Role of migratory bird population in a simple eco-epidemiological model

SAMRAT CHATTERJEE and J. CHATTOPADHYAY*

Agricultural and Ecological Research Unit, Indian Statistical Institute, 203 B. T. Road, Kolkata 700108, India

Migratory birds play a vital role in the spread of diseases such as West Nile Virus, Salmonella, etc. In this paper we propose and analyse (both analytically and numerically) a single-season mathematical model to observe the dynamical changes that take place due to the introduction of a disease by migratory birds. We observe that the force of infection and the predation rate play important roles in maintaining stability around the positive steady state. We also observe that proper predation may even result in the extinction of the infective migratory prey population from the system.

Keywords: Eco-epidemiological system; Migratory bird population; Multi-positive steady state; Predation

1. Introduction

Hadeler and Freedman [1] were probably the first to describe a predator–prey model where the prey is infected by a parasite, and in turn infects the predator with the parasite. After the pioneering work of Hadeler and Freedman [1] quite a large number of papers were published on the predator–prey system with infection in the prey [2–4], but, to the best of our knowledge, none of these papers studied the effect of migration of the prey population, especially if the migratory prey population has the ability to carry a disease. Migration may introduce a new disease to a new location, or can even re-introduce a disease that was totally eliminated from that location. For example, the 1962 epidemic of EEE in Jamaica resulted from transport of the virus by birds from the continental United States [5]. In another example, West Nile Virus (WNV) was introduced to the Middle East by migrating white storks [6]. Although not verified in the field, it has been observed that a predator can be infected after predating a prey infected by this virus [http://environmentalrisk.cornell.edu/WNV]. An epidemiological model for West Nile Virus has been proposed by Wonham *et al*. [7], but they neglected

the effect of predation (if any) on those migratory birds that are responsible for the spread of WNV. The same problem arises in the case of *Salmonella* bacteria. It was observed that some wild migrating birds are responsible for the spread of these bacteria [8]. In another example, the highly pathogenic avian influenza virus is suspected of being re-introduced into Japan by wild migrating birds from South Korea [9]. This is an important problem, but, unfortunately, no real effort has been made by researchers thus far to study the changes made by these migratory populations in an ecosystem.

In this paper we shall propose and analyse a one-season model where the prey population migrates from one location to another carrying a disease. We have divided the migratory prey population into two groups, namely the susceptible prey and the infective prey. The predator population, apart from other sources, consumes both prey populations. Diseases spread by migratory birds, such as salmonella [10] and WNV [7], are season dependent. We are interested in observing the behaviour of a dynamical system for a certain period when migratory birds are present in the system. This within-season model is an important first step in understanding the role played by migratory birds in spreading a disease, and the effect of predation on migratory birds. Our analytical and numerical results suggest that the introduction of a disease via a migratory population makes the system unstable. We have also observed that proper predation may prevent extinction of the species.

The paper is organized as follows. In section 2 we present the basic mathematical model and the boundedness of the solution of the system. In section 3 we analyse all possible subsystems of our model system. The analysis of the whole system is shown in section 4. We perform a numerical analysis in section 5 to support our analytical findings. Finally, we discuss our findings in section 6.

2. Mathematical model and the boundedness of the solutions

Let us consider a prey population that enters an ecosystem where its predator population is already present. We assume that some of the members of this migratory prey population are carrying a disease. Once they enter the system, they start to spread the disease to other members of the population. Therefore, the total migratory prey population N present in our considered system can be divided into two classes, the susceptible prey population s and the infective prey population i. Therefore, at any time t the total migratory prey population is $N(t) = s(t) + i(t)$. The population density of the predator which is already present in the system is denoted by p. Before formulating the model equation we make the following assumptions concerning the predator population and the migratory prey population present in the system.

- (A1) The model considered here is for one season and, therefore, instead of taking logistic growth in the prey population we have considered a growth term known as constant immigration with exponential deaths [11] for the migratory prey population. Let A be the constant rate of recruitment of the prey population (including newborns and migration) [12,13] and d is the natural death rate of the prey population. Then the growth rate of the migratory prey population is given by

$$\frac{dN}{dt} = A - dN.$$

  In this case the population approaches A/d as t goes to infinity.

- (A2) The infective prey population is generated by the infection of susceptible prey. It is assumed that the infective prey population is not in a state of reproduction. However, as time passes, some of them recover from the disease and again become susceptible. Therefore, the dynamics of the prey population may be written as

$$\frac{ds}{dt} = A - bsi - ds + fi,$$
$$\frac{di}{dt} = bsi - (e+f)i,$$

where b is the force of infection, f is the recovery rate, and e is the death rate of the infective prey population, which includes natural death and death due to the disease. Obviously, $e \geq d$.

- (A3) Here we shall study the dynamics of the system for the season when the migratory prey is present. However, in the absence of migratory prey, we assume that the predator population is present in the system. Therefore, in the absence of migratory prey, i.e. outside the considered season, there must exist some alternative resource for the growth of the predator population. Depending on that alternative resource the predator population is assumed to grow in a logistic fashion with carrying capacity $k > 0$ and intrinsic growth rate constant $r > 0$. Hence, in the absence of migratory prey, the growth equation of the predator is given by

$$\frac{dp}{dt} = rp\left(1 - \frac{p}{k}\right).$$

Now, in the presence of the migratory prey it is not possible for the predator population to switch its predation totally from the alternative resource to the newly available migratory prey population. Therefore, in our model formulation we assume that the growth rate of the predator population is governed by both the alternative source and the migratory prey population.

- (A4) Here we also assume that the predator population becomes infected after predation of the infective prey. For example, cats that predate on song birds infected with salmonella can pick up the illness and die [http://www.gov.nf.ca/agric/pubfact/salmonella.htm]. Therefore, predation of the infected prey population is included in the predator's growth equation with a negative sign, as done by Chattopadhyay *et al.* [14] in an eco-epidemiological context. However, it is assumed that the infection does not spread among the predator population because either the infected predators die immediately after becoming infected by the disease and are thus removed from the system [15], or they are the dead-end host of the disease like mammals in the case of WNV [7].
- (A5) Further, for mathematical simplicity we assume that the functional response (prey eaten per predator per unit of time) and the mode of disease transmission follow the simple law of mass action.

