Controlling disease in migratory bird population: a probable solution through mathematical study

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Controlling disease in migratory bird population: a probable solution through mathematical study

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The role of migratory birds in an eco-system cannot be ignored. It becomes more important if they carry a disease. Chatterjee and Chattopadhyay proposed and analysed a three component one season eco-epidemiological model consisting of susceptible migratory birds, infective migratory birds and their predator population (see Chatterjee, S. and Chattopadhyay, J., Role of migratory bird populations in a simple eco-epidemiological model, *Mathematical and Computer Modelling of Dynamical systems*, in press). They assumed that the recovered infective birds become susceptible again. But, it is observed that in diseases like salmonella and WNV, the recovered birds develop a permanent immunity. Keeping this in mind we modify the model of Chatterjee and Chattopadhyay by adding a recovered class. The main objective of this work is to observe the role of recovery and immunity in such a system. Numerical simulations for a hypothetical set of parameter values are presented to illustrate the analytical findings. It is observed that to obtain a disease free system proper vaccination and proper predation are necessary. The second factor was mentioned in the paper of Chatterjee and Chattopadhyay.

1. Introduction

Ecology and epidemiology are major fields of study in their own right. But there are some common features between these two systems. The study of ecological systems with the influence of epidemiological parameters is now termed eco-epidemiology. Hadeler and Freedman [1] were probably the first to describe a predator–prey model where prey is infected by a parasite, and the prey in turn infect the predator with the parasite. The literature on eco-epidemiological systems is now rich [2–5]. But, as far as we know, the effect of the migration of the prey population has not been considered, especially if the migratory prey population has the ability to carry a disease. Such a situation has its own importance. Migration may introduce a new disease to a new place, or can even reintroduce a disease which had disappeared from that place. For example, the 1962 epidemic of EEE in Jamaica resulted from transport of the virus by birds from the continental United States [6]. In another example, the

West Nile Virus (WNV) was introduced in the Middle East by migrating white storks [7]. It is observed that a predator can become infected by predation of a prey infected by WNV [8, 9]. An epidemiological model for West Nile Virus has been already proposed by Wonham *et al.* [10]. But they have neglected the effect of predation (if any) on those migratory birds who are responsible for the spread of WNV. The same problem arises in the case of salmonella bacteria. It is observed that some wild migrating birds were responsible for the spread of these bacteria [11]. There are many other examples, like the highly pathogenic Avian influenza virus that is suspected to have been reintroduced in Japan by some wild migrating birds from South Korea [12]. This is an important problem; unfortunately no real effort has been made so far by researchers to observe the changes made by these migratory populations in the system.

Chatterjee and Chattopadhyay [13] have proposed and analysed a one season model where the prey population migrates from one place to another and carries a disease with itself. They divided the migratory prey population into two groups, namely the susceptible prey and the infective prey. Since, most of the diseases that spread by the migratory birds, like salmonella [14] and WNV [10], are season dependent, they were mainly interested to see the behaviour of the dynamical system for the period when the migratory birds were present in the considered system. Their analytical and numerical results suggested that the introduction of a disease through a migratory population made the system unstable around the interior equilibrium point. They also observed that proper predation might prevent the extinction of the species, supporting some experimental results [15, 16]. Moreover, they proposed to use the predators as a controlling agent for controlling the spread of infection among the prey population. But in the real world it is very difficult to eradicate the disease from the system by predation. The removal experiments will fail if the migratory population is very large, for example, there are over 60 species of birds carrying WNV and it is not possible to eliminate WNV from all these population through predation. This drawback motivates us to modify the model proposed by Chatterjee and Chattopadhyay [13].

The models considered so far in the context of eco-epidemiological problems are mainly either SI (susceptible-infective) type [5] where the infective never recovers from the disease or SIS (susceptible-infective-susceptible) type [13] where the infection does not lead to immunity, so that the infectives become susceptible again after recovery. But it is seen that the migratory birds that have recovered from diseases like salmonella and WNV can develop permanent immunity at least for that particular season, for example North American birds that survive infection develop immunity [17]. When infectives develop a permanent immunity after recovery, they are placed in a separate class and such systems are called SIR systems. In such a system the immune or recovered class is also of great importance and cannot be ignored. So, in this paper we modify the model proposed by Chatterjee and Chattopadhyay [13] by dividing the migratory prey population into three class, namely the susceptible, the infective and the recovered (or immune) classes.

We first try to observe the effect of predation on the migratory birds, as done by Chatterjee and Chattopadhyay [13] in their paper. We find that by controlling the predation of the migratory birds one can make the system disease free. Since the inclusion of the recovery class in our model system is one of the main differences from most of the earlier works, our aim is to observe the role of recovery in such a system.

We have organized the paper as follows. In section 2, we outline the basic mathematical model followed by the proof of the boundedness of the solution of our system. We present the conditions for the existence of equilibria in section 3. The structural stability of the system is discussed in section 4. We perform a numerical analysis in section 5. Section 5 is divided into two subsections, 5.1 and 5.2. We observe the role of predation in 5.1, and in 5.2 we study the role of recovery. In subsection 5.2, we modify our model system by introducing a vaccination term and study the modified system numerically. The paper ends with a discussion in section 6.

2. The basic mathematical model

We have two population:

1. The migratory prey population, which is denoted by N. The prey population N present in our considered system is further divided into three classes: the susceptible population s, the infective population i, which is generated by the susceptible population through horizontal transmission and the recovered population r, the portion of the infective population that recover from the disease and develop immunity against the disease. Therefore, at time t the total migratory prey population is $N(t) = s(t) + i(t) + r(t)$.
2. The predator population is denoted by p.

