L^p(\Omega_1) \times L^p(\Omega_2)$ by $F : (u_1, u_2) \mapsto (u_1[r_1 - u_1 - \alpha_{12}U_2(u_2)], u_2[r_2 - u_2 - \alpha_{21}U_1(u_1)])$. (Since functions in X are also in

$C^{1,v}(\overline{\Omega}_1) \times C^{1,v}(\overline{\Omega}_2)$, the map F is well defined and smooth.) We can now write the model as

$$\frac{du}{dt} = Au + F(u) \tag{3.1}$$

which can be re-written as

$$u(t) = e^{tA}u(0) + \int_0^t e^{(t-s)A}F(u(s))ds. \tag{3.2}$$

Since A generates an analytic semigroup and F maps bounded sets in X into bounded sets in $L^p(\Omega_1) \times L^p(\Omega_2)$, we can apply the operator A^α to (3.2) as in (Henry 1981) and use the standard estimate $\|A^\alpha e^{tA}\| \leq Ct^{-\alpha}$ to conclude that if $u(0) \in X$ then $\|A^\alpha u\|_{L^p(\Omega_1)\times L^p(\Omega_2)}$ is bounded so that $u(t) \in X$. See (Henry 1981) for a detailed discussion. See also (Friedman 1976, Pazy 1983) for related discussion. It then follows as in (Henry 1981) that the model (2.11), interpreted as (3.2), generates a semiflow on X. To summarize, we have:

Theorem 3.2. *Under the hypotheses of Lemma 3.1, the model (2.11) can be given the abstract realizations (3.1) and (3.2). Under these realizations the model generates a semiflow on a space X which embeds in* $C^{1,v}(\overline{\Omega}_1) \times C^{1,v}(\overline{\Omega}_2)$.

Remarks. It follows as in (Friedman 1976, Henry 1981, Hutson and Schmitt 1992) that the smoothing properties of the semigroup imply that bounded orbits of (3.1) in X are precompact.

All of the terms in (2.11) are local, so the maximum principle and its various extensions apply just as in the case of a standard reaction-diffusion system. In particular, the system has the following properties:

Theorem 3.3. *The model (2.11) is order preserving with respect to the ordering where* $(u_1, u_2) \leq (v_1, v_2)$ *if and only if* $u_1 \leq v_1$ *and* $u_2 \geq v_2$. *The standard positive cone as defined by* $X^+ = \{(u_1, u_2) \in X : u_i > 0$ *on* Ω_i, $\partial u_i/\partial\eta < 0$ *on the parts of* $\partial\Omega_i$ *where* $\beta_i(x) = 1, i = 1, 2\}$ *is forward invariant. The set* $Y = \{(u_1, u_2) \in \overline{X}^+ : u_i \equiv 0$ *for some* $i\}$ *is also forward invariant. The semiflow maps* $\text{int}X^+ \cup Y$ *into itself for* $t > 0$. *The system is point dissipative in* $L^\infty(\Omega_1) \times L^\infty(\Omega_2)$*; dissipativity in X then follows via the smoothing properties of the semigroup in (3.2).*

Discussion. The order preserving properties of diffusive Lotka-Volterra models for two competing species, or for more general systems of two reaction-diffusion equations with competitive reaction terms, are well known (see Cosner and Lazer 1984, Hess 1991.) The invariance properties of the standard positive cone, the construction of a metric space upon which the abstract theory of permanence can be applied, and the dissipativity of the system, follow as in (Cantrell et. al. 1993a, Hutson and Schmitt 1992).

Theorems 3.2 and 3.3 provide the basis for the application of the abstract notions of persistence defined by permanence (Hutson and Schmitt 1992, Cantrell et al. 1993a) and compressivity (Hess 1991). However, to apply those notions we need to know more about the behavior of single species models which describe the semiflow in the boundary of the positive cone. To classify the behavior of those models and the full model we also need some background results on the principal eigenvalues of certain elliptic operators.

3.2. Eigenvalue problems and single species models

We now note the sorts of eigenvalue problems we must consider. Let $m(x)$ belong to $L^\infty(\Omega)$ for a bounded domain Ω; assume that the connected components of $\partial\Omega$ are smooth and that $\beta(x)$ is smooth on connected components of $\partial\Omega$ and takes values in $[0, 1]$. Note that $m(x)$ is allowed to change sign in Ω. We denote by $\sigma(\Omega, D, m, \beta)$ the principal eigenvalue for the problem

$$\begin{aligned} D\Delta\psi + m(x)\psi &= \sigma\psi \quad \text{in } \Omega \\ \beta\psi + (1-\beta)\frac{\partial\psi}{\partial\eta} &= 0 \quad \text{on } \partial\Omega; \end{aligned} \tag{3.3}$$

i.e., the unique real value of σ for which (3.3) admits an eigenfunction $\psi > 0$ in Ω. The existence of $\sigma(\Omega, D, m, \beta)$ in (3.3) follows from the classical variational formulation of eigenvalue problems as given, e.g., in (Courant and Hilbert 1953). In particular, $\sigma(\Omega, D, m, \beta)$ admits the following characterization: for $\beta \not\equiv 1$

$$\sigma(\Omega, D, m, \beta) = \sup_{u\in Z}\left(\frac{-D\int_\Omega |\nabla_u|^2 dx - D\oint_{\partial\Omega}\left(\frac{\beta}{1-\beta}\right)u^2 ds + \int_\Omega m(x)u^2 dx}{\int_\Omega u^2 dx}\right) \tag{3.4}$$

where Z is the completion in $W^{1,2}(\Omega)$ of the subspace $\{u \in C^1(\overline{\Omega}) : u(x) = 0 \text{ for } x \in \partial\Omega \text{ with } \beta(x) = 1\}$. In case $\beta \equiv 1$, the boundary integral no longer appears and $Z = W_0^{1,2}(\Omega)$. If $\beta(x) \le \beta_0 < 1$, then $Z = W^{1,2}(\Omega)$. Relation (3.4) implies that $\sigma(\Omega, D, m, \beta)$ is monotonically increasing with increasing $m(x)$.