From the above assumptions, we can now write down the following differential equations:

$$\begin{aligned}
\frac{ds}{dt} &= A - bsi - k_1 sp - ds + fi, \\
\frac{di}{dt} &= bsi - k_2 pi - (e+f)i, \\
\frac{dp}{dt} &= rp\left(1 - \frac{p}{k}\right) + k_1' sp - k_2' pi,
\end{aligned} \tag{1}$$

where $p(0) \geq 0$, $s(0) \geq 0$ and $i(0) \geq 0$. Here, k_1 and k_2 are the searching efficiency constants or the predation rate on the susceptible and infective prey population, respectively, and k_1' and k_2' are the growth rates of the predator due to predation of the susceptible and infective prey population. Obviously, $k_1 \geq k_1'$ and $k_2 \geq k_2'$. Also, $k_1 \leq k_2$, which is quite natural. For example, in the case of salmonella, sick birds often appear uncomfortable, with heads drooped, wings out, feathers fluffed up, and breathing heavily, and this type of behaviour leaves them more susceptible to predation by large birds or cats [http://www.gov.nf.ca/agric/pubfact/salmonella.htm] [16].

For simplicity, we write model (1) in dimensionless form by using the variable transformations $P = p/k$, $S = s$, $I = i$ and $\tau = rt$. With these quantities the system is transformed into a dimensionless form:

$$\begin{aligned} \frac{dS}{d\tau} &= B - \lambda SI - \alpha SP - \delta S + \mu I, \\ \frac{dI}{d\tau} &= \lambda SI - \beta IP - (\gamma + \mu)I, \\ \frac{dP}{d\tau} &= P(1 - P) + \alpha' SP - \beta' PI, \end{aligned} \tag{2}$$

where $\lambda = b/r$, $B = A/r$, $\delta = d/r$, $\gamma = e/r$, $\mu = f/r$, $\alpha = kk_1/r$, $\beta = kk_2/r$, $\alpha' = k_1'/r$ and $\beta' = k_2'/r$.

LEMMA 2.1 *All solutions of (2) that initiate in $\Re^3_{0,+}$ are uniformly bounded.*

Proof We define a function

$$W = S + I + P. \tag{3}$$

The time derivative of (3) along the solutions of (2) is given by

$$\begin{aligned} \frac{dW}{d\tau} &= B - \alpha SP - \delta S - \beta PI - \gamma I + P(1 - P) + \alpha' SP - \beta' PI \\ &\leq B - (\delta S + \gamma I) + P(1 - P) \quad (\text{since } \alpha' \leq \alpha). \end{aligned}$$

Taking $\eta > 0$ we obtain

$$qB + (\eta - \delta)S + (\eta - \gamma)I + (\eta + 1 - P)P.$$

Now if we choose $\eta \leq \min(\delta, \gamma)$, then

$$\begin{aligned} \frac{dW}{d\tau} + \eta W &\leq B + P(\eta + 1 - P) \\ &\leq B + \frac{(1 + \eta)^2}{4}. \end{aligned}$$

It is clear that the right-hand side of the above expression is bounded. Therefore, we can find a constant $l > 0$ such that

$$\frac{dW}{d\tau} + \eta W \leq l.$$

Applying a theory of differential inequality [17], we obtain

$$0 < W(S, I, P) < \frac{I}{\eta}(1 - e^{-\eta t}) + W(S(0), I(0), P(0))e^{-\eta t}.$$

For $t \to \infty$ we have $0 < W < I/\eta$. Hence, all solutions $(S(t), I(t), P(t))$ of (2) that initiate at $(S(0), I(0), P(0)) \varepsilon \Re^3_{0,+}$ are confined in the region

$$G = \left\{ (S, I, P) \varepsilon \Re^3_{0,+} : W = \frac{I}{\eta} + \theta, \quad \text{for any } \theta > 0 \right\},$$

for all $t \geq T$, where T depends on the initial values $(S(0), I(0), P(0))$. Thus, the set G is an invariant set which contains the $\Omega-$ limit set of all the paths of system (2) that initiate in the positive octant. □

Now we are interested in observing the dynamics of the following subsystems.

3. Dynamics of all possible subsystems of (2)

3.1 *Analysis of system (2) in the absence of disease*

In the absence of the disease, system (2) takes the form

$$\begin{aligned} \frac{dS}{d\tau} &= B - \alpha SP - \delta S, \\ \frac{dP}{d\tau} &= P(1 - P) + \alpha' SP. \end{aligned} \tag{4}$$

This system admits two equilibria, namely the axial equilibrium $E_0^1 \equiv (B/\delta, 0)$ and the interior equilibrium point $E_1^1 \equiv (\bar{S}, \bar{P})$, where $\bar{P} = 1 + \alpha' \bar{S}$ and $\bar{S}$ is given by

$$\alpha\alpha' \bar{S}^2 + (\alpha + \delta)\bar{S} - B = 0. \tag{5}$$

By Descartes' rule of signs, equation (5) has exactly one positive root. Therefore, the interior equilibrium exists for any parametric value.

THEOREM 3.1 *System (4) is globally asymptotically stable.*

Proof It can easily be verified that system (4) is bounded. Now, using the variational matrix method we find that E_0^1 is a saddle point and E_1^1 is a locally stable equilibrium point. Now, we will test for the existence or non-existence of a periodic solution around the positive equilibrium. To do this we shall use the Dulac criterion [18]. Let $h(S, P) = 1/SP$ and $D_1 = (\partial(hf)/\partial S) + (\partial(hg)/\partial P)$, where

$$\begin{aligned} f(S, P) &= B - \alpha SP - \delta S, \\ g(S, P) &= P(1 - P) + \alpha' SP. \end{aligned}$$

Therefore, $D_1 = -(B/PS^2) - (1/S) < 0$.