Before formulating the model equation we make the following assumptions on the predator and the migratory prey populations:

(A1) Here the prey population (in the absence of the disease and the predator) is assumed to be increased by a constant rate A either by birth or migration, as assumed by Chatterjee and Chattopadhyay [13]. Thus,

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

where d is the natural death rate of the migratory prey population.
(A2) The growth rate of the predator population is governed by the migratory prey population along with an alternative source, as assumed by Chatterjee and Chattopadhyay [13]. Depending on that alternative resource, the predator population is assumed to grow in a logistic fashion, with carrying capacity $\frac{e}{f} > 0$ and an intrinsic growth rate constant $e > 0$. Hence in the absence of the migratory prey the growth equation of the predator is given by

$$\frac{dp}{dt} = p(e - fp)$$

(A3) The predation of the infected prey population is included in the predator's growth equation with a negative sign, as assumed by Chatterjee and Chattopadhyay [13].
(A4) Here we have considered the force of infection follows the standard incidence rate $\beta i/Ns$, where β is the average number of adequate contacts of an individual per unit time rather than a simple mass action ηsi as assumed by Chatterjee and

Chattopadhyay [13], where η is a mass action coefficient, which is sometimes used for the horizontal incidence. The parameter η has no direct epidemiological interpretation, but comparing it with the standard formulation shows that $\beta = \eta N$, so that this form implicitly assumes that the contact rate β increases linearly with the population size [18]. But for human disease the contact rate seems to be only very weakly dependent on the population size [18]. Also, there are animal populations like mice in a mouse-room or animals in a herb [19], where the disease transmission primarily occurs locally from nearby animals. This is because in the case of a large population, finite and slow movements of individuals prevents it from making contact with a large number of individuals in unit time like migrating birds. Thus, such a mechanism is better described by $\beta si/N$ than ηsi. (For details, see [2, 20, 21].)

(A5) For mathematical simplicity we assume that the functional response term (prey eaten per predator per unit of time) follows the simple law of mass action.

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

$$\left.\begin{aligned}
\frac{ds}{dt} &= A - \frac{\beta si}{N} - (d + k_1 p)s \\
\frac{di}{dt} &= \frac{\beta si}{N} - k_2 pi - (\gamma + \epsilon + d)i \\
\frac{dr}{dt} &= \gamma i - (d + k_1 p)r \\
\frac{dN}{dt} &= A - k_1(s + r)p - k_2 pi - \epsilon i - dN \\
\frac{dp}{dt} &= p(e - fp + k_1'(s + r) - k_2' i)
\end{aligned}\right\} \tag{1}$$

with $s(0) \geq 0$, $i(0) \geq 0$, $r(0) \geq 0$, $N(0) \geq 0$, $p(0) > 0$.

The above non-negative parameters are defined as

A = the rate of recruitment into the susceptible class for prey population (including newborns and migrating prey population),
d = the per capita natural death rate of the prey populations,
β = the effective per capita contact rate of the infective with other member of the prey population,
ϵ = per capita death rate of the infective prey population due to the disease,
γ = the per capita natural recovery rate of the infective prey population with permanent immunity,
k_1 = the searching efficiency constants or the predation rate of the susceptible (or the recovered) prey populations,
k_2 = the searching efficiency constants or the predation rate of the infective prey populations,
$k_1'(\leq k_1)$ = the conversion factor associated with the predation of the susceptible (or recovered) prey population.
$k_2'(\leq k_2)$ = the conversion factor associated with the predation of the infective prey population.

Here we assume that $k_1 \leq k_2$. This is quite natural, since the infective prey may live in locations that are more accessible to the predator; for example, fish or aquatic snails may live close to the water surface or snails may live on top of vegetation rather than under protective plant cover [22].

Since $N(t) = s(t) + i(t) + r(t)$, so the system (1) becomes:

$$\left.\begin{aligned} \frac{ds}{dt} &= A - \frac{\beta si}{N} - (d + k_1 p)s \\ \frac{di}{dt} &= \frac{\beta si}{N} - k_2 pi - (\gamma + \epsilon + d)i \\ \frac{dr}{dt} &= \gamma i - (d + k_1 p)r \\ \frac{dp}{dt} &= p(e - fp + k_1'(s + r) - k_2' i) \end{aligned}\right\} \tag{2}$$

Boundedness of the solution of the system (2)

Lemma 2.1: *All the solution of* (2) *which initiate in* $\Re^4_{0,+}$ *are uniformly bounded in the region*

$$G = \{(s, i, r, p) \varepsilon \Re^4_{0,+} : W = \frac{l}{\eta} + \theta \text{ for any } \theta > 0 \text{ for all } t \geq T$$

Proof: See appendix A.

3. Equilibrium points and their existence conditions

The equilibria $E_1((A/d), 0, 0, 0)$ and $E_2(\bar{s}, 0, 0, \bar{p})$ where $\bar{s} = A//(d + k_1\bar{p})$ and

$$\bar{p} = \frac{(k_1 e - df) + \sqrt{(k_1 e - df)^2 + 4k_1 f(Ak_1' + de)}}{2k_1 f}$$

of the system (2) always exist.

The equilibrium $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$ exists if $R_0 > 1$, where

$$\hat{s} = \frac{A(\gamma + d)}{d(\beta - \varepsilon)}, \quad \hat{i} = \frac{A(R_0 - 1)}{\beta - \varepsilon}, \quad \hat{r} = \frac{A\gamma(R_0 - 1)}{d(\beta - \varepsilon)},$$

$R_0 = \beta//\gamma'$ and $\gamma' = \gamma + d + \varepsilon$.

To study the existence of interior equilibrium of the system (2), we denote the interior equilibrium point by $E^*(s^*, i^*, r^*, p^*)$. Using $N = s + i + r$ and the last four equations of the system (1) (by setting the time derivatives on the right-hand sides to zero),

we get

$$s^* = \frac{A(\gamma + d + k_1 p^*)}{(d + k_1 p^*)(\beta - \varepsilon - (k_2 - k_1)p^*)},$$

$$i^* = \frac{A(\beta - \gamma' - k_2 p^*)}{(\gamma' + k_2 p^*)(\beta - \varepsilon - (k_2 - k_1)p^*)},$$

$$r^* = \frac{A\gamma(\beta - \gamma' - k_2 p^*)}{(d + k_1 p^*)(\gamma' + k_2 p^*)(\beta - \varepsilon - (k_2 - k_1)p^*)}$$

and p^* is a positive real root of the following equation

$$b_0 p^4 + b_1 p^3 + b_2 p^2 + b_3 p + b_4 = 0, \tag{3}$$

where

$$\begin{aligned}
b_0 &= k_1 k_2 f(k_2 - k_1),\\
b_1 &= f(k_2 - k_1)(k_2 d - k_1(\beta - \gamma')) - \beta f k_1^2 - k_1 k_2(e(k_2 - k_1) - f\varepsilon),\\
b_2 &= (e(k_2 - k_1) - f\varepsilon)(k_1(\beta - \gamma') - k_2 d) + k_1\beta(k_1 e - df) + k_1 k_2(A(k_1' + k_2') - \varepsilon e)\\
&\quad - df(k_2 - k_1)(\beta - \gamma') - k_1 f\beta(\gamma + d)\\
b_3 &= (A(k_1' + k_2') - \varepsilon e)(k_2 d - k_1(\beta - \gamma')) + d(\beta - \gamma')\\
&\quad (e(k_2 - k_1) - f\varepsilon) + \beta(k_1(Ak_1' + de) + (\gamma + d)(k_1 e - df)),\\
b_4 &= \beta(\gamma + d)(Ak_1' + de) - d(\beta - \gamma')(A(k_1' + k_2') - \varepsilon e).
\end{aligned}$$