Let us denote by $\lambda_1^+(\Omega, \beta)$ the principal positive eigenvalue for the problem

$$\begin{aligned} -\Delta\phi &= \lambda\phi \quad \text{in } \Omega \\ \beta\phi + (1-\beta)\frac{\partial\phi}{\partial\eta} &= 0 \quad \text{on } \partial\Omega; \end{aligned} \tag{3.5}$$

i.e. the unique necessarily *nonnegative* value of λ for which (3.5) admits an eigenfunction $\phi > 0$ in Ω. As its symbol indicates, the quantity $\lambda_1^+(\Omega, \beta)$ depends only on the geometry of Ω and the boundary condition determined by β. It is a very widely used quantity in mathematics and its applications. It is explicitly known for a range of domains and boundary conditions and readily approximated when it is not known explicitly. Moreover, it is easy to see that if m in (3.3) is a constant then $\sigma(\Omega, D, m, \beta)$ and $\lambda_1^+(\Omega, \beta)$ are related by the formula

$$\sigma(\Omega, D, m, \beta) = m - D\lambda_1^+(\Omega, \beta) \tag{3.6}$$

and that $\lambda_1^+(\Omega, \beta)$ is realized via the variational formula

$$\lambda_1^+(\Omega, \beta) = \inf_{u\in Z}\left(\frac{\int_\Omega |\nabla u|^2 dx + \oint_{\partial\Omega}\frac{\beta}{1-\beta}u^2 ds}{\int_\Omega u^2 dx}\right).$$

When m in (3.3) is no longer a constant, (3.6) no longer applies. However, there is sometimes an extension of $\lambda_1^+(\Omega, \beta)$ to weighted eigenvalue problems of the form

$$\begin{aligned} -\Delta\phi &= \lambda m(x)\phi \qquad \text{in } \Omega \\ \beta(x)\phi + (1-\beta(x)\phi &= 0 \qquad \text{on } \partial\Omega \end{aligned} \tag{3.7}$$

that is very useful in mathematical analysis and which sometimes occurs in the mathematical biology literature, e.g., (Cantrell and Cosner 1991). Indeed, the results of this article could be expressed in terms of this quantity, which we denote as $\lambda_1^+(\Omega, m, \beta)$. However, since the σ formulation is more readily interpreted biologically as an average growth rate over a domain, we will use only the σ formulation and the quantity $\lambda_1^+(\Omega, \beta)$ in this article.

To be specific, $\lambda_1^+(\Omega, m, \beta)$ denotes the principal *positive* eigenvalue of (3.7) when one exists. It is well-known (see, for example, (Manes and Micheletti 1973, Hess and Kato 1980, Brown and Lin 1980, Hess 1991)) that $\lambda_1^+(\Omega, m, \beta)$ exists and is monotonically decreasing as m increases when $m(x) > 0$ on an open subset of Ω and $\beta \not\equiv 0$. If $\beta \equiv 0$, the additional condition

$$\int_\Omega m(x)dx < 0 \tag{3.8}$$

is required to assert the existence of $\lambda_1^+(\Omega, m, \beta)$.

It is immediate that if $\beta \not\equiv 0$, $\lambda_1^+(\Omega, \beta) = \lambda_1^+(\Omega, 1, \beta)$ and that $\lambda_1^+(\Omega, 0) = 0$. While the quantities $\sigma(\Omega, D, m, \beta)$ and $\lambda_1^+(\Omega, m, \beta)$ can no longer be related by an explicit formula such as (3.6) when m is variable on Ω, it is nevertheless the case that a useful relationship between the two quantities does exist and may be stated as follows.

Lemma 3.4. *Assume $m \in L^\infty(\Omega)$ and that $m(x) > 0$ for x in an open subset of Ω. If $\beta \not\equiv 0$ or if $\beta \equiv 0$ and (3.9) holds, then $\sigma(\Omega, D, m, \beta) > 0$ if an only $\lambda_1^+(\Omega, m, \beta) < \dfrac{1}{D}$. If $\beta \equiv 0$ and the inequality in (3.9) is strictly reversed, then $\sigma(\Omega, D, m, \beta) > 0$.*

Discussion. The case $\beta \equiv 0$ is proved in (Senn 1983). The case $\beta \equiv 1$ is discussed briefly in (Cantrell and Cosner 1991); see also Proposition 2.2 in (Belgacem and Cosner 1995). Some related results are given in (Hess 1991).

Since the equation

$$\begin{aligned} D\Delta\psi + rm(x)\psi &= \sigma\psi \qquad \text{in } \Omega \\ \beta(x)\psi + (1-\beta(x))\frac{\partial\psi}{\partial\eta} &= 0 \qquad \text{on } \partial\Omega \end{aligned}$$

is evidently equivalent to

$$\begin{aligned} \frac{D}{r}\Delta\psi + m(x)\psi &= \frac{\sigma}{r}\psi \qquad \text{in } \Omega \\ \beta(x)\psi + (1-\beta(x))\frac{\partial\psi}{\partial\eta} &= 0 \qquad \text{on } \partial\Omega \end{aligned}$$

for $r > 0$, it follows from Lemma 3.4 that $\sigma(\Omega, D, rm, \beta) > 0 \Leftrightarrow \lambda_1^+(\Omega, m, \beta) < \frac{r}{D}$.

3.3. Diffusive logistic equations

We can now state the basic existence and uniqueness result for diffusive logistic equations. The sort of equations we will need to consider have the form

$$\begin{aligned} \frac{\partial u}{\partial t} &= D\Delta u + m(x)u - u^2 \quad \text{in} \quad \Omega \times (0, \infty) \\ \beta(x)u + (1 - \beta(x))\frac{\partial u}{\partial \eta} &= 0 \quad \text{on} \quad \partial\Omega \times (0, \infty) \end{aligned} \tag{3.9}$$

(Recall that any constant coefficient in the u^2 term may be eliminated by rescaling.)

Theorem 3.5. *Assume the hypotheses of Lemma 3.1 are satisfied and that $m(x) \in L^\infty(\Omega)$ with $m(x) > 0$ on some open subset of Ω. If $\sigma(\Omega, D, m, \beta) \leq 0$, all positive solutions to (3.9) approach zero as $t \to \infty$, while if $\sigma(\Omega, D, m, \beta) > 0$ then there is a unique positive equilibrium u^* of (3.9) which is globally attracting among positive solutions. If $m(x)$ is smooth on any open subdomain Ω_0 of Ω with $\overline{\Omega_0} \subseteq \Omega$, then as $D \to \infty$, $u^* \to m(x)$ uniformly on $\overline{\Omega_0}$.*

Mathematical discussion. This result is widely known, although it is usually formulated in different terms in the mathematical literature. Recall from Lemma 3.4 that $\sigma(\Omega, D, m, \beta) > 0$ is equivalent to $\lambda_1^+(\Omega, m, \beta) < 1/D$, so that the condition $\sigma(\Omega, D, m, \beta) \leq 0$ in the statement of the result is given by $\lambda_1^+(\Omega, m, \beta) \geq 1/D$ in the mathematical literature. Likewise, $\sigma(\Omega, D, m, \beta) > 0$ is replaced with $\lambda_1^+(\Omega, m, \beta) < 1/D$. The case $\beta \equiv 1$ is treated in (Cantrell and Cosner 1989) in detail. Related results are given in (Senn 1983) for $\beta \equiv 0$ and (Hess 1991) for general cases including periodic-parabolic logistic equations. A fairly detailed treatment of the case $\beta \equiv 0$ is given in (Cantrell et al. 1996). In cases where $\beta \not\equiv 0$ the analysis is essentially the same as in (Cantrell and Cosner 1989). The results on the behavior of u^* as $D \to 0$ is proved in the case $\beta \equiv 1$ in (Cantrell and Cosner 1989) but the analysis is local on $\overline{\Omega_0}$ so the boundary conditions on $\partial\Omega$ are irrelevant, since $\overline{\Omega_0} \subset \Omega$.