This result shows that there is no non-trivial positive periodic solution around the interior equilibrium of subsystem (4). Thus, subsystem (4) is globally asymptotically stable. □

3.2 *Analysis of system (2) in the absence of the predator*

In the absence of the predator, system (2) takes the form

$$\begin{aligned}\frac{dS}{d\tau} &= B - \lambda SI - \delta S + \mu I,\\ \frac{dI}{d\tau} &= \lambda SI - (\gamma + \mu) I.\end{aligned} \tag{6}$$

This system also admits two equilibria, namely the axial $E_0^p \equiv (B/\delta, 0)$ and the interior equilibrium point

$$E_1^p \equiv \left(\frac{\gamma+\mu}{\lambda}, \frac{\lambda B - (\gamma+\mu)\delta}{\lambda\gamma}\right).$$

Note that E_1^p exists if $\lambda > [(\gamma+\mu)\delta]/B$.

THEOREM 3.2 *The existence of the positive interior equilibrium point E_1^p of subsystem (6) ensures its global asymptotic stability.*

Proof Here also it can easily be verified that subsystem (6) is bounded. Moreover, the existence of the interior equilibrium point E_1^p ensures that the equilibrium point E_0^p is a saddle point and that the interior equilibrium point E_1^p is a locally asymptotically stable point.

Now, we will test for the existence or non-existence of a periodic solution around the positive equilibrium. To do this we shall again use the Dulac criterion [18].

Let $h'(S, I) = 1/SI$ and $D_1 = (\partial(h'f')/\partial S) + (\partial(h'g')/\partial I)$, where

$$\begin{aligned}f'(S,I) &= B - \lambda SI - \delta S + \mu I,\\ g'(S,I) &= \lambda SI - (\gamma+\mu) I.\end{aligned}$$

Therefore, $D_1 = -(B/IS^2) - (\mu/S^2) < 0$.

This result shows that there is no non-trivial positive periodic solution around the interior equilibrium point. This proves the theorem. □

4. Analysis of the whole system (2)

4.1 *The equilibrium points and the conditions for their existence*

System (2) possesses the following biologically feasible equilibria. $E_1 \equiv (B/\delta, 0, 0)$, $E_2 \equiv (S', 0, P')$, where $S' = (P'-1)/\alpha'$ and P' is given by the positive root of the quadratic equation $\alpha P^2 + (\delta - \alpha)P - (\delta + B\alpha') = 0$, and $E_3 \equiv (\bar{S}, \bar{I}, 0)$, where $\bar{S} = (\gamma+\mu)/\lambda$ and $\bar{I} = [B\lambda - \delta(\gamma+\mu)]/\gamma\lambda$.

Remark 1 Equilibria E_1 and E_2 exist for any parametric value, whereas E_3 exists if $\lambda > [(\gamma+\mu)\delta]/B$.

We now seek the regions of the parameter space for which model system (2) admits a feasible interior equilibrium (equilibria). Any feasible equilibrium must correspond to a positive root S^* of the quadratic equation

$$g(x) = 0, \tag{7}$$

where

$$g(x) = w_1 S^2 + w_2 S + w_3,$$

and w_1, w_2 and w_3 are given by

(i) $w_1 = \lambda(\lambda - \beta\alpha' - \alpha\beta')$,
(ii) $w_2 = \alpha\beta'(\gamma+\mu) - \lambda(\beta+\gamma+\mu) - \delta\beta\beta' + \mu(\alpha'\beta - \lambda)$,
(iii) $w_3 = B\beta\beta' + \mu(\beta+\gamma+\mu)$,

for which, additionally,

$$I^* = \frac{(\beta\alpha' - \lambda)S^* + (\beta+\gamma+\mu)}{\beta\beta'} \qquad \text{and} \qquad P^* = \frac{\lambda S^* - (\gamma+\mu)}{\beta}.$$

Now, we describe the range of possibilities for which an interior positive equilibrium (equilibria) exists.

Case Ia. If $\lambda < \beta\alpha'$, then by Descartes' rule of signs there exists exactly one positive root of equation (7). Also, $I^* > 0$. Now, if this positive root is greater than $(\gamma+\mu)/\lambda$, then $P^* > 0$. Hence, if $\lambda < \beta\alpha'$, and the positive root of equation (7) is greater than $(\gamma+\mu)/\lambda$, then there exists a unique interior equilibrium point $E^*(S^*, I^*, P^*)$.

Case IIa. If $\beta\alpha' < \lambda < \beta\alpha' + \alpha\beta'$, then by Descartes' rule of signs, here also equation (7) has exactly one positive root. Now for $I^* > 0$, this positive root must be less than $(\beta+\gamma+\mu)/(\lambda - \beta\alpha')$, and for $P^* > 0$ this positive root must be greater than $(\gamma+\mu)/\lambda$. Hence, if $\beta\alpha' < \lambda < \beta\alpha' + \alpha\beta'$, and the positive root of equation (7) lies in the interval $((\gamma+\mu)/\lambda, (\beta+\gamma+\mu)/(\lambda - \beta\alpha'))$, then in this case also there exists a unique interior equilibrium point $E^*(S^*, I^*, P^*)$.

Case IIIa. If $w_1 > 0$ and $w_2 < 0$, then by Descartes' rule of signs, equation (7) has two positive roots. For $I^* > 0$ and $P^* > 0$ these positive roots must lie in the interval $((\gamma+\mu)/\lambda, (\beta+\gamma+\mu)/(\lambda - \beta\alpha'))$. Now, if one positive root lies in the interval $((\gamma+\mu)/\lambda, (\beta+\gamma+\mu)/(\lambda - \beta\alpha'))$, then there exists one interior equilibrium point, and if both positive roots lie in the interval $((\gamma+\mu)/\lambda, (\beta+\gamma+\mu)/(\lambda - \beta\alpha'))$, then there exists two interior equilibrium points. However, if no root lies within that interval, then no interior stationary solution exists.