It can be easily shown that s^*, i^* and r^* are positive if

$$p^* < \gamma'(R_0 - 1)/k_2. \tag{4}$$

It is interesting to note that there is a chance for the existence of multiple interior equilibrium points. But, here we have only studied the dynamics of the system when a unique interior equilibrium point exists. The different possibilities for the existence of a unique interior positive equilibrium are as follows:

Case I: When the number of variations of signs in the sequence of coefficients of equation (3), i.e. $\{b_0, b_1, b_2, b_3, b_4\}$, is exactly one, then by Descartes' rule of signs there exists exactly one positive real root of equation (3) and if that positive root of equation (3) satisfies the inequality (4), then there exists a unique interior equilibrium point $E^*(s^*, i^*, r^*, p^*)$.

Case II: When the number of variations of signs in the sequence of coefficients of equation (3), i.e. $\{b_0, b_1, b_2, b_3, b_4\}$ is more than one, then by Descartes' rule of signs there may exist more than one positive real root of equation (3). Now, if there exists more than one positive root of equation (3) and out of these positive roots, if only one root satisfies the inequality (4), then also there exists a unique interior equilibrium point $E^*(s^*, i^*, r^*, p^*)$.

Case (I) is quite clear. To understand the case (II) in a better way, we give the following example by taking a hypothetical set of parameter values.

Example 3.1: Let us consider the following hypothetical set of parameter values:

$A = 10$ individuals ha^{-1} day^{-1}, $\beta = 2.7$ day^{-1}, $d = 0.0025$ day^{-1}, $k_1 = 0.7$ ha individuals^{-1} day^{-1}, $k_2 = 4.3$ ha individuals^{-1} day^{-1}, $\gamma = 1$ day^{-1}, $\epsilon = 0.002$ day^{-1}, $k_1' = 30\%$ of k_1, $k_2' = 30\%$ of k_2, $f = 70$ ha individuals^{-1} day^{-1}, $e = 2.5\,\text{day}^{-1}$.

For the above parameter values we have $b_0 = 758.52$, $b_1 = -415.656$, $b_2 = -171.306$, $b_3 = -25.842$, and $b_4 = 5.6375$. So, by Descartes' rule of signs there exists either no or two positive real roots of equation (3). Substituting the value of b_i's, $i = 0, 1, 2, 3, 4$ in the equation (3), we get four roots, among which two roots are positive real roots viz., 0.114 and 0.849, and the other two roots are complex conjugate viz., -0.2074 ± 0.1831 i. Out of these two positive real roots only 0.114 satisfies the inequality (4). Substituting the values of the parameter and p^* in the expression of s^*, i^*, r^* we get a unique interior equilibrium point (s^*, i^*, r^*, p^*). Thus, this a typical example for the existence of a unique interior point following case (II).

4. Local stability analysis and persistence result

In this section we shall discuss the stability of different steady states of the model system (2).

Theorem 4.1: *The equilibrium $E_1(A/d, 0, 0, 0)$ is always unstable.*

Proof: See appendix B.

Theorem 4.2: *If $R_0 \leq 1 + k_2\bar{p}/\gamma'$, where*

$$\bar{p} = \frac{(k_1 e - df) + \sqrt{(k_1 e - df)^2 + 4k_1 f(Ak_1' + de)}}{2k_1 f},$$

then $E_2(\bar{s}, 0, 0, \bar{p})$ is locally asymptotically stable (LAS).

Proof: See appendix B.

Theorem 4.3: *The equilibrium point $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$ is LAS if and only if the following conditions hold*

(i) $R_0 > \dfrac{d(e\beta - e\epsilon + k_1'A + k_2'A)}{(A(k_2'd - k_1'\gamma))}$

(ii) $\gamma(\beta + \gamma + d)R_0^2 - 2\beta\gamma R_0 - \beta d > 0$

Proof: See appendix B.

Now in order to observe the structural stability of our model system around the positive steady state, we shall prove the persistence of the system. If the following three conditions hold then the system is persistent [23]:

(i) the system should be bounded,
(ii) the boundary equilibria of that system should be repellers, and
(iii) there should not exist any periodic solution around the positive steady state of that system.

We have already proved that system (2) is bounded. We will now show that the boundary equilibria are repellers under certain conditions.

Theorem 4.4: *If the following conditions holds, then all the boundary equilibria of the system* (2) *are repeller.*

$$(1)\quad R_0 > 1 + k_2\bar{p}/\gamma', \quad \text{where } \bar{p} = \frac{(k_1 e - df) + \sqrt{(k_1 e - df)^2 + 4k_1 f(Ak_1' + de)}}{2k_1 f}$$

$$(2)\quad R_0 < \frac{d(e\beta - e\epsilon + k_1' A + k_2' A)}{(A(k_2' d - k_1'\gamma))}$$

or, $\gamma(\beta+\gamma+d)R_0^2 - 2\beta\gamma R_0 - \beta d < 0$ *or both.*

Proof: See appendix B.

Next we shall find the conditions for non-existence of the periodic solutions around the interior equilibrium point E^*.

Here we shall apply the Li–Muldowney's criteria (for details see appendix B) for the non-existence of the periodic solutions of system. The logarithmic norm μ_∞, endowed by the norm $|X|_\infty$ of the second additive compound matrix $J^{[2]}$, associated with the jacobian matrix J, computed on E^*, is negative if and only if the suprema of the following satisfy

$$\frac{-i^*}{N^*}\{\beta - 2(\gamma' + k_2 p^*)\} - (d + k_1 p^*) + k_1 s^* + k_2 i^* < 0 \tag{5}$$

$$\frac{-\beta i^*}{N^*} - 2(d + k_1 p^*) + (\gamma + \gamma' + k_2 p^*) + k_1(s^* + r^*) < 0 \tag{6}$$

$$\frac{-i^*}{N^*}(\beta - \gamma' - k_2 p^*) - (d + k_1 p^*) + \gamma' + k_2 p^* + p^*(k_1' + k_2' - f) < 0 \tag{7}$$

$$\frac{i^*}{N^*}\{\beta - 2(\gamma' + k_2 p^*)\} - (d + k_1 p^*) + k_1 r^* + k_2 i^* < 0 \tag{8}$$

$$\frac{i^*}{N^*}(\beta - \gamma' - k_2 p^*) - p^*(f - 2k_1') < 0 \tag{9}$$

$$(\gamma - d) + p^*\{k_2' - (k_1 - k_1') - f\} < 0 \tag{10}$$

A direct application of the Li–Muldowney method shows that if the above condition is satisfied, then there exists no periodic solution around E^* for the system (2).