Lemma 3.6. *The equilibrium u^* of (3.9) depends continuously in the norm of $L^\infty(\Omega)$ on β and is monotonically increasing with respect to β.*

Proof. (Sketch) Suppose that $u^*(\beta_1)$ and $u^*(\beta_2)$ are equilibria of (3.9) for $\beta = \beta_1(x)$ and $\beta = \beta_2(x)$ respectively, and that on $\partial\Omega$ we have $\beta_1 > \beta_2$. It is easy to see that on $\partial\Omega$ we must have

$$\beta_2 u^*(\beta_1) + (1 - \beta_2)\frac{\partial u^*}{\partial \eta}(\beta_1) \leq 0$$

so that $u^*(\beta_1)$ is a subsolution of the equilibrium equation for (3.9) with $\beta = \beta_2$. Since any sufficiently large constant is a supersolution and since $u^*(\beta_2)$ is the

unique solution, we must have $u^*(\beta_2) \geq u^*(\beta_1)$. Suppose now that we fix $\beta(x)$ and choose any sequence $\{\beta_k(x)\}$ which converges uniformly and monotonically to $\beta(x)$. Suppose the sequence is increasing; the decreasing case is similar. We have that the sequence $\{u^*(\beta_k)\}$ is decreasing and is bounded below by $u^*(\beta)$. Thus, it must have a convergent subsequence. Elliptic regularity implies that a subsequence must converge to an equilibrium of (3.9); the boundary conditions converge to those of $u^*(\beta)$, so since $u^*(\beta)$ is the unique equilibrium with those boundary conditions we must have the subsequence converging to $u^*(\beta)$. The entire sequence must then converge to $u^*(\beta)$ by monotonicity. Since the original sequence is arbitrary, this establishes continuity of u^* with respect to β.

3.4. Persistence and possible extinction

We can now give criteria for the persistence of the system (2.11).

Theorem 3.7. *Suppose that the hypotheses of Lemma 3.1 are satisfied. Suppose further that for* $i = 1, 2$

$$\sigma(\Omega_i, D_i, r_i, \beta_i) > 0. \tag{3.10}$$

Let u_i^* *denote the unique positive equilibrium of*

$$\begin{aligned} &\frac{\partial u_i}{\partial t} = D_i \Delta u + [r_i - u_i]u_i \quad \text{in } \Omega_i \times (0, \infty) \\ &\beta_i u_i + (1 - \beta_i)\frac{\partial u_i}{\partial \eta} = 0 \qquad \text{on } \partial\Omega_i \times (0, \infty) \end{aligned} \tag{3.11}$$

$$\text{Let } U_i(u_i^*) = \begin{cases} u_i^* & \text{on } \Omega_E \\ 0 & \text{on } \Omega_j \backslash \Omega_E, \quad j \neq i. \end{cases}$$

The system (2.11) is permanent and compressive if the principal eigenvalues for the problems

$$\begin{aligned} &D_i \Delta \psi + [r_i - \alpha_{ij} U_j(u_j^*)]\psi = \sigma\psi \quad \text{in } \Omega_i \\ &\beta_i \psi + (1 - \beta_i)\frac{\partial \psi}{\partial \eta} = 0 \qquad \text{on } \partial\Omega_i, \end{aligned} \tag{3.12}$$

namely $\sigma(\Omega_i, D_i, r_i - \alpha_{ij} U_j(u_j^*), \beta_i)$, *are positive for* $i = 1, 2$. *If* $\sigma(\Omega_i, D_i, r_i - \alpha_{ij} U_j(u_j^*), \beta_i) \leq 0$ *for some* i, *then* $u_i \to 0$ *as* $t \to \infty$ *if* $u_j(x, 0) \geq u_j^*(x)$ *and* $u_i(x, 0)$ *is sufficiently small.*

Discussion. Theorem 3.7 is essentially a version of Theorem 5.7 of (Cantrell et al. 1993a) and the results in (Hess 1991, section IV.33) adapted to the system (2.11). An alternative approach to permanence is discussed in (Cantrell et al. 1993b); see also (Hutson and Schmitt 1992, Cosner 1994). The condition (3.10) is required so that each species can persist even if the other is absent. The condition that the principal eigenvalue is positive in (3.12) implies that the state where $u_j = u_j^*$ and

$u_i = 0$ is unstable, i.e. if u_i is small but positive for $t = 0$ then u_i will increase. Thus, the condition implies invasibility by u_i when u_j is present at equilibrium. In that sense Theorem 3.7 is a precise formulation of the idea that invasibility implies coexistence. Conversely, if $\sigma(\Omega_i, D_i, r_i - \alpha_{ij}U_j(u_j^*), \beta_i) \leq 0$, then the state $u_i = 0$, $u_j = u_j^*$ is locally stable, so if $u_j \geq u_j^*$ and u_i is small at $t = 0$ then $u_i \to 0$ as $t \to \infty$. (If $\overline{u}_i(x)$ is small enough that the trajectory starting at $u_i = \overline{u}_i(x)$, $u_j = u_j^*(x)$ approaches $u_i = 0$, $u_j = u_j^*$ as $t \to \infty$, then the same will be true for any trajectory with $u_i(x, 0) \leq \overline{u}_i(x)$ and $u_j(x, 0) \geq u_j^*(x)$ by the order preserving property of (2.11).)

4. Analysis

4.1. Conditions for permanence

The particular species interactions described in Sections 1 and 2 motivating the formulation of (2.11) have the feature that only one of the species (species 1) risks extinction due to its interaction with the other species in the overlapping portion Ω_E of the two habitats Ω_1 and Ω_2. In this section, we begin by capturing this feature via an additional assumption on the parameters in (2.11) which guarantees (via Theorem 3.7) that the model predicts persistence of species 2 for any choice of Ω_E and boundary condition on Ω_1. We then analyze how the intrinsic growth rates r_1 and r_2, diffusion coefficients D_1 and D_2, interaction coefficient α_{12}, overlapping region Ω_E, and boundary conditions on Ω_1 and Ω_2 affect the predictions of (2.11) regarding the persistence of species 1. Before proceeding, some observations are in order. First, under the assumption that species 2 survives in the absence of species 1, we shall see that the condition we place on the parameters of (2.11) to guarantee the persistence of species 2 independent of choice of Ω_E and boundary conditions on Ω_1 can be regarded as the requirement that the coefficient α_{21} measuring the negative effect of species 1 on species 2 be sufficiently small. Second, an examination of (3.11) and (3.12) in Theorem 3.7 reveals that α_{21} plays no role in determining the logistic equilibrium for species 2 in the absence of interactions between species, and consequently in deciding whether species 1 can invade Ω_1 when species 2 is at its logistic equilibrium in Ω_2. Consequently, the additional assumptions on α_{21} that guarantee that the model predicts persistence of species 2 independent of Ω_E and boundary condition on Ω_1 do not affect the generality of our analysis concerning the persistence of species 1.