4.2 *Local stability analysis (LAS)*

The variational matrix J of system (2) is given by

$$J = \begin{bmatrix} -\lambda I - \alpha P - \delta & -\lambda S + \mu & -\alpha S \\ \lambda I & \lambda S - \beta P - (\gamma+\mu) & -\beta I \\ \alpha' P & -\beta' P & 1 - 2P + \alpha' S - \beta' I \end{bmatrix}.$$

THEOREM 4.1 *The axial equilibrium E_1 is a saddle point with the S axis as a stable manifold and the IP plane as an unstable manifold if $\lambda > [(\gamma+\mu)\delta]/B$ or the P axis as an unstable manifold and the SI plane as a stable manifold if $\lambda < [(\gamma+\mu)\delta]/B$.*

THEOREM 4.2 *If*

$$\lambda > \frac{\alpha'\{\beta(\delta-\alpha-Q)-2\alpha(\gamma+\mu)\}}{\delta+\alpha-Q},$$

where $Q = \sqrt{(\delta-\alpha)^2+4\alpha(\delta+B\alpha')} > 0$ *holds, then E_2 is a LAS.*

Proof The variational matrix of system (2) at E_2 is given by

$$J_2 = \begin{bmatrix} -\alpha P' - \delta & -\lambda S' + \mu & -\alpha S' \\ 0 & \lambda S' - \beta P' - (\gamma+\mu) & 0 \\ \alpha' P' & -\beta' P' & -P' \end{bmatrix}.$$

The characteristic equation is given by

$$\{x - (\lambda S' - \beta P' - (\gamma+\mu))\}\left\{x^2 + \left(\frac{B}{S'} + P'\right)x + \frac{BP'}{S'} + \alpha\alpha' S' P'\right\} = 0. \tag{8}$$

Therefore, the system around E_2 is locally asymptotically stable if $\lambda S' < \beta P' + (\gamma+\mu)$, i.e. if $P' < [(\gamma+\mu)\alpha' + \lambda]/(\lambda - \beta\alpha')$. Substituting the value of P' and after some simple algebraic calculations we obtain the required conditions for the LAS of E_2.

THEOREM 4.3 *If $\lambda > [(\gamma+\mu)(\alpha'\gamma+\beta'\delta)]/(\beta' B - \gamma)$ holds, then E_3 is locally asymptotically stable (LAS).*

Proof The variational matrix of system (2) at E_3 is given by

$$J_3 = \begin{bmatrix} -\lambda\bar{I} - \delta & -\lambda\bar{S} + \mu & -\alpha\bar{S} \\ \lambda\bar{I} & 0 & -\beta\bar{I} \\ 0 & 0 & 1 + \alpha'\bar{S} - \beta'\bar{I} \end{bmatrix}.$$

The characteristic equation is given by

$$\{x - (1 + \alpha'\bar{S} - \beta'\bar{I})\}\{x^2 + (\lambda\bar{I} + \delta)x + \lambda^2\bar{S}\bar{I} - \mu\lambda\bar{I}\} = 0. \tag{9}$$

Since $\lambda\bar{S} > \mu$, E_3 is LAS if $1 + \alpha'\bar{S} - \beta'\bar{I} < 0$, i.e. if

$$\lambda > \frac{(\gamma+\mu)(\alpha'\gamma+\beta'\delta)}{\beta' B - \gamma}.$$

For the local stability analysis of the positive equilibrium point we follow the technique used by Chattopadhyay and Pal [19]. □

THEOREM 4.4 *If there is exactly one interior stationary solution $E^*(S^*, I^*, P^*)$, $S^* > 0$, $I^* > 0$, $P^* > 0$, then it is a sink provided $w_1 > 0$ and $w_2 < 0$. If there are two interior stationary solutions, then one is a saddle and the other is a sink.*

Proof The variational matrix of system (2) at E^* is given by

$$J^* = \begin{bmatrix} -\frac{B+\mu I^*}{S^*} & -\lambda S^* + \mu & -\alpha S^* \\ \lambda I^* & 0 & -\beta I^* \\ \alpha' P^* & -\beta' P^* & -P^* \end{bmatrix}.$$

Here,

$$\begin{aligned} \text{trace } J^* &= -\left(\frac{B+\mu I^*}{S^*} + P^*\right) < 0, \\ \det J^* &= -\frac{I^* P^*}{S^*}(w_1 S^{*2} - w_3), \\ D(J^*) &= \text{trace } J^* \times M(J^*) - \det J^*, \end{aligned}$$

therefore

$$\begin{aligned} D(J^*) = -\Bigg[& \frac{(B+\mu I^*)\lambda I^*}{S^*}(\lambda S^* - \mu) + \frac{(B+\mu I^*)^2 P^*}{S^{*2}} + \frac{(B+\mu I^*)P^{*2}}{S^*} \\ & + (B+\mu I^*)\alpha\alpha' P^* + \alpha\alpha' S^* P^{*2} + \beta\alpha'(\lambda S^* - \mu) P^* I^* \\ & + \beta'\{(\alpha - 1)\lambda S^* + \gamma + \mu\} P^* I^* \Bigg], \end{aligned}$$

where $M(J^*)$ is the sum of the second-order principal minors of J^*.