5. Numerical analysis

We know that the contact rate β, the predation rates k_1 and k_2 play important roles in the dynamics of a predator–prey system with infection in the prey [22, 24]. But in our model system recovery rate also plays an important role, since the inclusion of the recovery class in our model is a vital modification of most of the earlier models. So, we have first focused our study on observing the role of the contact and predation rates on the dynamics of the system (see, section 5.1). Then, in section 5.2, we have tried to find the role of recovery in our model system.

5.1. *Role of the contact and predation rates*

We will focus our study around the unique positive steady state (s^*, i^*, r^*, p^*) obtained from the set of parameter values discussed in Example 3.1 of section 3.

We here assume the initial values of the susceptible, infective, recovered prey population and their predator population to be 30 individuals ha^{-1}, 5 individuals ha^{-1}, 2 individuals ha^{-1} and 10 individuals ha^{-1} respectively. The initial value of all the population is kept fixed through out the entire numerical section. For the above set of parameter values, the eigenvalues associated with the variational matrix computed around the positive steady state are -8.03, -0.38, -0.0114, -0.295. This shows that the above unique interior equilibrium point is locally asymptotically stable.

With this set of parameter values and the initial values, we get figure 1.

Figure 1 shows that for the considered hypothetical set of parameter values, the unique positive steady state is locally asymptotically stable.

Now we will try to observe the role of the above mentioned three key parameters β, k_1, and k_2 one by one. We begin with the parameter k_2, the predation rate of the infective prey population. By increasing the value of k_2 from 4.3 to 4.4 and retaining the values of the other parameters fixed, we get figure 2.

To understand the change in the solution of the system due to the change in the predation rate, we present in figures 3 and 4 the four dimensional phase diagram projected in the three dimensional SIP axes.

From the above numerical simulation it is clear that to keep the system stable around the interior equilibrium point we shall have to keep the predation rate of the infected prey population k_2 under a certain threshold value, namely (k_2^c).

Next we observe the role of the other two key parameters k_1 and β. For this we begin with the set of parameter values for which we obtained figure 2 and then tried to bring back stability in the system around the positive equilibrium point by changing the value of k_1 and β. We start with the parameter k_1 (the rate of predation of the susceptible and the recovered prey population). By slightly increasing the value of k_1 from 0.7 to 0.8 and retaining the other parameters the same as in figure 4, we get figure 5.

Next we have tried to find the role of the parameter β. Decreasing the value of β from 2.7 to 2.6 and retaining the other parameter values as fixed as in figure 2, we get figure 6.

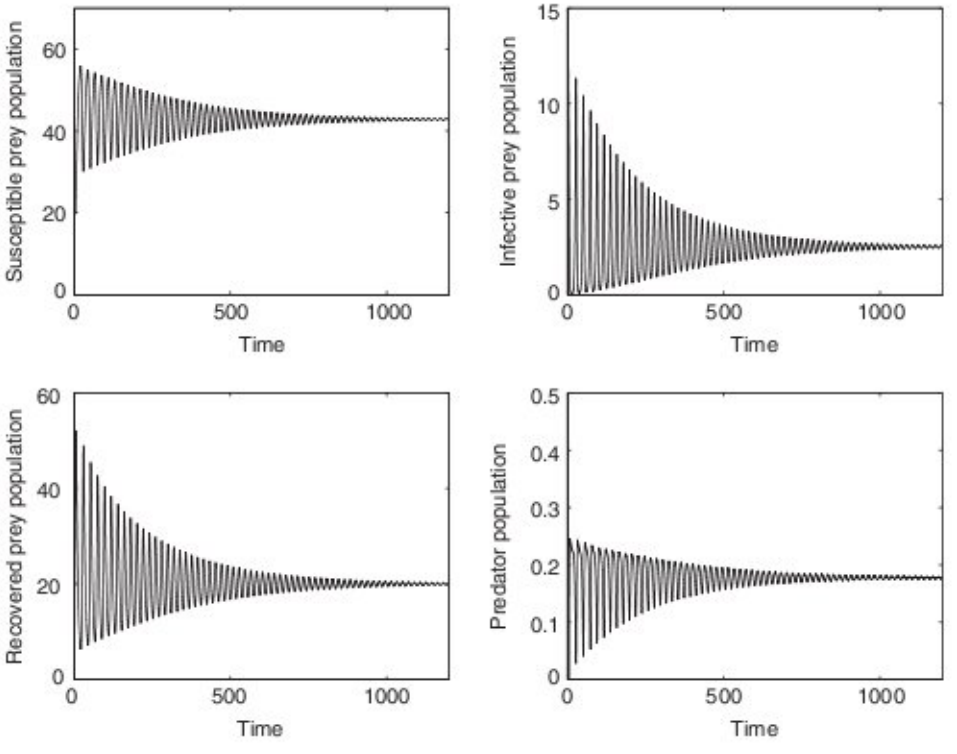

Figure 1. The different population distribution showing coexistence of all four populations.

Thus it is observed that either by keeping the value of the parameter k_1 (the predation rate of the susceptible and recovered prey) above a certain critical value (k_1^c), or by keeping the value of the parameter β (the contact rate) below a certain critical value (β^c) we can bring back the stability in our system around the positive equilibrium point from the situation shown in figure 2. Thus, these three key parameter equally play an important role in maintaining the stability of the system around the interior equilibrium point. Moreover, to understand the role of the predator in making the system disease free we have constructed table 1. But, before this we would like to make remark (5.1.1).

