On a population pathogen model incorporating species dispersal with temporal variation in dispersal rate

Rakhi Bhattacharyya · Banibrata Mukhopadhyay

Received: 12 December 2010 / Accepted: 27 March 2011 /
Published online: 2 May 2011
© Springer Science+Business Media B.V. 2011

Abstract In the present paper, we consider a mathematical model of ecosystem population interaction where the population suffers from a susceptible–infectious–susceptible disease. Dispersal of both the susceptible and the infective is incorporated using reaction–diffusion equations. We first study the stability criteria of the basic (non-spatial) model around the disease-free and the infected steady states. We find that the loss rate of the infective species controls disease prevalence. Also without predation pressure, the disease will continue to exist among the population. Then we analyze the spatial model with species dispersal in constant as well as in time-varying form. It is observed that though constant dispersal is unable to generate diffusion-driven instability, dispersal with sinusoidal variation in dispersion rate can generate diffusive instability when the wave number of the perturbation lies within a given range. Numerical simulations are performed to illustrate analytical studies.

Keywords Susceptible · Infected · Saturation incidence · Diffusive instability · Time-varying dispersal

1 Introduction

It is well established that the majority of ecological populations suffer from various infectious diseases. This has necessitated the study of epidemiological parameters in the ecological context. Anderson and May [1] pioneered this study by analyzing the role of disease in a predator–prey model. Subsequently, researchers formulated and studied various predator–prey models in the presence of pathogens [2–11].

R. Bhattacharyya
Department of Science, The Calcutta Technical School, 110, S.N. Banerjee Road,
Kolkata, 700 013, India

B. Mukhopadhyay (✉)
Department of Science, Central Calcutta Polytechnic, 21, Convent Road, Kolkata, 700 014, India
e-mail: banibrat001@yahoo.co.in

The primary issues that were addressed in the above-mentioned studies are species mortality, changes in reproductive rate and population size together with disease-induced oscillation of population states [12]. Anderson and May [1] investigated a predator–prey model with infected prey and observed infection-induced destabilization. An SI modification of the Rosenzweig predator–prey model was considered by Haderl and Freedman [2]. They obtained thresholds above which an infected equilibrium or an infected periodic solution exists. Han et al. [13] studied eco-epidemiological models with susceptible–infectious–susceptible and susceptible–infectious–removed diseases with standard and mass action incidence. Venturino investigated predator–prey models with infection in the prey [3] and the predator [4]. Haque and Chattopadhyay [14] studied the role of transmissible diseases in a prey-dependent predator–prey system with infected prey. Mukhopadhyay and Bhattacharyya [10] analyzed the impact of switching in a predator–prey model with prey infection.

In a real ecosystem, the individuals of a population do not mix homogeneously over the entire region available to them but instead develop within separate sub-regions. This necessitates extension of non-spatial models to spatial ones in order to allow individuals of either species to migrate between separate sites. This migration may be required for a variety of reasons; search for food, territorial extension for ever-increasing population size, widening the available gene pool, and minimizing the probability of extinction are to name a few [15]. Migration can occur in a local context such as the aquatic life in a pond; it can also be in the form of extensive migration patterns covering a large part of earth's surface, e.g., birds, humans, viruses, etc. Consequently, the emergence of spatial structure from an initially homogeneous state is one of the most exciting and widely studied problems in mathematical biology. Existing theoretical studies and field observations have suggested that spatial factors have a crucial role in controlling population evolution [16–19]. Ryabov and Blasius [17] reviewed a number of reaction–diffusion–advection equation models of varied complexity for the growth of plankton populations within a heterogeneous environment and discussed the criteria for population survival. Morozov and Arashkevich [16] performed a comparative analysis of different response functions on plankton community models with vertical heterogeneity and investigated their role on zooplankton patterns. Their study revealed that the sigmoidal response function is the most appropriate in this context. Interconnections between spatial heterogeneity and the paradox of enrichment in a phytoplankton–zooplankton system were studied by Poggiale et al. [18]. The recent book by Malchow and co-authors [20] is an excellent reference on pattern formation. The role of spatial interactions in models of disease-dominated ecological populations has also been studied [21–24]. These models are capable of describing more clearly the spatial transmission dynamics of the epidemic. In studying these spatially structured models, different techniques have been utilized to describe “space” [25]. Reaction–diffusion equations have been playing an important role in this context for a long time.

In studying morphogenesis, Turing [26] considered reaction–diffusion equations of two chemicals and found that diffusion could destabilize an otherwise stable equilibrium, which ultimately leads to non-uniform spatial patterns. This kind of instability is known as diffusion-driven instability or Turing instability [27, 28]. Segel and Jackson [29] first applied diffusion-driven instability in ecology. They presented an example of a predator–prey interaction where the inclusion of random dispersal results in an instability of the normal uniform steady-state distribution.