We now derive a condition for persistence of species 2 independent of Ω_E and boundary condition on Ω_1. We have from (3.12) that the model predicts that species 2 persists if the principal eigenvalue of the problem

$$\begin{aligned} D_2\Delta\psi + [r_2 - \alpha_{21}U_1(u_1^*)]\psi &= \sigma\psi \quad \text{in } \Omega_2 \\ \beta_2\psi + (1-\beta_2)\frac{\partial\psi}{\partial\eta} &= 0 \quad \text{on } \partial\Omega_2 \end{aligned} \tag{4.1}$$

is positive, i.e. $\sigma(\Omega_2, D_2, r_2 - \alpha_2 U_1(u_1^*), \beta_2) > 0$. The maximum principle (Protter and Weinberger 1967) implies that $u_1^* \leq r_1$ and hence that $U_1(u_1^*) \leq r_1\chi_E \leq r_1$ on Ω_2. It follows that $\sigma \geq \rho$ where ρ is the principal eigenvalue of

$$\begin{aligned} D_2\Delta w + [r_2 - \alpha_{21}r_1]w &= \rho w \quad \text{in } \Omega_2 \\ \beta_2 w + (1-\beta_2)\frac{\partial w}{\partial \eta} &= 0 \qquad \text{on } \partial\Omega_2 \end{aligned} \tag{4.2}$$

and so $\sigma > 0$ whenever $\rho > 0$. But now by (3.6) $\rho = r_2 - \alpha_{21}r_1 - D_2\lambda_1^+(\Omega_2, \beta_2)$. We know from Lemma 3.6 that $u_1^* \to r_1$ as β_1 decreases to 0. Consequently, in order for the model to predict persistence of species 2 for any choice of boundary conditions on Ω_1, and any choice of Ω_E, we must require

$$r_2 > \alpha_{21}r_1 + D_2\lambda_1^+(\Omega_2, \beta_2). \tag{4.3}$$

With the assumption (4.3), Theorem (3.7) can be reduced to the following.

Corollary 4.1. *Suppose (3.10) and (4.3) hold. Then (2.11) is permanent if and only if the principal eigenvalue of the problem*

$$\begin{aligned} D_1\Delta\psi + [r_1 - \alpha_{12}U_2(u_2^*)]\psi &= \sigma\psi \quad \textit{in } \Omega_1 \\ \beta_1\psi + (1-\beta_1)\frac{\partial\psi}{\partial\eta} &= 0 \qquad \textit{on } \partial\Omega_1, \end{aligned} \tag{4.4}$$

is positive; i.e. $\sigma(\Omega_1, D_1, r_1 - \alpha_{12}U_2(u_2^*), \Omega_1) > 0$.

A condition sufficient for permanence in (2.11) may be obtained from Corollary 4.1 by replacing $U_2(u_2^*)$ in (4.4) with $M_2\chi_{\Omega_E}$ (where $M_2 = \sup\{u_2^*(x), x \in \Omega_E\}$) and requiring the positivity of the principal eigenvalue in the resulting boundary value problem. So doing overstates the impact of species 2 on species 1 in the model, in effect providing a "worst case scenario" for species 1.

As before, we know from the maximum principle that $M_2 \le r_2$. Let $m \in [M_2, r_2]$ and consider

$$\begin{aligned} D_1\Delta w + [r_1 - \alpha_{12}m\chi_{\Omega_E}]w &= \rho w \quad \text{in } \Omega_1 \\ \beta_1 w + (1-\beta_1)\frac{\partial w}{\partial\eta} &= 0 \qquad \text{on } \partial\Omega_1. \end{aligned} \tag{4.5}$$

If the principal eigenvalue ρ of (4.5) is positive, we have permanence in (2.11) by Corollary 4.1, as noted. We now examine (4.5) more closely, so as to interpret the condition for permanence (i.e. $\rho > 0$) in terms of inequalities relating its coefficients.

It follows directly from (3.4) that ρ satisfies

$$\rho = \sup_{u\in Z}\left(\frac{-D_1\int_{\Omega_1}|\nabla u|^2dx - D_1\oint_{\partial\Omega_1}\left(\frac{\beta_1}{1-\beta_1}\right)u^2ds + \int_{\Omega_1}(r_1 - \alpha_{12}mX_{\Omega_E})u^2dx}{\int_{\Omega_1}u^2dx}\right) \tag{4.6}$$

where the space of test functions Z and the interpretation of the boundary integral are as in the discussion following (3.4). It is immediate from (4.6) that

$$\rho > \sup_{u\in Z} \frac{\left(-D_1\int_{\Omega_1}|\nabla u|^2dx - D_1\oint_{\partial\Omega_1}\frac{\beta}{1-\beta_1}u^2ds\right)}{\int_{\Omega_1}u^2dx} + (r_1 - \alpha_{12}m)$$

$$= r_1 - D_1\lambda_1^+(\Omega_1,\beta_1) - \alpha_{12}m.$$

We now have the following result.

Corollary 4.2. *Suppose the conditions of Corollary 4.1 hold and let*

$$r_1 > D_1\lambda_1^+(\Omega_1,\beta_1) + \alpha_{12}m, \tag{4.7}$$

where $m \in [M_2, r_2]$, M_2 as before. Then (2.11) is permanent for any choice of overlapping region Ω_E.

A somewhat more explicit sufficient condition for permanence than (4.7) follows from (4.5) if $\beta_1(x) < 1$ on $\partial\Omega_1$. In such a case w in (4.5) does not vanish on $\overline{\Omega}$, and (4.5) can be written

$$D_1\frac{\Delta w}{w} + [r_1 - \alpha_{12}mX_{\Omega_E}] = \rho \tag{4.8}$$

in Ω_1. Now $D_1\dfrac{\Delta w}{w} = D_1\left[\operatorname{div}\left(\dfrac{\nabla w}{w}\right) + \dfrac{|\nabla w|^2}{w^2}\right]$ in Ω_1 and $\dfrac{1}{w}\dfrac{\partial w}{\partial\eta} = \dfrac{-\beta_1}{1-\beta_1}$ on $\partial\Omega_1$. Consequently, if we integrate (4.8) and apply the Divergence Theorem, we get that

$$\rho = r_1 + \frac{D_1}{|\Omega_1|}\left[\int_{\Omega_1}\frac{|\nabla w|^2}{w^2}dx - \int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds\right] - \alpha_{12}m\frac{|\Omega_E|}{|\Omega_1|}. \tag{4.9}$$

We now have the following result.

Theorem 4.3. *Suppose the conditions of Corollary 4.1 hold and that $\beta_1(x) < 1$ on Ω_1. Then (2.11) is permanent if*

$$r_1 > D_1\int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds/|\Omega_1| + \alpha_{12}m\frac{|\Omega_E|}{|\Omega|}. \tag{4.10}$$

Remarks. (i) If β_1 is constant, (4.10) simplifies to

$$\frac{r_1}{D_1} > \frac{\beta_1}{1-\beta_1}\frac{|\partial\Omega_1|}{|\Omega_1|} + \frac{\alpha_{12}m}{D_1}\frac{|\Omega_E|}{|\Omega_1|}, \tag{4.11}$$

delineating rather clearly relationships among the ratio of intrinsic rate of growth to rate of diffusion of species 1, boundary conditions on Ω_1, the geometry of Ω_1, impact of species 2 on species 1 and relative size of Ω_E within Ω_1 sufficient to guarantee permanence. Note in particular that when $\beta = 1/2$, the first term on the

right hand side of (4.11) becomes the perimeter to area (or surface area to volume) ratio.