Remark 5.1.1: It should be noted that our system (2) reduces to a simple SIR model, for $p = 0$. The system in the absence of the predator admits two equilibrium points, viz. one is the axial $E_{0p}(A/d, 0, 0)$ and another is interior equilibrium point $E_{1p}(s', i', r')$, where,

$$s' \equiv \frac{A(d+\gamma)}{d(\beta-\epsilon)}, \quad i' \equiv \frac{A(\beta-\gamma')}{\gamma'(\beta-\epsilon)}, \quad r' \equiv \frac{A\gamma(\beta-\gamma')}{d\gamma'(\beta-\epsilon)}.$$

The axial equilibrium exists for any set of parametric values, but the interior equilibrium exists if and only if $R_0 > 1$. It can be seen here that the existence of

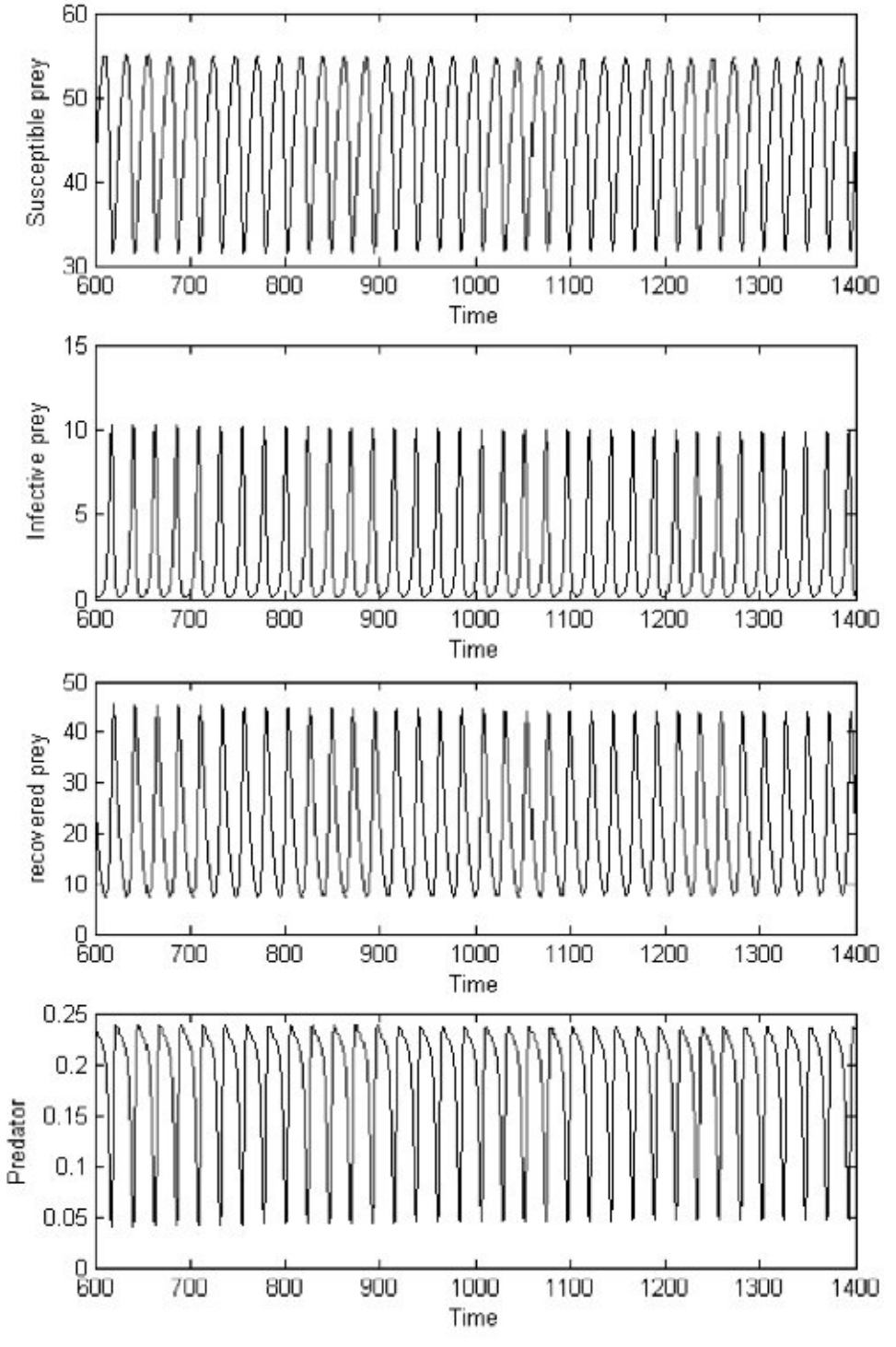

Figure 2. The different population components for the parametric value of $k_2 = 4.4$.

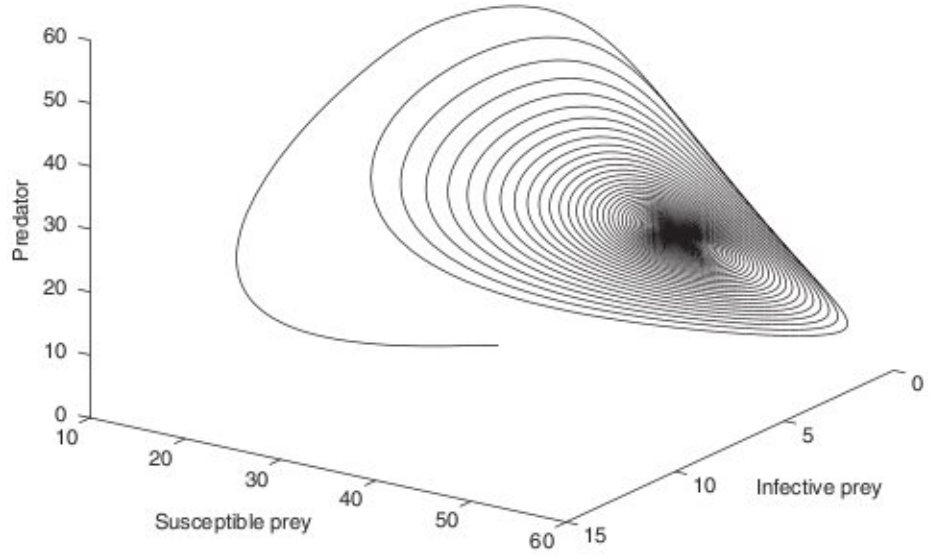

Figure 3. The projection of the phase diagram in the SIP plane when $k_2 = 4.3$.