Initially, the study of the Turing mechanism was limited to reaction–diffusion equations with no explicit dependence on space and time. However, in a real ecosystem, parameters

may vary in space. Such variations have important implications on the resulting pattern [30–33]. It has also been shown that when the diffusion coefficients vary spatially, patterns appear that vary either in amplitude or in wavelength [34–38]. Apart from these spatial variations, diffusion rates may also vary in time. Such variations are usually in the form of either a slow seasonal variation or a rapid daily variation [37]. Timm and Okubo [39] analyzed the model of Levin and Segel [40] for the predator–prey interaction between different species of planktons where the dispersal rate of zooplankton varies sinusoidally in time. Using a specific set of parameter values, they observed numerically that the homogeneous steady state becomes more stable as the amplitude of temporal variation in dispersal rate increases with constant mean. They also demonstrated analytically this stabilizing effect in the case of small temporal variation. Sherratt [38] derived explicit conditions for diffusive instability when the predator dispersal rate oscillates with a long period between two constant values.

In the present paper, we formulate a bi-dimensional mathematical model of an ecosystem population which suffers from micro-parasite infection. We consider dispersal of both the susceptible and the infectives using a reaction–diffusion mechanism. In Section 3, we study the stability criteria of the non-spatial model and the model with constant dispersal rate. Section 4 analyzes the model with variable dispersal of the susceptible species in an attempt to compare the impact of constant and time-varying dispersal on disease-dominated ecological populations.

2 The model

We consider a mathematical model consisting of two dynamical equations—one for the susceptible population $S(t)$ and the other for the infected population $I(t)$. Only the susceptible is capable of reproducing. A logistic function is used to model the birth process with intrinsic growth rate r and environmental carrying capacity K . The infected population is removed by death or by predation before the possibility of reproducing. However, the infected population $I(t)$ contributes to resource consumption and hence competes with the sound population for their exploitation. A susceptible becomes infected when it comes in contact with infected individuals, and this contact process is assumed to follow saturation incidence kinetics with β measuring the force of infection and α_1 the inhibition effect. This incidence rate is more realistic than the bilinear one, as it includes the behavioral change and crowding effect of the infected individuals and also prevents unboundedness of the contact rate. The infected population suffer losses due to death and recovery at a rate $d_2 (= d_1 + \gamma)$. They join the susceptible class after recovery at a rate γ . The susceptible population also suffer losses due to natural death at a rate d_1 . Following the experimental observations of Lafferty and Morris [41], it is assumed that only the infected species that are weakened by the disease suffer biomass loss due to predation pressure.

In the proposed model, the infected population suffer biomass losses due to predation pressure (at a rate P). It is assumed that though the predators benefit from consumption of their prey, this food is only a portion of their diet and there is alternative food for the predators. Thus, it is reasonable to study the impact of predation on prey dynamics without considering directly the effects of prey on the population dynamics of the predator. Such a situation has been described in the context of plankton–fish dynamics in a lake ecosystem by Scheffer et al. [42]. In the lake ecosystem, since the plankton species are only a portion of the food for the fish population, the overall fish density depends on the productivity of the

lake and does not relate directly to plankton density. The term recording the loss of infected prey due to predation represents the impact of the predator community as a whole. In reality, different categories of predators forage on prey with different functional responses. Therefore, a realistic solution is to mimic the effect of different species of predator switching to prey at different moments with different efficiencies. As most of the predators usually switch to prey that are significantly abundant [43, 44], predation pressure is expected to increase more than linearly with prey density over the initial range. This switching behavior of the predator is modeled by using a sigmoidal function. The maximum consumption rate P is set directly rather than as a product of the predator biomass and their weight-specific maximum intake [42]. We also incorporate species dispersal into our model equations. The dispersal of species is taken to be random so that Fick's law holds. This gives the flux term as

$$\frac{\partial y_i}{\partial t} = D_i \frac{\partial^2 y_i}{\partial x^2}$$

where $0 \leq x \leq 1$ is the one-dimensional space coordinate and D_i are the constant dispersal coefficients. With these assumptions, we write down the model equations as

$$\begin{aligned} \frac{\partial S}{\partial t} &= rS \left[1 - \frac{S+I}{K} \right] - \frac{\beta SI}{c + \alpha_1 I} - d_1 S + \gamma I + D_1 \frac{\partial^2 S}{\partial x^2} \\ \frac{\partial I}{\partial t} &= \frac{\beta SI}{c + \alpha_1 I} - d_2 I - \frac{PI^2}{I^2 + h^2} + D_2 \frac{\partial^2 I}{\partial x^2} \end{aligned} \quad (2.1)$$

with non-negative initial conditions $S(0, x) \equiv S_0(x) \geq 0$; $I(0, x) \equiv I_0(x) \geq 0$ and homogeneous Neumann boundary conditions

$$\frac{\partial S(0, t)}{\partial t} = \frac{\partial S(1, t)}{\partial t} = \frac{\partial I(0, t)}{\partial t} = \frac{\partial I(1, t)}{\partial t} = 0. \quad (2.2)$$

In the next proposition, we state that the linear combination of the susceptible and the infected prey is less than a finite quantity, the proof of which is straightforward [45].