To examine the local stability of the interior equilibrium (equilibria), suppose that there is one positive equilibrium $E^*(S^*, I^*, P^*)$ or two positive equilibria $E_1^*(S_1^*, I_1^*, P_1^*)$ and $E_2^*(S_2^*, I_2^*, P_2^*)$. Then (S_i^*, I_i^*, P_i^*) satisfy

$$g(x_i^*) = w_1 S_i^{*2} + w_2 S_i^* + w_3 = 0,$$

and

$$\begin{aligned} I_i^* &= \frac{(\beta\alpha' - \lambda)S_i^* + (\beta + \gamma + \mu)}{\beta\beta'}, \\ P_i^* &= \frac{\lambda S_i^* - (\gamma + \mu)}{\beta}, \quad i = 1, 2. \end{aligned}$$

Let us define the function

$$h(x_i^*) = w_1 S_i^{*2} - w_3.$$

The signs of $h(x_i^*)$ and $\det J^*$ are opposite. It is useful to compare the two functions g and h. Suppose these two functions coincide at $S = S_q$. Then,

$$g(S_q) = h(S_q),$$

or, equivalently,

$$S_q = \frac{-2w_3}{w_2}.$$

At S_q,

$$g(S_q) = -\frac{w_3(w_2{}^2 - 4w_1w_3)}{w_2{}^2}.$$

Since we are only interested in the real roots of equation (7), we shall confine our attention to the case $w_2^2 - 4w_1w_3 > 0$. Thus, the sign of $g(S_q)$ is opposite to the sign of w_3. As $w_3 > 0$, $g(S_q) < 0$. We now consider each of the cases of theorem 4.4 in turn.

Case Ib. We observe that, if $\lambda < \beta\alpha'$, then there exists exactly one positive equilibrium $E^*(S^*, I^*, P^*)$. It is easy to verify that $\det J^* > 0$, since $w_1 < 0$ and $w_3 > 0$. Hence E^* is a saddle.

Case IIb. We observe that, if $\beta\alpha' < \lambda < \beta\alpha' + \alpha\beta'$, then there is also a unique positive equilibrium $E^*(S^*, I^*, P^*)$. Here also, $J^* > 0$, since $w_1 < 0$ and $w_3 > 0$. Hence E^* is a saddle.

Case IIIb. If $w_1 > 0$ and $w_2 < 0$, then it can easily be verified that

(iv) trace $J^* < 0$;
(v) $D(J^*) = \text{trace } J^* \times M(J^*) - \det J^* < 0$.

For the above conditions we observe that equation (7) has two positive roots. Denote these roots by S_1 and S_2 with $0 < S_1 < S_2$ (say), and corresponding $I_1 > I_2 > 0$, since $\lambda > \beta\alpha'$. Assume that there exists at least one interior equilibrium point, so that $I_1 > 0$. Now, at $S = S_q$, $g(S_q) < 0$. Hence $0 < S_1 < S_q < S_2$ and, therefore,

$$\det J^*(S_2, I_2, P_2) = -\frac{I^*P^*}{S^*}h(S_2) < -\frac{I^*P^*}{S^*}g(S_2) = 0.$$

Hence, $E_2^*(S_2^*, I_2^*, P_2^*)$ is a sink. If we have two interior equilibria, then $I_1 > I_2 > 0$ and

$$\det J^*(S_1, I_1, P_1) = -\frac{I^*P^*}{S^*}h(S_1) > -\frac{I^*P^*}{S^*}g(S_1) = 0.$$

In this case, $E_1^*(S_1^*, I_1^*, P_1^*)$ is a saddle.

5. Numerical analysis

The dynamics of system (1) around the positive steady state has been simulated numerically for a wide range of parameter values. The force of infection b, and the predation rate on the susceptible prey population k_1 and the infective prey population k_2 are the three key parameters that directly influence the dynamics of the system. Thus, we have studied the dynamics of the system for a wide variation in b, k_1 and k_2. We have also studied the dynamics of the system in the absence of the recovery rate f to determine the role of recovery in the system.

5.1 *Dynamics of system (1) when $f \neq 0$*

In our numerical experiments we have taken a hypothetical set of parametric values: $A = 7$ individuals ha^{-1} day^{-1}, $r = 0.33$ day^{-1}, $k = 40$ individuals ha^{-1},

$k_1' = 0.6 \times k_1$ ha per individual day^{-1}, $k_2' = 0.6 \times k_2$ ha per individual day^{-1}, $d = 0.009$ day^{-1}, $f = 1.8$ day^{-1}, $e = 0.0097$ day^{-1}, $b = 0.1$ ha per individual day^{-1}, $k_1 = 0.003$ ha per individual day^{-1}, and $k_2 = 0.003$ ha per individual day^{-1}. For these values $w_1 > 0$ and $w_2 < 0$ and so equation (7) has two positive roots. Substituting these parameter values in equation (7) and solving we obtain $S_1 = 18.41$ and $S_2 = 19.15$. Again substituting these parameter values and S_1 and S_2 in the expression for I^* and P^* we observe that system (2) possesses two positive equilibria, namely $E_1^*(S_1^* = 18.41, I_1^* = 154.64, P_1^* = 0.25)$ and $E_2^*(S_2^* = 19.15, I_2^* = 41.96, P_2^* = 0.87)$. The eigenvalues associated with the variational matrix of system (2) at E_1^* and E_2^* are $(-66.96, -0.476, 0.133)$ and $(-33.03, -0.03, -0.98)$, respectively. Therefore, the positive equilibrium point E_1^* is a saddle, whereas the positive equilibrium point E_2^* is a sink, supporting our analytical results. Therefore, we will concentrate our analysis around the positive steady state E_2^*. One should note here that these population values at E_2^* are in dimensionless form. The actual values of the populations at E_2^* are (19.15, 41.96, 34.80). Substituting these parametric values in system (1) and taking the initial population of the susceptible prey, the infective prey and the predator as 3.4 individuals ha^{-1}, 0.94 individuals ha^{-1} and 1.2 individuals ha^{-1}, respectively (which remains fixed for the entire numerical analysis), we obtain the results shown in figure 1.

Now keeping the other parameters fixed we studied the dynamics of the system for a wide variation in b $(0.001 \le b \le 4)$, k_1 $(0.001 \le k_1 \le 0.01)$ and k_2 $(0.003 \le k_2 \le 0.9)$, keeping in mind that $k_1 \le k_2$. We start with the parameter b. Keeping the value of k_1 $(=0.003)$ and k_2 $(=0.01)$ fixed we varied the parameter b and observed that all the population of the system coexists for a wide variation in b $(0.052 \le b \le 0.111)$.