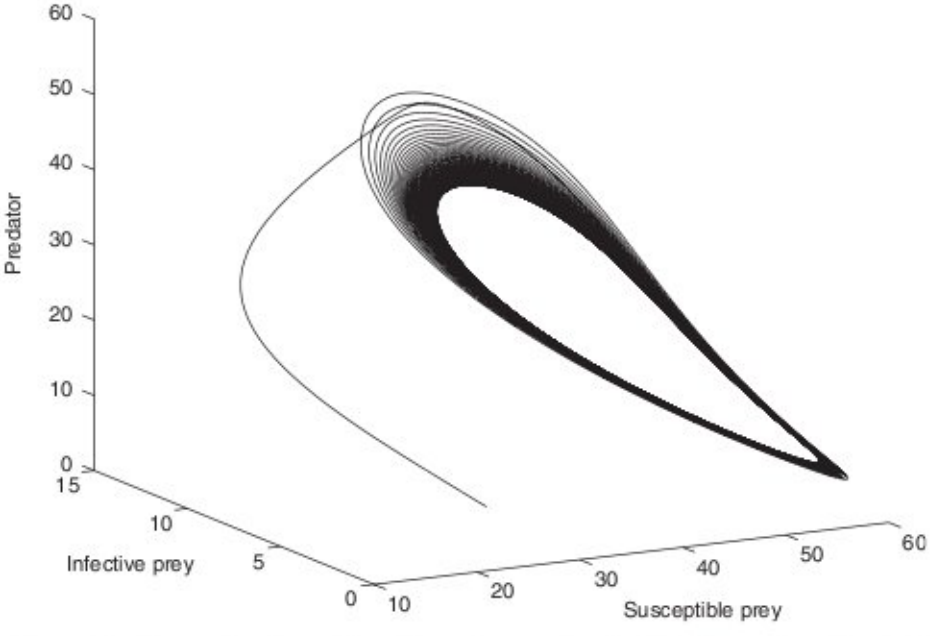

Figure 4. The projection of the phase diagram in the SIP plane when $k_2 = 4.4$.

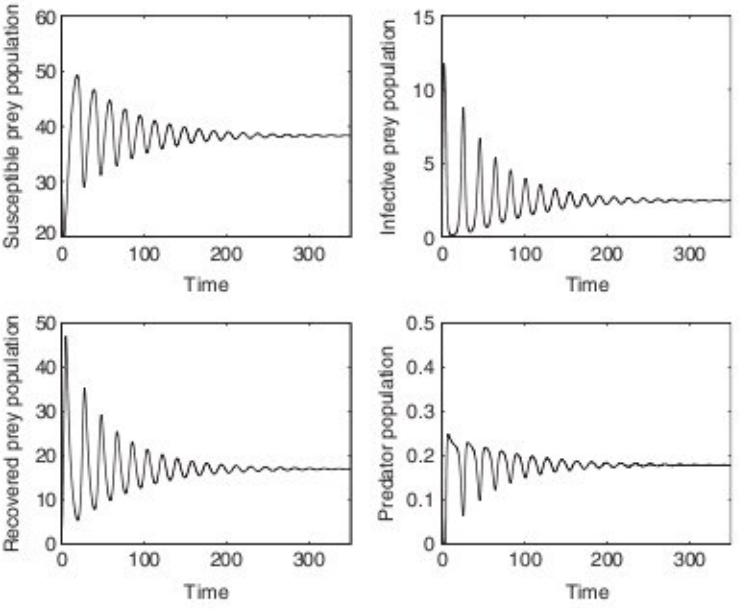

Figure 5. The stable population distribution around the interior equilibrium point for $k_1 = 0.8$.

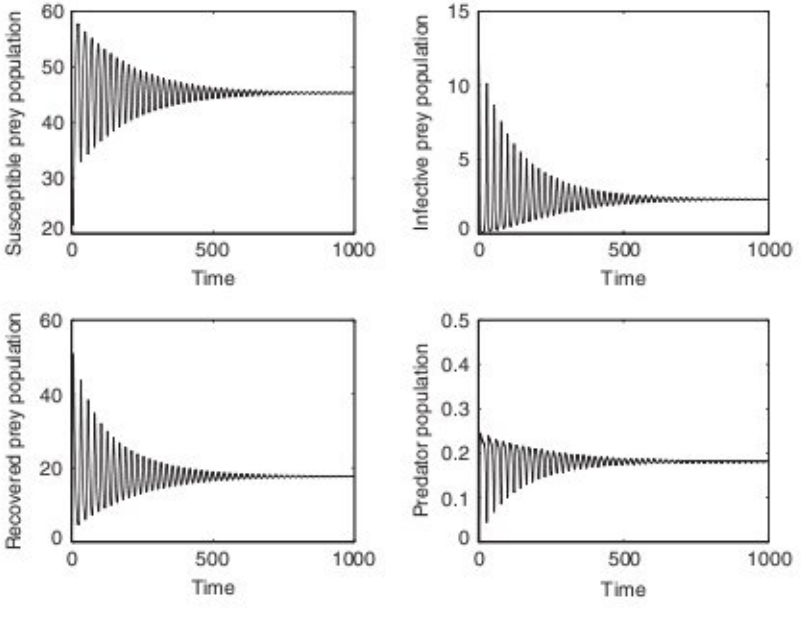

Figure 6. The stable population distribution around the interior equilibrium point for $\beta = 2.6$.

the interior equilibrium point ensures the instability of the axial equilibrium point for the system. It can also be seen that the system is locally asymptotically stable around the interior point (s', i', r') if and only if

$$R_0 > \frac{1+\epsilon+\sqrt{(1+\epsilon)^2+4\gamma d}}{2\gamma}.$$

Table 1 shows the model system (2) both in the absence and presence of the predator.

From table 1 we observe that in the absence of the predator population the system remains disease free for $R_0 \leq 1.0047$, while in the presence of the predator population the system remains disease free for $R_0 \leq 1.923$. So, predation plays an important role in keeping the system disease free for a wider range of R_0. Next we have studied the role of the recovery in the system.

5.2. *Role of the recovery*

It is interesting to observe that in the absence of the recovery (i.e., $\gamma = 0$), the predator population goes to extinction for the same set of parameter values as considered in the table 1 with $\beta = 2.7 (R_0 \approx 2.6)$, but one can see from table 1 that for this value of R_0 all the population coexists both in the presence as well as in the absence of the predator population. We also observe that the population of the infective prey increases from five individuals ha^{-1} to 191.1179 individuals ha^{-1}, while the population of the susceptible prey decreases from 30 individuals ha^{-1} to 3.79 individuals ha^{-1}. So, one can see that not only the predator population goes to extinction, but also there is a huge increase in the infective prey population. So, the recovery rate plays an important role in avoiding the outbreak of the disease. This is clearer from table 2.

We observe from table 2 that if the natural recovery rate γ is kept above a certain critical value ($\gamma^c = 0.91$) then all the populations coexist, below which the predator population goes to extinction. Figures 7 and 8 present two phase diagrams, by projecting the four dimensional phase diagram in the three dimensional SIR axes to understand the role of the recovery rate in a better way.