Proposition 2.1 *All solutions of (2.1) are uniformly bounded.*

3 Stability criteria

The steady states of system (2.1) with no diffusion are

1. The trivial state $E_0 \equiv (0, 0)$.
2. The infection free boundary state $E_1 \equiv (S_1, 0) = \left[\frac{K(r-d_1)}{r}, 0 \right]$.
3. The interior steady state $E^* \equiv (S^*, I^*)$ where

$$S^* = \left[d_2 + \frac{PI^*}{I^{*2} + h^2} \right] \left(\frac{c + \alpha_1 I^*}{\beta} \right) \quad (3.1)$$

and I^* is the positive root of

$$\left[d_2 + \frac{Px}{x^2 + h^2} \right] \left(\frac{c + \alpha_1 x}{\beta} \right) \times \left[r \left\{ 1 - \frac{d_2 + \frac{Px}{x^2 + h^2} + x}{K} \right\} - \frac{\beta x}{c + \alpha_1 x} - d_1 \right] + \gamma x = 0. \quad (3.2)$$

We first summarize the stability criteria of the homogeneous system corresponding to (2.1).

The model system is stable around the trivial state E_0 if $d_1 > r$. The infection-free boundary state E_1 exists if E_0 is unstable, and will exhibit stable behavior if

$$d_2 > \overline{d_2} \equiv \frac{\beta K(r - d_1)}{r}. \quad (3.3)$$

The endemic state E^* will be locally asymptotically stable for small γ when

$$-\frac{\beta S^* I^*}{(c + \alpha_1 I^*)^2} \alpha_1 - \frac{P I^* (h^2 - I^{*2})}{(I^{*2} + h^2)^2} < 0. \quad (3.4)$$

Stability analysis of the diffusive system (2.1), on the other hand, shows that it will remain stable when the corresponding homogeneous system is stable. Thus, diffusion-driven instability will not occur when the dispersal rates are assumed to be constant.

4 Time-varying dispersal

So far we have assumed the dispersal rates to be constants. However, in a real-world situation, dispersal rates can vary in time. A very good example can be derived from the context of oceanic diffusion. The vertical current shear in the ocean arising out of variable horizontal currents at different depths can generate horizontal dispersion due to interaction with a vertical mixing process, and consequently, the generated horizontal dispersion is most likely to vary with time. Bennett and Denmann [46] studied time-varying horizontal dispersion in zooplankton and inferred that the diurnal vertical migration of zooplankton, together with vertical current shear, is a major component in producing time-varying species dispersal. In this section, we investigate the spatial system where the diffusion coefficient of the susceptible population varies sinusoidally in time with small period of oscillation.

For this, we consider dimensionless time $\omega t = \tau$ where ω is the frequency of variation in D_1 . Here D_2 , namely the diffusion coefficient of the infected species, is assumed to be a constant, and D_1 a function of time τ . We express the solution of the linearized version of (2.1) in the form

$$s = \phi_1 e^{iqx}; \quad i = \phi_2 e^{iqx}. \quad (4.1)$$

Here ϕ_i are the amplitude of the solutions and q is the wave number of the solutions. The system of equations for ϕ_i are

$$\begin{aligned} \frac{d\phi_1}{d\tau} &= (a_{11} - q^2 D_1(\tau)) \frac{1}{\omega} \phi_1 + a_{12} \frac{1}{\omega} \phi_2, \\ \frac{d\phi_2}{d\tau} &= a_{21} \frac{1}{\omega} \phi_1 + \{a_{22} - q^2 D_2\} \frac{\phi_2}{\omega}. \end{aligned} \quad (4.2)$$

For analytical simplicity, we consider time-varying diffusivity as [39]

$$D_1(\tau) = D_2[\beta_1 + \alpha \sin \tau] \quad (4.3)$$

with $\beta_1 > 1$ and $\beta_1 > |\alpha|$. This condition implies that the diffusion rate for the susceptible population is more than that of the infectives.

Substituting the first equation of (4.2) into the second one, we obtain

$$\begin{aligned} \frac{d^2 \phi_2}{d\tau^2} - \frac{1}{\omega} [a_{11} + a_{22} - q^2(D_1(\tau) + D_2)] \frac{d\phi_2}{d\tau} \\ + [\{a_{22} - q^2 D_1(\tau)\}(a_{11} - q^2 D_2) - a_{12} a_{21}] \frac{1}{\omega^2} \phi_2 = 0. \end{aligned} \quad (4.4)$$

Next we transfer (4.4) into an equation without the first-order derivative [47]. For this we substitute

$$\phi_2 = u(\tau)v(\tau). \quad (4.5)$$

Substituting (4.5) into (4.4), we get

$$u''v + 2u'v' + v''u - q_1(\tau)(u'v + v'u) + q_2(\tau)uv = 0 \quad (4.6)$$

where ' denotes the derivative with respect to τ and

$$\begin{aligned} q_1(\tau) &= [a_{11} + a_{22} - q^2\{D_1(\tau) + D_2\}] \frac{1}{\omega}, \\ q_2(\tau) &= [a_{11}a_{22} - a_{12}a_{21} - q^2(D_1a_{22} + D_2a_{11}) + q^4D_1D_2] \frac{1}{\omega^2}. \end{aligned} \quad (4.7)$$