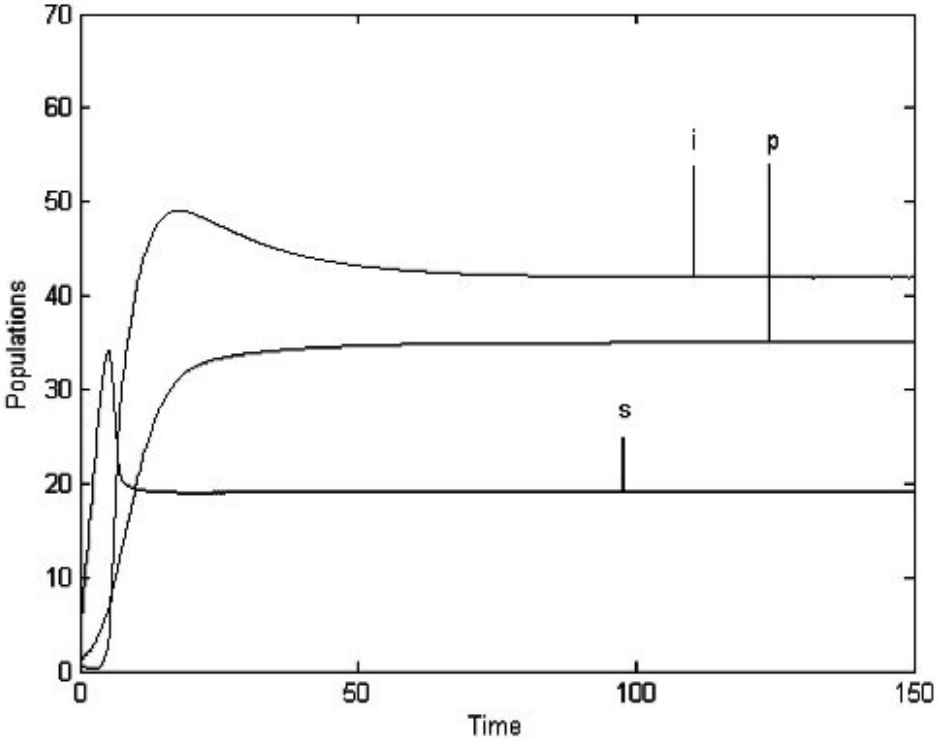

Figure 1. Time evolution of all populations for $b = 0.1$.

However, if we take a value of b below 0.052 ($b_{\min}$), then we observe that the infective prey is eliminated from the system (see figure 2(a)). If b becomes greater than 0.111 ($b_{\max}$), then it leads to the extinction of the predator population from the system (see figure 2(b)).

Therefore, it is clear that by keeping the force of infection below $b_{\min}$, one can make the system disease-free. This result also indicates that, to save the predator population, we have to keep the force of infection below $b_{\max}$.

In addition to the epidemiological parameter (b), the predation rates on the susceptible and infective prey, i.e. k_1 and k_2 respectively, also play vital roles in the dynamics of the system. Therefore, we then varied the parameters k_1 and k_2 keeping the value of b (0.1) fixed. We first varied the value of k_1 keeping k_2 (0.01) fixed, and then varied k_2 keeping k_1 (0.003) fixed. We observed that all three populations coexist for a wide variation in k_1 ($0.0024\ (k_{1(\min)}) \leq k_1 \leq 0.0059\ (k_{1(\max)})$) and k_2 ($0.003\ (k_{2(\min)}) \leq k_2 \leq 0.012\ (k_{2(\max)})$). Beside these ranges of k_1 and k_2 either the predator population or the infective prey population is eliminated from the system. For example, if we take a value of k_1 below $k_{1(\min)}$, then the predator population is eliminated from the system (see figure 3). Thus these threshold phenomena of predation may provide some input for environmental ecologists to develop a suitable policy for controlling the dynamics of such systems.

To understand the above analysis better, we refer the reader to table 1.

The time evolution of all populations of system (1) for the fixed set of parameter values of table 1 with $b = 0.1$, $k_1 = 0.003$ and $k_2 = 0.01$ is depicted in figure 4.

5.2 *Dynamics of system (1) when $f = 0$*

The time evolution of different populations of system (1) with $f = 0$ is depicted in figure 5 with the same set of parameter values and initial populations as in figure 4.

It is interesting to note that, in this case, for the same parameter values, the predator population becomes extinct in the absence of recovery (see figure 5), whereas in the

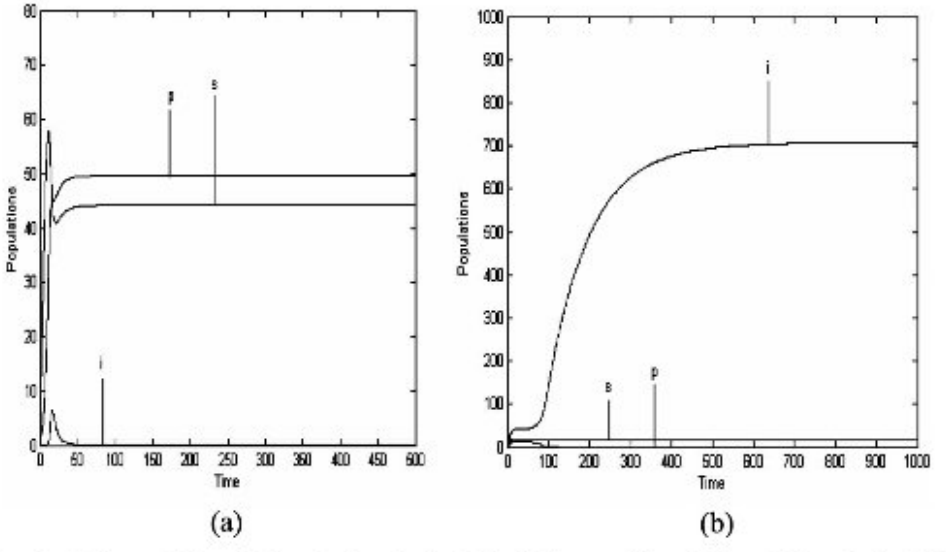

Figure 2. (a) Time evolution of all populations for $b = 0.051$. (b) Time evolution of all populations for $b = 0.112$.