In both these figures we observe that all the populations coexist (as stated in table 2), but E^* becomes stable when $\gamma \geq 0.96$. This result shows that the recovery enhances the persistence of the species, which is quite natural. But it is not possible for us to control the natural recovery rate. However we can increase the immunity among the migratory birds by giving them vaccines. To see whether the application of the vaccination programmes will really help us to control the disease or not, we modify our model system (2) by introducing a vaccination term. Though the vaccine will be given to all classes of the migratory birds, it will affect only the susceptible class, so the vaccine term is added in the susceptible class only [25]. We assume that the susceptible population is vaccinated at a constant rate $\mu\,day^{-1}$. We also assume that the vaccine used is 100%

Table 1. Simulation experiments of model system (2) with the fixed parameter values: $A = 10$, $d = 0.0025$, $\gamma = 1$, $\epsilon = 0.002$, $f = 70$, $e = 2.5$ $k_1 = 0.7$, $k_2 = 4.25$, $k_1' = 0.3 \times k_1$, $k_2' = 0.3 \times k_2$.

Parameters kept fixed	Parameter varied	Ranges in which R_0 was varied due to the variation in β	Behaviour of the populations in the presence of the predator population	Behaviour of the populations in the absence of the predator population
$A, d, k_1, k_2, \gamma, \varepsilon, k_1', k_2', f, e$	β	$R_0 \leq 1$	Only i & r goes to extinction	Only i & r goes to extinction
		$1 < R_0 \leq 1.0047$	Only i & r goes to extinction	Only i & r goes to extinction
		$1.0048 \leq R_0 \leq 1.923$	Only i & r goes to extinction	All the populations coexists
		$1.924 \leq R_0 \leq 2.8119$	All the populations coexists	All the populations coexists

Table 2. Simulation experiments of model system (2) with the same set of fixed parameter values as taken in the table 1 with $\beta = 2.7$ except the parameter γ.

Parameter varied	Ranges in which the parameter γ was varied	Behaviour of the population of the system
γ	$0 \leq \gamma \leq 0.91$	Only p goes extinction
	$0.92 \leq \gamma \leq 1$	All the populations Coexists

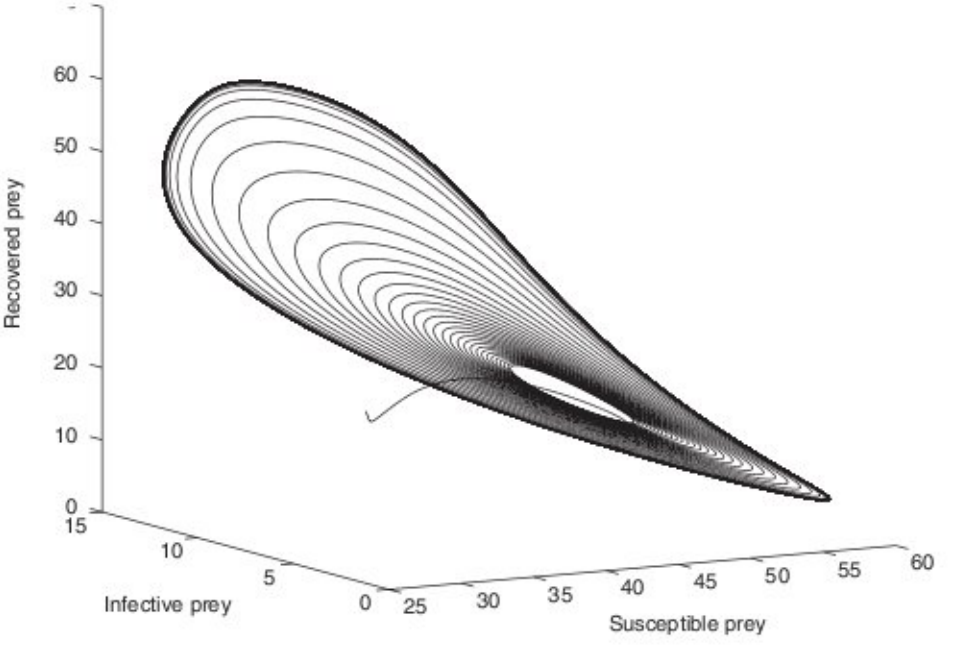

Figure 7. The projection of the phase diagram in the SIR plane when $\gamma = 0.96$.

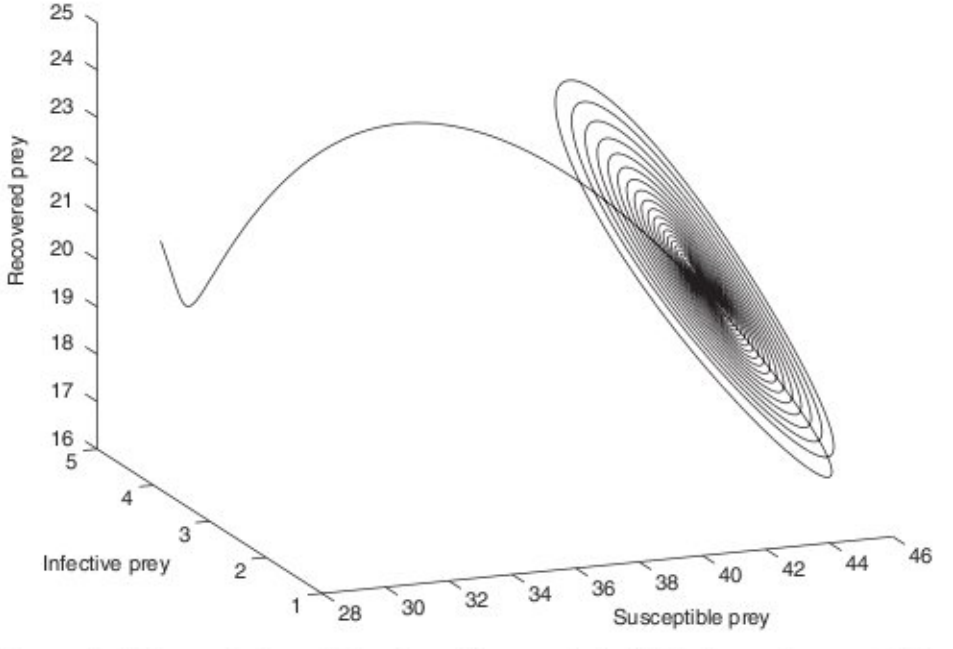

Figure 8. The projection of the phase diagram in the SIR plane when $\gamma = 0.97$.

effective in reducing the infection. With this assumptions the model system (2) takes the following form:

$$\left.\begin{aligned}\frac{ds}{dt} &= A - \frac{\beta si}{N} - (d + k_1 p)s - \mu s \\ \frac{di}{dt} &= \frac{\beta si}{N} - k_2 pi - (\gamma + \epsilon + d)i \\ \frac{dr}{dt} &= \gamma i + \mu s - (d + k_1 p)r \\ \frac{dp}{dt} &= p(e - fp + k_1'(s + r) - k_2' i)\end{aligned}\right\} \tag{11}$$

To understand the role of the vaccination, see table 3. We have taken the same set of parameter values as considered in table 2 except γ. To study the effect of the vaccination we have taken a value of γ below γ^c so that in the absence of the vaccination programme the predator population will extinct from the system. Hence, we choose $\gamma = 0.3$, see table 2.