To make (4.6) free from first-order derivatives, we set

$$2u' - q_1u = 0 \quad (4.8)$$

which gives

$$u = \exp\left(\frac{1}{2} \int q_1(\tau) d\tau\right), \quad (4.9)$$

$$u'' = \left[\frac{1}{2}q_1' + \frac{1}{4}q_1^2\right]u. \quad (4.10)$$

Using (4.8) and (4.10), (4.6) reduces to

$$v'' + \left[\frac{1}{2}q_1' - \frac{1}{4}q_1^2 + q_2\right]v = 0. \quad (4.11)$$

Putting the values of q_1 and q_2 in (4.11), we get

$$\begin{aligned} v'' + \left[-\frac{1}{4\omega^2} (a_{22} + a_{11} - q^2 D_2)^2 + \frac{D_2 \beta_1}{2\omega^2} (a_{22} + a_{11} - q^2 D_2) - \frac{a^4 D_2^2}{4\omega^2} \beta_1^2 \right. \\ \left. + \frac{1}{\omega^2} \{ (a_{11} a_{22} - a_{12} a_{21} - q^2 D_2 a_{11}) - D_2 \beta_1 a_{22} q^2 + q^4 \beta_1 D_2^2 \} \right. \\ \left. + \sin \tau \left\{ \frac{\alpha D_2}{2\omega^2} (a_{11} + a_{22} - q^2 D_2) - \frac{q^4 D_2^2 \beta_1}{2\omega^2} \alpha - a_{22} \frac{q^2 D_2 \alpha}{\omega^2} + \alpha \frac{D_2^2 q^4}{\omega^2} \right\} \right. \\ \left. - \frac{q^4 D_2^2 \alpha^2}{4\omega^2} \sin^2 \tau - \frac{1}{2} \frac{q^2}{\omega} D_2 \alpha \cos \tau \right] v = 0. \end{aligned} \quad (4.12)$$

Now, since the amplitude of the variability in D_1 is small, αD_2 is also small. We denote $\eta = \frac{q^2 D_2 \alpha}{\omega}$ where η is a small parameter. Using η , we rewrite (4.12) as

$$\begin{aligned} v'' + \left[Q_1 + \sin \tau \left\{ (a_{22} + a_{12}) \frac{\eta}{2q^2 \omega} - \frac{\eta D_2}{2\omega} - \frac{\eta^2 \beta_1}{2\omega^2 \alpha} - \frac{a_{22}}{\omega} \eta + \frac{q^2 D_2 \eta}{\omega} \right\} \right. \\ \left. - \frac{1}{4} \eta^2 \sin^2 \tau - \frac{1}{2} \eta \cos \tau \right] v = 0 \end{aligned} \quad (4.13)$$

where

$$\begin{aligned} Q_1 = -\frac{1}{4\omega^2} (a_{11} + a_{22} - q^2 D_2)^2 + \frac{D_2 \beta_1}{2\omega^2} (a_{11} + a_{22} - q^2 D_2) \\ - \frac{q^4 D_2^2}{4\omega^2} \beta_1^2 + \frac{1}{\omega^2} \{ (a_{11} a_{22} - a_{12} a_{21} - q^2 a_{11} D_2) - D_2 \beta_1 a_{22} q^2 + q^4 \beta_1 D_2^2 \}. \end{aligned}$$

From (4.8) we have

$$\begin{aligned} u &= A \exp \left[\frac{\{ (a_{11} + a_{22}) - q^2 D_2 \} \tau}{2\omega} + \frac{\alpha q^2 D}{\omega} \cos \tau \right] \\ &= A \exp \left[\frac{(a_{11} + a_{22}) - q^2 D_2}{2\omega} \tau + \eta \cos \tau \right]. \end{aligned} \quad (4.14)$$

where A is a constant. Now, to find the analytical form of ϕ_1 , we have to find the solution of (4.13). In this direction, we consider

$$v = v_{10} + \eta v_{11} + \eta^2 v_{12} + \dots \quad (4.15)$$

Substituting (4.15) into (4.13) and equating like powers of η , we write

$$v''_{10} + Q_1 v_{10} = 0, \quad (4.16)$$

$$\begin{aligned} v''_{11} + Q_1 v_{11} + \left[\sin \tau \left\{ \frac{a_{11} + a_{22}}{2q^2 \omega} - \frac{D_2}{2\omega} - \frac{a_{22}}{\omega} + \frac{q^2 D_2}{\omega} \right\} - \frac{1}{2} \cos \tau \right] v_{10} = 0. \end{aligned} \quad (4.17)$$