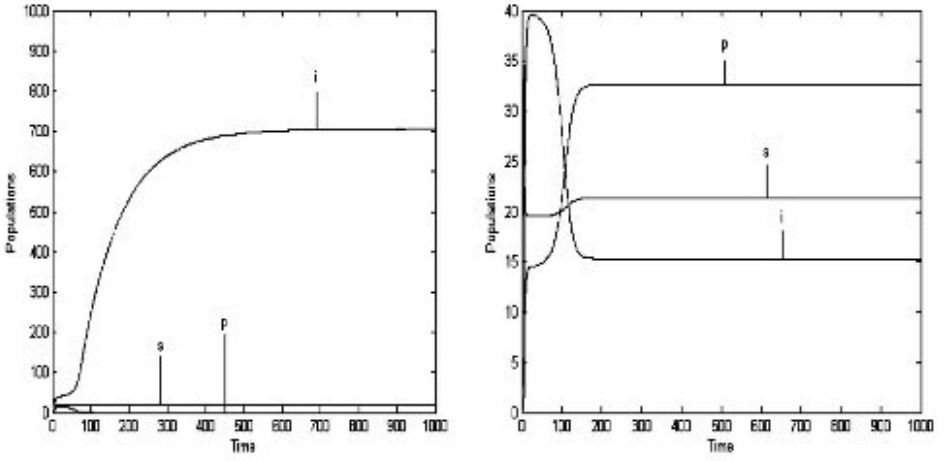

Figure 3. (*a*) Time evolution of all the populations for $k_1 = 0.0023$. (*b*) Time evolution of all populations for $k_1 = 0.0024$.

Table 1. Simulation experiments for model system (1) with fixed parameter values: $A = 7$, $r = 0.33$, $k = 40$, $k_1' = 0.6 \times k_1$, $k_2' = 0.6 \times k_2$, $d = 0.009$, $f = 1.8$, $e = 0.0097$.

Parameters fixed	Value of fixed parameter	Parameter varied	Ranges in which parameter is varied	Dynamic behaviour
k_1, k_2	$k_1 = 0.003$, $k_2 = 0.01$	b	$0.001 \le b \le 0.051$	Infective prey population becomes extinct
			$0.052 \le b \le 0.111$	All populations coexist
			$0.112 \le b \le 4$	Predator population becomes extinct
b, k_2	$b = 0.1$, $k_2 = 0.01$	k_1	$0.001 \le k_1 \le 0.0023$	Predator population becomes extinct
			$0.0024 \le k_1 \le 0.0059$	All populations coexist
			$0.006 \le k_1 \le 0.01$	Infective prey population becomes extinct
b, k_1	$b = 0.1$, $k_1 = 0.003$	k_2	$0.003 \le k_2 \le 0.012$	All populations coexist
			$0.013 \le k_2 \le 0.193$	Predator population becomes extinct
			$0.194 \le k_2 \le 0.9$	Infective prey population becomes extinct

presence of recovery, all populations persist (see figure 4). Moreover, from figure 5, we can also observe that there is a remarkable variation in the susceptible and infective prey population levels. The susceptible population declines from an initial population of 3.4 individuals ha^{-1} to 0.0978 individuals ha^{-1}; on the other hand, the infective population increases from an initial population of 0.94 individuals ha^{-1} to 715.591 individuals ha^{-1}. This remarkable increment in the infective prey population leads to the extinction of the predator population. To observe the role of the force of infection in such a situation, we keep all the other parameters fixed and only vary the parameter b. Note that the predator population becomes extinct if $b < 0.015$, whereas the infective population dies out if $b < 0.012$. Thus, in the interval $0.012 \le b \le 0.014$, all three populations coexist. However, when $f \neq 0$, we observe from table 1 that the

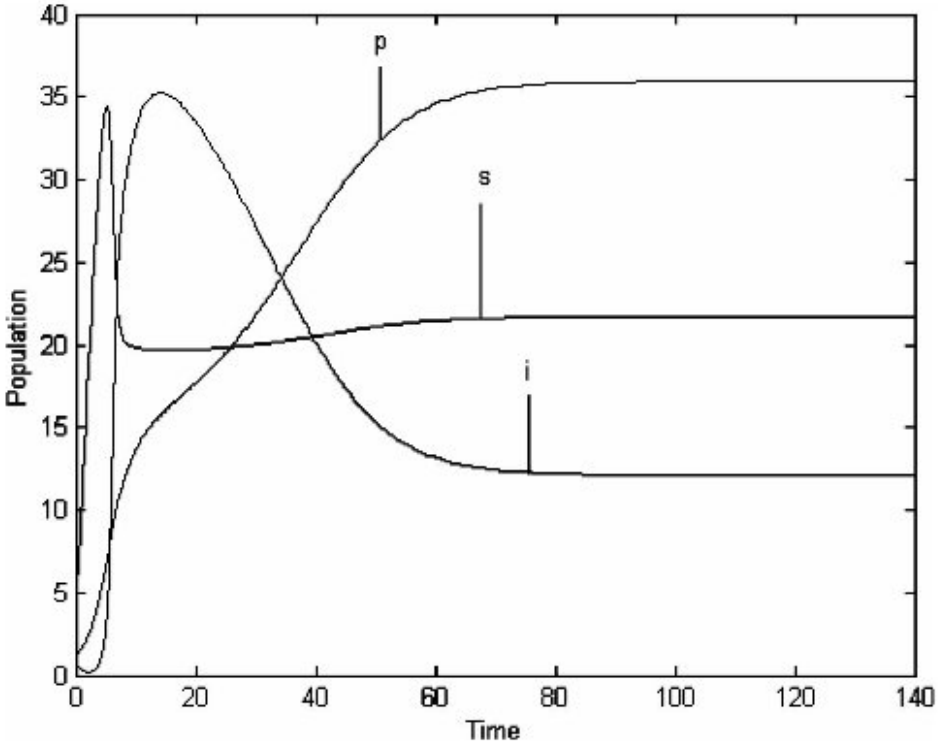

Figure 4. The coexistence of all populations.