(ii) The two sufficient conditions for permanence (4.7) and (4.10) may be viewed as complementary to each other. Formula (4.10) is certainly more explicit in showing relationships among system parameters sufficient to guarantee permanence. It also takes the size of the overlapping region Ω_E relative to the size of Ω_1 into account. If Ω_E is small relative to Ω_1, then $\alpha_{12}m\frac{|\Omega_E|}{|\Omega_1|}$ is much smaller than $\alpha_{12}m$. On the other hand, if we let v be the eigenfunction for the eigenvalue problem corresponding to $\lambda_1^+(\Omega_1, \beta_1)$ and compute as in (4.8), we obtain

$$\frac{\int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds}{|\Omega_1|} = \lambda_1^+(\Omega_1, \beta_1) + \frac{1}{|\Omega_1|}\int_\Omega \frac{|\nabla v|^2}{v^2},$$

so that $\frac{\int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds}{|\Omega_1|} > \lambda_1^+(\Omega_1, \beta_1)$ always. As $\beta_1 \to 0$, the corresponding eigenfunction v tends to a constant, so that $\lim\limits_{\beta_1\to 0}\frac{\int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds}{|\Omega_1|} = \lim_{\beta_1\to 0}\lambda_1^+(\Omega_1, \beta_1) = 0$. Consequently, the boundary condition/geometry of habitat terms in the two sufficient conditions for permanence are comparable for β_1 small. However, $\lim_{\beta_1\to 1}\frac{\int_{\partial\Omega_1}\frac{\beta_1}{1-\beta_1}ds}{|\Omega_1|} = +\infty$ while $\lim_{\beta_1\to 1}\lambda_1(\Omega_1, \beta_1) = \lambda_1(\Omega_1, 1)$, so that (4.7) is a far less restrictive criterion for permanence when β is near 1. Indeed, (4.7) has the advantage of being applicable for any admissible boundary condition on Ω_1.

(iii) When both habitats have reflecting boundaries, i.e., when both β_1 and β_2 equal 0, (4.11) simplifies in a manner worth noting. In this case, $u_2^* \equiv r_2$, so that $[M_2, r_2] = \{r_2\}$ and (4.11) becomes the requirement

$$r_1 > \alpha_{12}r_2\frac{|\Omega_E|}{|\Omega_1|}. \tag{4.12}$$

(iv) To apply the preceding results to real biological populations, researchers would have to quantify a variety of model parameters. These include 1) the population growth and dispersal rates of the focal species, 2) population densities and per capita impacts of the reservoir species, 3) the relative sizes of the edge habitat and principal habitat of the focal species, and 4) measures of the size and relative hostility of the boundary of the focal species' habitat. Though they are numerous, each of these parameters could be at least approximated through extensive field work. For example, the hostility parameter β_1 could be determined by quantifying the fraction of individuals of the focal species that permanently emigrate from its principal habitat. Consequently, we eventually hope to apply our results to real populations. For now, however, we explore a fictitious example.

Consider a series of rectangular blocks of habitat, each inhabited by the focal species. Furthermore, the outer portion of each habitat block constitutes edge habitat that is also inhabited by the reservoir species. The habitat blocks differ among themselves in both the hostility of their boundaries and in the relative sizes of

the edge habitat. Such an array of habitat blocks would be a plausible representation of many ecologically heterogeneous landscapes, such as a forested landspace subjected to clearcutting.

To ensure permanence within a given patch, the ratio of population growth to dispersal for the focal species must exceed the critical threshold given in Eq. 4.11. This threshold reflects that the focal species must contend with the dual negative effects of edge hostility and disease-mediated competition from the reservoir species to persist. Whether the focal species can do so successfully or not will contribute to its presence or absence from different patches.

Figure 1 demonstrates how the critical threshold ratio becomes more stringent for patches featuring increased boundary hostility or increased dominance by edge habitat. Note that increasing edge hostility has a nonlinear effect and that the importance of its effect is most pronounced in small patches (i.e., large perimeter to area ratio). In contrast, increasing either the relative extent of the edge habitat or the total impact of the reservoir species on the focal species (quantified via the term $\alpha_{12}^* m$) would have linear effects on the critical threshold in Eq. 4.11. Consequently, patches with a low density of the reservoir species spread over a large edge region could exhibit comparable disease-related effects as patches with a high density of the reservoir species concentrated within a small edge habitat. Interestingly, both disease-related factors would have relatively stronger influences in large patches, where the consequences of boundary hostility would be lessened (Fig. 1A).

4.2. Extinction results

If $\sigma < 0$ in Corollary 4.1, so that permanence fails for (2.11), it is not necessarily the case that any componentwise positive solution (u_1, u_2) of (2.11) has the property that $u_1 \to 0$ as $t \to \infty$. However, by imposing further restrictions on the parameters of (2.11), we may guarantee extinction of species 1.

Let (u_1, u_2) be a componentwise positive solution of (2.11). Then

$$\begin{aligned} &\frac{\partial u_1}{\partial t} \le D_1 \Delta u_1 + (r_1 - u_1)u_1 \quad \text{in } \Omega_1 \times (0,\infty) \\ &\beta_1 u_1 + (1-\beta_1)\frac{\partial u_1}{\partial \eta} = 0 \qquad \text{on } \partial\Omega_1 \times (0,\infty). \end{aligned} \tag{4.13}$$

It follows from (3.10), (4.13), the method of upper and lower solutions and Theorem 3.5 that for any $\varepsilon > 0$, there is a $t(\varepsilon) > 0$ so that if $t \ge t(\varepsilon)$ and $x \in \overline{\Omega}_1$, then $u_1(x,t) < r_1 + \varepsilon$. As a consequence, $u_2(x,t)$ is an upper solution to

$$\begin{aligned} &\frac{\partial u_2}{\partial t} = D_2 \Delta u_2 + (r_2 - \alpha_{21}(r_1+\varepsilon) - u_2)u_2 \quad \text{on } \Omega_2 \times (t(\varepsilon),\infty) \\ &\beta_2 u_2 + (1-\beta_2)\frac{\partial u_2}{\partial \eta} = 0 \qquad\qquad\qquad\qquad \text{on } \partial\Omega_2 \times (t(\varepsilon),\infty) \end{aligned} \tag{4.14}$$

By (4.3), $r_2 - \alpha_{21}(r_1+\varepsilon) > D_2\lambda_1^+(\Omega_2, \beta_2)$ for $\varepsilon > 0$ and sufficiently small. In such a case, $\delta\phi$ is a lower solution for $\delta > 0$ and sufficiently small, where $\phi > 0$