We also have

$$\begin{aligned}
 Q_1 &= q^4 \left[\frac{\beta_1 D_2^2}{\omega^2} - \frac{\beta_1^2 D_2^2}{4\omega^2} - \frac{D_2^2}{4\omega^2} \right] \\
 &\quad + q^2 \left[\frac{D_2}{2\omega^2} (a_{11} + a_{22}) - \frac{D_2^2 \beta_1}{2\omega^2} - \frac{1}{\omega^2} (a_{22} \beta_1 D_2 + a_{11} D_2) \right] \\
 &\quad + \left[(a_{11} + a_{22}) \frac{\beta_1 D_2}{2\omega^2} - \frac{(a_{11} + a_{22})^2}{4\omega^2} + \frac{1}{\omega^2} (a_{11} a_{22} - a_{12} a_{21}) \right] \\
 &= M_1 q^4 + M_2 q^2 + M_3.
 \end{aligned} \tag{4.18}$$

Let

$$\bar{q}^2 = \frac{-M_2 \pm \sqrt{M_2^2 - 4M_1 M_3}}{2M_1}. \tag{4.19}$$

Now, $\bar{q}^2$ can never be positive if M_1, M_2, M_3 are all negative, whereas $\bar{q}^2$ has two distinct values if M_1, M_3 are negative and M_2 is positive, and in this case at least one of the values of $\bar{q}^2$ is positive.

Let us suppose that there exists at least one positive $\bar{q}^2$, say, $\bar{q}_{\max}^2$ and we denote the other value by $\bar{q}_{\min}^2$. Thus for,

$$\max \{0, \bar{q}_{\min}^2\} \leq q^2 \leq \bar{q}_{\max}^2 \tag{4.20}$$

Q_1 will be negative say, $Q_1 = -\delta^2$. In that case, from (4.16) we obtain

$$v_{10} = A_1 \exp(\delta\tau) + A_2 \exp(-\delta\tau), \tag{4.21}$$

$$\begin{aligned}
 \phi &= A \exp \left\{ \frac{a_{11} + a_{22} - q^2 D_2}{2\omega} \tau + \eta \cos \tau \right\} \\
 &\quad \times [A_1 \exp(\delta\tau) + A_2 \exp(-\delta\tau) + \eta v_{11} + \dots],
 \end{aligned} \tag{4.22}$$

where A, A_1, A_2 are integration constants.

Though u is bounded as $\tau \rightarrow \infty$, ϕ_1 is unbounded as v_{10} is unbounded. And hence a system which is stable under constant dispersal may become unstable with time-varying dispersal coefficient.

Next we take up the case $q^2 > \bar{q}_{\max}^2$. In this case $Q_1 = \delta^2$ and from (4.16) we can write

$$v_{10} = A_1 \cos \delta\tau + A_2 \sin \delta\tau. \tag{4.23}$$

The corresponding ϕ_1 will be

$$\phi_1 = A \exp \left[\frac{a_{11} + a_{22} - q^2 D_2}{2\omega} \tau + \eta \cos \tau \right] [A_1 \cos \delta\tau + A_2 \sin \delta\tau + \eta v_{11} + \dots]. \tag{4.24}$$

Now to solve for v_{11} , we have from (4.17)

$$v_{11} = \frac{1}{2(D^2 + \delta^2)} \left[\left(A_1 C_1 - \frac{A_2}{2} \right) \sin(1 + \delta)\tau - \left(A_2 C_1 + \frac{A_1}{2} \right) \cos(1 + \delta)\tau \right. \\ \left. + \left(A_1 C_1 + \frac{A_2}{2} \right) \sin(1 - \delta)\tau + \left(A_2 C_1 - \frac{A_1}{2} \right) \cos(1 - \delta)\tau \right] \quad (4.25)$$

where

$$C_1 = \frac{a_{11} + a_{22}}{2q^2\omega} + \frac{2q^2 D_2 - 2a_{11} - D_2}{2\omega}.$$

In a more compact form,

$$v_{11} = \frac{1}{2} \left[\frac{E_1}{-1 - 2\delta} \sin(1 + \delta)\tau + \frac{E_2}{1 + 2\delta} \cos(1 + \delta)\tau \right. \\ \left. + \frac{E_3}{2\delta - 1} \sin(1 - \delta)\tau + \frac{E_4}{2\delta - 1} \cos(1 - \delta)\tau \right] \quad (4.26)$$

where

$$E_1 = \left(A_1 C_1 - \frac{A_2}{2} \right), \quad E_2 = \left(A_2 C_1 + \frac{A_1}{2} \right), \\ E_3 = \left(A_1 C_1 + \frac{A_2}{2} \right), \quad E_4 = \left(A_2 C_1 - \frac{A_1}{2} \right). \quad (4.27)$$

So, v_{11} is bounded as $\tau \rightarrow \infty$. Hence, ϕ_1 is also bounded, considering η to be a small parameter and hence neglecting the terms $O(\eta^2)$.