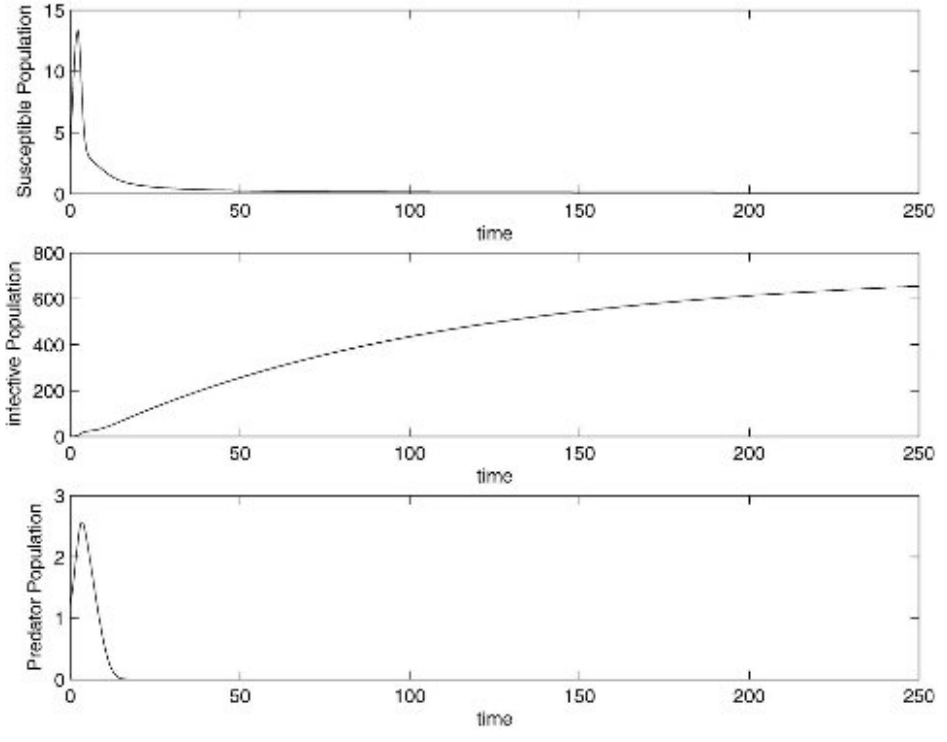

Figure 5. Time evolution of different populations in the absence of recovery.

system is stable around the interior equilibrium point in the interval $0.052 \leq b \leq 0.111$ and the system becomes disease-free if the value of $b \leq 0.052$. This result shows that recovery enhances the persistence of the species, which is quite natural.

6. Discussion

In this paper we have attempted to observe the dynamical behaviour of an ecosystem where disease is introduced by a migratory population. We have shown that, in the absence of disease in the migratory population, the system is globally stable (see theorem 4.4). However, the introduction of a disease by the migratory population makes the system unstable around the interior equilibrium point. This result is similar to the result of Beltrami and Carroll [20].

We have shown that the system is stable around the interior equilibrium point provided $w_1 > 0$ and $w_2 < 0$ (see theorem 4.4). Moreover, we have proved (both analytically and numerically) that if there are two interior equilibria, one is a sink and the other is a saddle. We have also shown that, by controlling the parameter λ (i.e. controlling the force of infection b), the system will be disease-free under realistic biological conditions (see theorem 4.2). Our observations from theorem 4.3 indicate that we shall have to monitor the parameter λ (which is directly associated with the rate of infection b) very carefully, otherwise there is a high possibility of the extinction of the predator population. This finding is more transparent from our numerical experiments. Our numerical result shows that the system is disease-free for $b < b_{\min}$. Thus we may conclude that the force of infection plays an important role in system dynamics.

We also observe from our numerical analysis that, in the absence of a recovery rate, i.e. when $f = 0$, it is very difficult to make the system disease-free. Also, it is very difficult to keep the system stable around the interior equilibrium point. Therefore, recovery plays an important role in avoiding the outbreak of the disease.

The predation rate is another parameter that plays a vital role in the dynamics of the system. We observe that, in the absence of the predator population, both the susceptible and the infective prey population coexists and the system is globally stable (see theorem 3.2). However, in the presence of a predator, system (2) is stable around the equilibrium point $(S', 0, P')$ for a suitable range of b. From our numerical experiments it is clear that the system will be disease-free if we keep the predation parameter $k_1 > k_{1(\max)}$ and $k_2 > k_{2(\max)}$. Thus, we may conclude that proper predation may help to make the system disease-free. This claim is also in agreement with the observations of Sih *et al.* [21]. They reviewed predator-removal experiments in which the predator population was removed from the system and observed the effect on the prey population infected by a transmissible disease. They found 54 of 135 systems in which the prey population subsequently declined. The same result was obtained by Hudson *et al.* [22] in another experiment, where they examined the interaction between red grouse, the parasite nematode *Trichostrongylus tenuis* and their predators.

However, in the real world it is very difficult to eliminate an infection totally from a system by predation. Removal experiments will fail if the migratory population is very large, for example if there are over 60 species of birds carrying WNV.

Finally, we would like to mention that there are several ways to extend our mathematical model. In our model formulation we have not considered the case where the disease can spread from one predator to another. We have also assumed that the predator is infected by predation only, but there are other ways by which the predator

population may become infected. In spite of these limitations, we believe that our approach and findings will inspire modelers and experimental ecologists to carry out further studies.

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