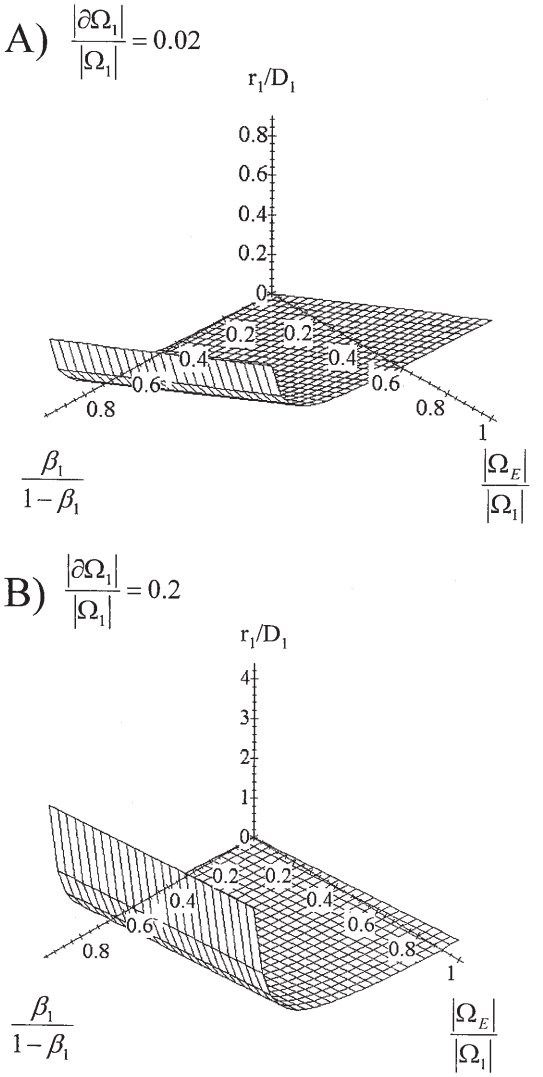

Fig. 1. Effects of edge habitat on species permanence in the face of a disease threat. Plotted surfaces are the critical values of r_1/D_1 (Eq. 4.11) that must be exceeded for the species to be permanent despite exposure to a pathogen in the edge region Ω_E in large habitat patches (A: small perimeter to area ratio) and small habitat patches (B: large perimeter to area ratio). Note that in large patches the critical value of r_1/D_1 is most sensitive to the relative size of Ω_E (except for patches with very hostile boundary conditions [i.e., high β_1]). whereas in small patches increasing hostility of the patch boundary quickly swamps the effects of edge-mediated disease transmission. For both panels, we set the term $\frac{\alpha_{12}m}{D_1} = 0.25$.

is an eigenfunction corresponding to $\lambda_1^+(\Omega_2, \beta_2)$. Theorem 3.5 and the method of upper and lower solutions imply that there is a $\bar{t}(\varepsilon) > t(\varepsilon)$ so that for $t \geq \bar{t}(\varepsilon)$ and $x \in \overline{\Omega}_2$,

$$u_2(x,t) \geq (1-\varepsilon)u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}(r_1+\varepsilon)) \ , \tag{4.15}$$

where $u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}(r_1+\varepsilon))$ denotes the unique globally attracting positive equilibrium to (4.14) (i.e., (3.9) with $\Omega = \Omega_2$, $D = D_2$, and $m = r_2 - \alpha_{21}(r_1+\varepsilon)$) whose existence is guaranteed by (4.3) and Theorem 3.5 for $\varepsilon > 0$ and sufficiently small.

Using (4.15), we have that u_1 is a lower solution to

$$\begin{aligned} \frac{\partial \overline{u}_1}{\partial t} &= D_1 \Delta \overline{u}_1 + [r_1 - \alpha_{12}U_2((1-\varepsilon)u_2^*(\Omega_2, D_2, r_2 \ - \alpha_{21}(r_1+\varepsilon))) - \overline{u}_1]\overline{u}_1 \\ &\qquad \text{in } \Omega_1 \times (\bar{t}(\varepsilon), \infty) \\ \beta_1 \overline{u}_1 + (1-\beta_1)\frac{\partial \overline{u}_1}{\partial \eta} &= 0 \qquad \text{on } \partial\Omega_1 \times (\bar{t}(\varepsilon), \infty) \ . \end{aligned} \tag{4.16}$$

Hence $u_1(x,t) \leq \tilde{u}_1(x,t)$, where $\tilde{u}_1(x,t)$ is the unique solution to (4.16) satisfying

$$u_1(x, \bar{t}(\varepsilon)) = \tilde{u}_1(x, \bar{t}(\varepsilon))$$

on $\overline{\Omega}_1$. If $\sigma(\Omega_1, D_1, r_1 - \alpha_{12}U_2(u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}r_1)), \beta_1) < 0$, then $\sigma(\Omega_1, D_1, r_1 - \alpha_{12}U_2((1-\varepsilon)u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}(r_1+\varepsilon))), \beta_1) < 0$ for $\varepsilon > 0$ and sufficiently small. (All the relevant quantities depend continuously on ε; see (Cantrell and Cosner 1993, 1996, 1998). In this case Theorem 3.5 implies that $\tilde{u}_1 \to 0$ as $t \to \infty$, and hence so does u_1.

We now have the following result.

Theorem 4.4. *Suppose that (3.10) and (4.3) hold. Then if*

$$\sigma(\Omega_1, D_1, r_1 - \alpha_{12}U_2(u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}r_1)), \beta_1) < 0 \tag{4.17}$$

any componentwise positive solution (u_1, u_2) to (2.11) has the property that $u_1 \to 0$ as $t \to \infty$.

Remark. Theorem 3.5 implies that $\lim_{D_2 \to 0} u_2^*(\Omega_2, D_2, r_2 - \alpha_{21}r_1) = r_2 - \alpha_{21}r_1$ uniformly on any open subdomain Ω of Ω_2 with the property that $\overline{\Omega} \subseteq \Omega_2$. Consequently, (4.17) will hold (and hence the model predicts the extinction of species 1) for D_2 small provided

$$\sigma(\Omega_1, D_1, r_1 - \alpha_{12}(r_2 - \alpha_{21}r_1)\chi_{\Omega_E}, \beta_1) < 0 \ . \tag{4.18}$$

Condition (4.18) is satisfied in turn provided $\dfrac{D_1\lambda_1^+(\Omega_1, \beta_1)}{r_1 - \alpha_{12}(r_2 - \alpha_{21}r_1)} = D_1\lambda_1^+(\Omega_1, r_1 - \alpha_{12}(r_1 - \alpha_{21}r_1), \beta_1) > 1$ and $|\Omega_1| - |\Omega_E|$ is small.

Remark. Related methods and results are discussed briefly in (Cantrell et al. 1993b) and in some detail and generality in (Cantrell and Cosner 1996).

4.3. Alternate scenario

We noted a secondary modeling scenario in Sections 1 and 2 which again employed (2.11). The local interactions between the two species, as measured by α_{12} and α_{21}, are the same as before, with $\alpha_{12} > \alpha_{21}$. In this model, however, species 2 is no longer assumed immune to an extinction threat, and we consider the efficacy of managing the impacts of species 2 on species 1 by controlling the population of species 2 in the overlapping region Ω_E.