Thus for large wave number $q^2 \geq \bar{q}_{\max}^2$, ϕ_1 is bounded. Since ϕ_1 is the measure of perturbation around the interior equilibrium point, the diffusive system will remain stable for a perturbation with large wave number.

5 Discussion

Dispersal is an essential part in the life history of most living organisms; it has a profound effect on population dynamics. In reality, dispersal of different populations does not take place in a uniform fashion; rather, it is influenced in a non-uniform way by various factors like seasonal and daily variations, environmental factors, etc. and also by various intrinsic factors which are inherently internal in nature [28].

In the present research, we have proposed and analyzed a population-pathogen model with dispersal. We first studied the basic non-spatial model. From our study of the disease-free equilibrium state, we found that the system will exhibit stable behavior around this state when the loss rate of infected prey exceeds a critical value $\bar{d}_2$. It is seen from the expression of $\bar{d}_2$ (in (3.3)) that when the infection propagation rate is low, the disease will die out

for lower loss rate of infected species (through death or recovery), and when the spread of infection is high, a larger loss rate of infected individuals is required for eradication of the disease. A numerical simulation study of the model system around the endemic state with parameter values satisfying (3.4) is shown in Fig. 1 exhibiting stable population coexistence. It may be noted here that we have used a saturation functional response instead of the more traditional bilinear one (namely that $\alpha_1 = 0$ in (2.1)) to model the infection process. To emphasize the significance of this saturation incidence over the bilinear one, we have simulated the model with bilinear incidence using the same set of parameter values around the corresponding endemic state (Fig. 2). It has been observed that there is a decrease in the population concentration of the susceptible individuals and an increase in that of the infected individuals when saturation incidence is replaced by the bilinear form. It is also interesting to observe that if the predation pressure on the infective individuals is omitted, the system will exhibit local asymptotic stability for small γ . So, without predation pressure, the disease will continue to exist among the population, whereas when predation pressure is taken into account, the disease will remain existent under certain parameter conditions given by (3.4).

Next we have investigated the reaction–diffusion system modeling prey dispersal. An analysis of the model with constant dispersal showed that the diffusive model remained stable under the parameter conditions for which the non-diffusive system is stable. Thus,

Fig. 1 Numerical simulation of the non-spatial model around the infected equilibrium point $E^* = (14.424, 14.916)$ with $d_2 > \bar{d}_2$. The parameter values are $r = 11.2$, $K = 15$, $\beta = 0.2$, $\alpha_1 = 2.5$, $d_1 = 0.4$, $d_2 = 0.413$, $\gamma = 0.0133$, and $P = 0.08$; $h = 0.5$. The system exhibits stable behavior

Fig. 2 Numerical simulation of the non-spatial model with bilinear incidence (that is $\alpha_1 = 0$) around the infected equilibrium with the same set of parameter values as in Fig. 1

the system is unable to exhibit diffusion-driven instability when the dispersal is assumed to be constant. Finally, we have analyzed the situation where dispersal is assumed to vary sinusoidally with time. From the ecological standpoint, this variation represents seasonal and/or daily variation in population dispersal due to vertical migration. It is worthwhile to

Fig. 3 Numerical simulation of the spatial model around the infected equilibrium point E^* with constant diffusivity. The parameter values are the same as those of Fig. 2 together with $D_1 = 0.0065$; $D_2 = 0.005$. The simulation is done for $0 \leq x \leq 1$ with zero flux boundary condition. The figure exhibits stable pattern of susceptible prey

Fig. 4 Numerical simulation of the spatial model around the infected equilibrium point E^* with constant diffusivity. The parameter values are the same as those of Fig. 3. The figure exhibits a stable pattern of infected prey

note that we have made the dispersal rate of the susceptible population vary in time, whereas that of the infective population is kept constant. This is reasonable as the susceptible population that is more fit to contract the disease is more prone to migration than the infectives. The choice of the parameters α and β_1 also ensures that the susceptible disperse more than the infectives. Using perturbation technique, we have shown that when the wave number of the perturbation lies within a given range (deduced in (4.20)), the system, which remained stable in case of constant diffusivity, may exhibit diffusion-induced instability under time-varying dispersal. On the other hand, when this wave number is greater than a threshold value, variation in dispersal is unable to destabilize the system. Thus, our analysis of time-varying diffusivity in the species-pathogen context revealed that time-varying dispersal may or may not generate diffusive instability, and moreover, it is the wave number of the perturbation that plays the key role in this context. A numerical simulation

Fig. 5 Numerical simulation of the spatial model around the infected equilibrium point with variable diffusivity of susceptible individuals. The parameter values are same as those of Fig. 4 together with $\beta_1 = 3$; $\alpha = 1.5$. The simulation is done for $0 \leq x \leq 1$ with zero flux boundary condition. The pattern indicates diffusive instability for susceptible prey

Fig. 6 Numerical simulation of the spatial model around the infected equilibrium point with variable diffusivity of susceptible. The parameter values are same as those of Fig. 5. The pattern indicates diffusive instability for infected prey

study of the reaction–diffusion model is performed around the infected equilibrium point with the same set of parameter values as those of the non-diffusive system. The simulation is done for $0 \leq x \leq 1$ with zero flux boundary condition. The resulting population patterns with constant diffusivity are shown in Figs. 3 and 4, and patterns with sinusoidally varying diffusivity of the susceptible prey are demonstrated in Figs. 5 and 6. Finally, we have numerically experimented with the situation where dispersal of both the susceptible and the infective populations varies sinusoidally with time. We observed occurrence of diffusion-driven instability in this case also. The resulting population patterns are shown in Figs. 7 and 8.

Fig. 7 Numerical simulation of the spatial model around the infected equilibrium point with variable diffusivity of both species. The pattern indicates diffusive instability for the susceptible prey

Fig. 8 Numerical simulation of the spatial model around the infected equilibrium point with variable diffusivity of both species. The pattern indicates diffusive instability for infected prey

Acknowledgements The authors are grateful to the reviewers and the Editor for their valuable comments and suggestions. Financial support from the Department of Science and Technology, Government of India (Grant No. SR/S4/MS:296/05) is also acknowledged.

References

1. Anderson, R.M., May, R.M.: The invasion, persistence and spread of infectious diseases within animal and plant communities. *Philos. Trans. R. Soc. Lond. B* **314**, 533–570 (1986)
2. Haderl, K.P., Freedman, H.I.: Predator–prey populations with parasite infection. *J. Math. Biol.* **27**, 609–631 (1989)
3. Venturino, E.: Epidemics in predator–prey models: diseases in the prey. In: Arino, O., Axelrod, D., Kimmel, M., Langlais, M. (eds.) *Mathematical Population Dynamics: Analysis of Heterogeneity*, vol. 1: *Theory of Epidemics*, pp. 381–393. Wuerz, Winnipeg (1995)
4. Venturino, E.: Epidemics in predator–prey models: disease in the predator. *IMA J. Math. Appl. Med. Biol.* **19**, 185–205 (2002)
5. Chattopadhyay, J., Arino, O.: A predator–prey model with disease in the prey. *Nonlinear Anal.* **36**, 747–766 (1999)
6. Xiao, Y., Chen, L.: Modeling and analysis of a predator–prey model with disease in the prey. *Math. Biosci.* **171**, 59–82 (2001)
7. Hethcote, H.W., Wang, W., Han, L., Ma, Z.: A predator–prey model with infected prey. *Theor. Popul. Biol.* **66**, 259–268 (2004)
8. Hall, S.R., Duffy, M.A., Caceres, C.E.: Selective predation and productivity jointly drive complex behavior in host–parasite systems. *Am. Nat.* **165**(1), 70–81 (2005)
9. Mukhopadhyay, B., Bhattacharyya, R.: Dynamics of a delayed epidemiological model with nonlinear incidence: the role of infected incidence fraction. *J. Biol. Syst.* **13**(4), 341–361 (2005)
10. Mukhopadhyay, B., Bhattacharyya, R.: Role of predator switching in an eco-epidemiological model with disease in the prey. *Ecol. Model.* **220**, 931–939 (2009)
11. Fenton, A., Rands, S.A.: The impact of parasite manipulation and predator foraging behavior on predator–prey communities. *Ecology* **87**(11), 2832–2841 (2006)
12. Mena-Lorca, J., Hethcote, H.W.: Dynamic models of infectious diseases as regulators of population sizes. *J. Math. Biol.* **30**, 693–716 (1992)
13. Han, L., Ma, Z., Hethcote, H.W.: Four predator prey models with infectious diseases. *Math. Comput. Model.* **34**, 849–858 (2001)