We start with a situation in which β_2 is at or near 0 so that the boundary of species 2's region is almost completely reflecting. In this case, $u_2^* \approx r_2$ essentially independent of D_2. If r_1, D_1, α_{12} and β_1 are left alone, reducing $u_2^*|_{\Omega_E}$ is the only means available of reducing the impact of species 2 on species 1. We know from Lemma 3.8 that u_2^* is reduced if β_2 is increased. With this knowledge in mind, let's assume that β_2 is increased along $\partial\Omega_2 \cap \overline{\Omega}_E$ so that $\beta_2|_{\partial\Omega_2\cap\overline{\Omega}_E} \approx 1$ and that β_2 is left unchanged along the remainder of $\partial\Omega_2$, and then examine the ramifications of this assumption. Three basic outcomes emerge, depending upon a combination of the diffusivity of species 2 and the relative size of $\partial\Omega_2 \cap \overline{\Omega}_E$ compared to the whole of $\partial\Omega_2$.

The first basic possibility is that D_2 is small. In this case, there is a sharp boundary layer dropoff of u_2^* near $\partial\Omega_2 \cap \overline{\Omega}_E$, as noted in Theorem 3.7. However, as also noted in Theorem 3.7, u_2^* will still be approximately equal to r_2 in the vast majority of the interior of Ω_2, including Ω_E. In this scenario, the persistence of species 2 is not at risk, but the impact of species 2 on species 1 is not significantly reduced.

Alternatively, if the diffusion rate for species 2 is high, then u_2^* is reduced significantly in Ω_E, and the impact of species 2 on species 1 is also reduced. However, it is also important to assess whether the reduction imperils the survival of species 2 in Ω_2. If $\partial\Omega_2 \cap \overline{\Omega}_E$ is insignificant in size when compared to the whole of $\partial\Omega_2$, then u_2^* should remain near r_2 throughout much of Ω_2 and does not face an extinction threat. However, if $\partial\Omega_2 \cap \overline{\Omega}_E$ constitutes a large enough portion of $\partial\Omega_2$, then increasing β_2 toward 1 on $\partial\Omega_2 \cap \overline{\Omega}_E$ when D_2 is high reduces u_2^* significantly not just in Ω_E but throughout much of Ω_2. Indeed, in this case, if D_2 is large enough then $u_2^* = 0$ and hence species 2 is driven to extinction.

5. Conclusions

The model we have analyzed here provides a means of investigating the dynamics of a variety of ecological scenarios in which edge habitats help determine the persistence or extinction of interacting species. As outlined in the introduction, ecologists have studied at least two such scenarios in detail. The first or "remnant population" case is most applicable to situations in which threatened species in remnant patches of habitat face an added risk of extinction due to the transmission of diseases or other pathogens from reservoir species. Such reservoir species chiefly reside outside the remnant habitats but come in contact with the threatened species in edge regions. As we have outlined here, the persistence of the remnant species is dependent upon several factors with clear ecological interpretations. As is common to many partial differential equation formulations of population dynamics (Skel-

lam 1951, Kierstead and Slobodkin 1953, Okubo 1980, Cantrell and Cosner 1991) the ratio of a species' intrinsic rate of increase to its diffusion rate is once again a key factor in our model. In particular, this ratio must exceed a threshold value which varies among habitats, but is dependent upon 1) the lethality of the habitat boundaries, 2) the geometry of the habitat patch edge region in question (including, but not limited to, the habitat's perimeter-to-area ratio and size of the edge region relative to the species' entire habitat), and 3) the severity of the negative impacts the remnant species suffers because of its interactions with the reservoir species in the edge region.

The second major case investigated here was in many ways analogous to the brucellosis example outlined earlier. The brucellosis case features a key departure from the remnant population case: we eliminated our assumption that the disease-harboring species (e.g., bison in the brucellosis case) was immune to extinction and sought to investigate the extent to which increased mortality of that species in the edge region could lead to its extinction throughout its habitat. In this second analysis we found that if the dispersal rate of the disease-harboring species was small, then even the imposition of highly lethal boundary conditions in the edge region (e.g., shooting of dispersing bison) will have little impact on the reservoir species as a whole. In contrast, if the dispersal rate of the reservoir species was high, then the population might well be at risk of extinction due to edge-related mortality, but only if the boundary of the edge region (i.e., the lethal boundary) constituted a large enough fraction of the entire habitat boundary for that species.

Taken together these mathematical results suggest that in ecological systems where edge-mediated transmission of pathogens among species are suspected to play important roles (e.g. Anderson 1972, Brittingham and Temple 1983, Snyder et al. 1987, Dobson and Meagher 1996), investigations of species' reproductive rates, disease impacts, and, in particular, the frequency of cross-edge dispersal for a given patch geometry, would provide valuable data for assessing whether such factors need to be dealt with in detail by conservation managers.

References

1. Agmon, S., Douglis, A., Nirenberg, L.: Estimates near the boundary for solutions of elliptic partial differential equations satisfying general boundary conditions I. Communications in Pure and Applied Mathematics **12**, 623–727 (1959)
2. Anderson, R.C.: The ecological relationships of meningeal worm, (*Pneumostrongylus tenuis* Dougherty) and native cervids in North America. Journal of Wildlife Disease **7**, 304–310 (1972)
3. Anderson, R.M., May, R.M.: The invasion, persistence, and spread of diseases within animal and plant communities. Philosophical Transactions of the Royal Society of London, B. **314**, 493–496 (1986)
4. Begon, M., Bowers, R.G., Kadianakis, N., Hodgkinson, D.E.: Disease and community structure: the importance of host self-regulation in a host-pathogen model. American Naturalist. **139**, 1131–1150 (1992)
5. Belgacem, F., Cosner, C.: The effects of dispersal along environmental gradients on the dynamics of populations. Canadian Applied Mathematics Quarterly **3**, 379–397 (1995)