14. Haque, M., Chattopadhyay, J.: Role of transmissible disease in an infected prey-dependent predator–prey system. *Math. Comput. Model. Dyn. Syst.* **13**, 163–178 (2007)
15. Renshaw, E.: *Modelling Biological Populations in Space and Time*. Cambridge University Press, Cambridge (1995)
16. Morozov, A., Arashkevich, E.: Patterns of zooplankton functional response in communities with vertical heterogeneity: a model study. *Math. Model. Nat. Phenom.* **3**(3), 131–148 (2008)
17. Ryabov, A.B., Blasius, B.: Population growth and persistence in a heterogeneous environment: the role of diffusion and advection. *Math. Model. Nat. Phenom.* **3**(3), 42–86 (2008)
18. Poggiale, J.-C., Gauduchon, M., Auger, P.: Enrichment paradox induced by spatial heterogeneity in a phytoplankton–zooplankton system. *Math. Model. Nat. Phenom.* **3**(3), 87–102 (2008)
19. Siekmann, I., Malchow, H.: Local collapses in the Truscott–Brindley model. *Math. Model. Nat. Phenom.* **3**(3), 114–130 (2008)
20. Malchow, H., Petrovskii, S., Venturino, E.: *Spatiotemporal Patterns in Ecology and Epidemiology*. CRC, Boca Raton (2008)
21. Rhodes, C.J., Anderson, R.M.: Persistence and dynamics in lattice models of epidemic spread. *J. Theor. Biol.* **180**, 125–133 (1996)
22. Hui, C., Li, Z.Z.: Dynamical complexity and metapopulation persistence. *Ecol. Model.* **164**, 201–209 (2003)
23. Zhang, F., Hui, C., Han, X., Li, Z.: Evolution of cooperation in patchy habitat under patch decay and isolation. *Ecol. Res.* **20**, 461–469 (2005)
24. Hui, C., McGeoch, M.A., Warren, M.: A spatially explicit approach to estimating species occupancy and spatial correlation. *J. Anim. Ecol.* **75**, 140–147 (2006)
25. Tilman, D., Kareiva, P.: *Spatial Ecology: The Role of Space in Population Dynamics and Interspecific Interactions*. Princeton University Press, Princeton (1997)
26. Turing, A.M.: The chemical basis of morphogenesis. *Philos. Trans. R. Soc. Lond. B* **237**, 37–72 (1952)
27. Murray, J.D.: *Mathematical Biology*. Springer, Berlin (2002)
28. Okubo, A.: Diffusion and ecological problems: modern perspectives. In: *Interdisciplinary Applied Mathematics*, vol. 14. Springer, Berlin (2001)
29. Segel, L.A., Jackson, J.L.: Dissipative structure: an explanation and an ecological example. *J. Theor. Biol.* **37**, 545–559 (1972)
30. Auchmuty, J.F.G., Nicolis, G.: Bifurcation analysis of nonlinear reaction–diffusion equations—I. Evolution equation and the steady state solutions. *Bull. Math. Biol.* **37**, 323–365 (1975)
31. Pacala, S.W., Roughgarden, J.: Spatial heterogeneity and interspecific competition. *Theor. Popul. Biol.* **21**, 92–113 (1982)
32. Shigesada, N.: Spatial distribution of rapidly dispersing animals in heterogeneous environment. In: Levin, S.A., Hallam, T.G. (eds.) *Lecture Notes in Biomathematics*, vol. 54, pp. 478–491. Springer, Berlin (1984)
33. Cantrell, R.S., Cosner, C.: The effect of spatial heterogeneity in population dynamics. *J. Math. Biol.* **29**, 315–338 (1991)
34. Maini, P.K., Benson, D.L., Sherratt, J.A.: Pattern formation in reaction diffusion models with spatially inhomogeneous diffusion coefficients. *IMA J. Math. Appl. Med. Biol.* **9**, 197–213 (1992)
35. Benson, D.L., Sherratt, J., Maini, P.K.: Diffusion driven instability in an inhomogeneous domain. *Bull. Math. Biol.* **55**, 365–384 (1993)
36. Benson, D.L., Sherratt, J., Maini, P.K.: Pattern formation in reaction-diffusion models with spatially inhomogeneous diffusion coefficients. *Math. Comput. Model.* **17**, 29–34 (1993)
37. Sherratt, J.A.: Diffusion-driven instability in oscillating environments. *Eur. J. Appl. Math.*, **6**, 355–372 (1995)
38. Sherratt, J.A.: Turing bifurcations with a temporally varying diffusion coefficient. *J. Math. Biol.* **33**, 295–308 (1995)
39. Timm, U., Okubo, A.: Diffusion-driven instability in a predator–prey system with time varying diffusivities. *J. Math. Biol.* **31**, 307–320 (1992)
40. Levin, S.A., Segel, L.A.: Hypothesis for origin of planktonic patchiness. *Nature* **259**, 659 (1976)
41. Lafferty, K.D., Morris, A.K.: Altered behavior of parasitized killfish increases susceptibility to predation by bird final hosts. *Ecology* **77**, 1390–1397 (1996)
42. Scheffer, M., Rinaldi, S.A., Kuznetsov, Yu.A.: Effects of fish on plankton dynamics: a theoretical analysis. *Can. J. Fish. Aquat. Sci.* **57**, 1208–1219 (2000)
43. Lammens, E.H.R.R.: A test of a model for planktivorous filter feeding by bream *Abramis brama*. *Environ. Biol. Fishes* **13**, 289–296 (1985)

44. Mills, E.L., Forney, J.L.: Impact on *Daphnia pulex* of predation by young yellow perch *Perca flavescens* in Oneida Lake, New York. Trans. Am. Fish. Soc. **112**, 154–161 (1983)
45. Haque, M., Venturino, E.: The role of transmissible diseases in Holling–Tanner predator–prey model. Theor. Popul. Biol. **70**(3), 273–288 (2006)
46. Bennett, A.F., Dennman, K.L.: Phytoplankton patchiness: inferences from particle statistics. J. Mar. Res. **43**, 307–335 (1985)
47. Nayfeh, A.H.: Introduction to Perturbation Techniques. Wiley, New York (1993)