6. Bertrand, M.R., Wilson, M.L.: Microclimate-dependent survival of unfed adult *Ixodes scapularis* (Acari: Ixodidae) in nature: Life cycle and study design implications. Journal of Medical Entomology **33**, 619–627 (1996)
7. Brown, K.J., Lin, S.S.: On the existence of positive eigenfunctions for an eigenvalue problem with indefinite weight function, Journal of Mathematical Analysis and Applications **75**, 112–120 (1980)
8. Cantrell, R.S., Cosner, C.: Diffusive logistic equations with indefinite weights: population models in disrupted environments. Proceedings of the Royal Society of Edinburgh **112A**, 293–318 (1989)
9. Cantrell, R.S., Cosner, C.: Diffusive logistic equations with indefinite weights: population models in disrupted environments II. SIAM Journal on Mathematical Analysis **22**, 1043–1064 (1991)
10. Cantrell, R.S., Cosner, C.: Should a park be an island? SIAM Journal on Applied Mathematics **53**, 219–252 (1993)
11. Cantrell, R.S., Cosner, C.: Practical persistence in ecological models via comparison methods. Proceedings of the Royal Society of Edinburgh **126A**, 247–272 (1996)
12. Cantrell, R.S., Cosner, C.: On the effects of spatial heterogeneity on the persistence of interacting species. Journal of Mathematical Biology **37**, 103–145 (1998)
13. Cantrell, R.S., Cosner, C., Hutson, V.: Permanence in ecological systems with spatial heterogeneity. Proceedings of the Royal Society of Edinburgh **123A**, 533–559 (1993a)
14. Cantrell, R.S., Cosner, C., Hutson, V.: Permanence in some diffusive Lotka-Volterra models for three interacting species. Dynamic Systems and Applications **2**, 505–530 (1993b)
15. Cantrell, R.S., Cosner, C., Hutson, V.: Ecological models, permanence, and spatial heterogeneity. Rocky Mountain Journal of Mathematics **26**, 1–35 (1996)
16. Cosner, C.: Persistence (permanence), compressivity, and practical persistence in some reaction-diffusion models from ecology, in Comparison Methods and Stability Theory, eds. X. Liu and D. Siegel, Marcel Dekker, New York (1994)
17. Cosner, C., Lazer, A.C.: Stable coexistence states in the Volterra-Lotka competition model with diffusion. SIAM Journal on Applied Mathematics **44 (6)**, 1112–1132 (1984)
18. Courant, R., Hilbert, D.: Methods of Mathematical Physics v.1. Interscience, New York (1953)
19. Dobson, A., Meagher, M.: The population dynamics of brucellosis in Yellowstone National Park. Ecology **77**, 1026–1036 (1996)
20. Duffy, D.C., Clark, D.D., Campbell, S.R., Gurney, S., Perello, R., Simon, N.: Landscape patterns of abundance of *Ixodes scapularis* (Acari: Ixodidae) on Shelter Island, New York. Journal of Medical Entomology **31**, 875–879 (1994)
21. Fagan, W.F., Cantrell, R.S., Cosner, C.: How habitat edges change species interactions. American Naturalist **153**, 165–182 (1999)
22. Friedman, A.: Partial Differential Equations. Krieger, Huntington New York (1976)
23. Henry, D.: Geometric Theory of Semilinear Parabolic Equations, Lecture Notes in Mathematics **840**. Springer, Berlin (1981)
24. Hess, P.: Periodic-Parabolic Boundary Value Problems and Positivity, Pitman Reseach Notes in Mathematics #247. Longman Scientific and Technical, Harlow, Essex, United Kingdom (1991)
25. Hess, P., Kato, T.: On some linear and nonlinear eigenvalue problems with an indefinite weight function. Communications in Partial Differential Equations **5**, 999–1030 (1980)
26. Holmes, J.C.: Parasites as threats to biodiversity in shrinking ecosystems. Biodiversity and Conservation **5**, 975–983 (1996)

27. Holmes, E.E.: Basic epidemiological concepts in a spatial context. Pp. 111–136 In: Spatial Ecology: the role of space in population dynamics and interspecific interactions. D. Tilman and P. Kareiva, eds. Princeton University Press (1997)
28. Holt, R.D., Lawton, J.H.: Apparent competition and enemy-free space in insect host-parasitoid communities. American Naturalist **142**, 623–645 (1993)
29. Holt, R.D., Lawton, J.H.: The ecological consequences of shared natural enemies. Annual Review of Ecology and Systematics **25**, 495–520 (1994)
30. Hutson, V., Schmitt, K.: Permanence in dynamical systems. Mathematical Biosciences **111**, 1–71 (1992)
31. Kermack, W.O., McEndrick, A.G.: A contribution to the mathematical theory of epidemics. Proceedings Royal Society of London, **A. 115**, 700–721 (1927)
32. Kierstead, H., Slobodkin, L.B.: The size of water masses containing plankton bloom. Journal of Marine Resources **12**, 141–147 (1953)
33. Laurence, W.F., McDonald, K.R., Speare, R.: Epidemic disease and the catastrophic decline of Australian rain forest frogs. Conservation Biology **10**, 406–413 (1996)
34. Loye, J., Carroll, S.: Birds, bugs and blood: avian parasitism and conservation. Trends in Ecology and Evolution **10**, 232–235 (1995)
35. Lyles, A.M., Dobson, A.P.: Infectious disease and intensive management: population dynamics, threatened hosts, and their parasites. Journal of Zoo and Wildlife Medicine **24**, 315–326 (1993)
36. MacDonald, D.W.: Dangerous liaisons and disease. Nature **379**, 400–401 (1996)
37. Manes, A., Micheletti, A.-M.: Un'estensione della teoria variazionale classica degli autovalori per operatori ellittici del secondo ordine. Bollettino della Unione Matematica Italiana, Series 4, **7**, 285–301 (1973)
38. Meagher, M., Meyer, M.E.: On the origin of brucellosis in bison of Yellowstone National Park: A review. Conservation Biology **8**, 645–653 (1994)
39. Murray, J.D.: Mathematical Biology, Biomathematics **19**. Springer, Berlin (1989)
40. Okubo, A.: Diffusion and Ecological Problems: Mathematical Models, Biomathematics **10**. Springer, Berlin (1980)
41. Pazy, A.: Semigroups of Linear Operators and Applications to Partial Differential Equations. Springer, Berlin (1983)
42. Protter, M.H., Weinberger, H.F.: Maximum Principles in Differential Equations. Prentice Hall, Englewood Cliffs, New Jersey (1967)
43. Roelke-Parker, M.E., Munson, L., Packer, C., Kock, R., Cleaveland, S., Carpenter, M., O'Brien, S.J., Popischil, A., Hofmann-Lehmann, R., Lutz, H., Mwamengele, G.L.M., Mgasa, M.N., Machange, G.A., Summers, B.A., Appel, M.J.G.: A canine distemper virus epidemic in Serengeti lions (*Panthera leo*). Nature **379**, 441–445 (1996)
44. Senn, S.: On a nonlinear elliptic eigenvalue problem with Neumann boundary conditions with an application to population genetics. Communications in Partial Differential Equations **8**, 92–113 (1983)
45. Skellam, J.G.: Random dispersal in theoretical populations. Biometrika **38**, 196–218 (1951)
46. Schmitz, O.J., Nudds, T.D.: Parasite-mediated competition in deer and moose – how strong is the effect of meningeal worm on moose. Ecological Applications **4**, 91–103 (1994)
47. Snyder, N.F.R., Wiley, J.W., Kepler, C.B.: The parrots of Luquillo, Natural History and Conservation of the Puerto Rican parrot. Western Foundation of Vertebrate Zoology (1987)
48. Turbak, G.: Where the buffalo roam. Wildlife Conservation **98(6)**, 26–35 (1995)
49. Wilkinson, T.: No end to the slaughter. Audubon **100(2)**, 20 (1998)
50. Woodbury, R.: Arctic cats and buffalo. Time **149(11)**, 62–63 (1997)