We observe from table 3 that for a fixed value of the recovery rate (here for $\gamma = 0.3$), if the rate of vaccination is kept above a certain lower critical value ($\mu_1^c = 0.009$), then we can prevent the predator population. Moreover, the vaccination programme can even make the system disease free provided that the rate of vaccination is increased above a certain upper critical value ($\mu_2^c = 0.19$).

6. Discussion

The effect of diseases in an ecological system is an important issue from a mathematical and experimental point of view. This becomes more important and difficult to control if the disease is spread by migratory birds. In this paper we have modified

Table 3. Simulation experiments of model system (11) with the same set of parameter values as taken in the table 1 except the parameter γ. Here the value of γ was kept fixed at 0.3.

Parameter Varied	Ranges in which the parameter μ was varied	Behaviour of the population of the system
μ	$0 \le \mu \le 0.008$	Only p goes to extinction
	$0.009 \le \mu \le 0.18$	All the populations coexists
	$0.19 \le \mu \le 0.8$	Only i goes to extinction

the model proposed by Chatterjee and Chattopadhyay [13] by adding a recovered class.

Here we have tried to observe the dynamical behaviour of an ecosystem where the disease is introduced by some migratory bird population. The main objective of this paper was to find the role of the recovery with permanent immunity in controlling the outbreak of such disease. We have first showed that all the solutions which initiate in $\Re_{0,+^4}$ are uniformly bounded. The system admits three boundary equilibria and one interior equilibrium under suitable parametric conditions. We have observed that the equilibrium point $E_1(A/d, 0, 0, 0)$ is an unstable saddle with respect to all perturbations. The disease free equilibrium $E_2(\bar{s}, 0, 0, \bar{p})$, which always exists, is stable if $R_0 \leq 1 + k_2\bar{p}/\gamma'$, where

$$\bar{p} = \frac{(k_1 e - df) + \sqrt{(k_1 e - df)^2 + 4k_1 f(Ak_1' + de)}}{2k_1 f}.$$

The equilibrium point $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$, which exists if $R_0 > 1$, and is stable if

$$R_0 > \frac{d(e\beta - e\epsilon + k_1'A + k_2'A)}{(A(k_2'd - k_1'\gamma))}$$

and $\gamma(\beta + \gamma + d)R_0^2 - 2\beta\gamma R_0 - \beta d > 0$, where $R_0(= \beta/(\gamma + d + \epsilon))$ is known as the basic reproductive number. We have also shown two different ways for which a unique interior equilibrium exists $E^*(s^*, i^*, r^*, p^*)$. We have also found conditions for the persistence of the system around the positive steady state. Since the structure of the model presented here is a complex one so the nature of the dynamics of the model system (2) around the positive equilibrium point is mainly studied numerically.

We have divided our numerical section into two subsections. In the first section i.e., section 5.1 we have tried to observe the role of predation. In the second section i.e., section 5.2 we have tried to observe the role of recovery. We begun our numerical analysis by taking a set of parameters for which a unique positive equilibrium point exists. We found that to keep the system stable around the interior equilibrium point under some fixed parameter values we shall have to either keep the contact rate below a certain threshold value β^c, or the predation rate of the infective prey below a certain critical value k_2^c, or the predation rate of the susceptible (or the recover prey) above a certain critical value k_1^c. From table 1, we observe that for both in the absence as well as in the presence of the predator population the system remains disease free for $R_0 \leq 1$, which also validates our analytical findings. But the interesting result is observed for $R_0 \geq 1$. In the absence of the predator population the disease is absent from the system for $R_0 \leq 1.0047$, while in the presence of the predator population the same is true for $R_0 \leq 1.923$. Thus, it is observed that in the presence of the predator population the system remains disease free for a wider range of R_0. Hence, it can be concluded that the predator population plays an important role in controlling the disease, as claimed by Chatterjee and Chattopadhyay [13]. But, the method of making the system disease free by using the predator as a controlling agent has a disadvantage. The removal experiment will

fail if the migratory population is very large in comparison to the predator population. Thus, we have tried to suggest an alternative way to control the outbreak of the disease. In doing so we found that recovery with permanent immunity plays that alternative way.

It was observed from our numerical result (see, section 5.2) that in the absence of the recovery (i.e., when $\gamma = 0$), the predator population goes to extinction for the same set of fixed parameter values for which all the populations coexist in the presence of the recovery rate (i.e., $\gamma \neq 0$). It was also observed that in the absence of the recovery the population of the infective prey increases from 5 individuals ha^{-1} to 191.1179 individuals ha^{-1}, while the population of the susceptible prey decreases from 30 individuals ha^{-1} to 3.79 individuals ha^{-1}. So, in the absence of the recovery rate, not only the predator population goes to extinction but also there is a huge increase in the infective prey population. Hence, the natural recovery plays an important role in avoiding the outbreak of the disease. Moreover, from table 2 it was observed that, to prevent the predator population from extinction and for the coexistence of all the populations, we have to keep the recovery rate above a certain threshold value (γ^c). Thus, the increase in recovery rate enhances the persistence of the system. But it is not possible to control the natural recovery rate. So, what we can do is increase the immunity of the migratory birds by giving vaccines. Keeping this in mind we modified our model system (2). Then through our numerical simulation, see table 3, we found that if the vaccination rate is kept above a certain upper critical value (μ_2^c) then the vaccination programme can make the system disease free, provided that the vaccine is 100% effective. To make the vaccination programme effective, it must be kept at least above a certain lower critical value (μ_1^c), otherwise the predator population will be extinct from the system.

Hence, we finally conclude that the use of a predator as a controlling agent is a very difficult method to apply in real world situations especially if the prey population is migratory in nature (though it seems to work in an experimental environment [24]). But if we can give a vaccine (which is 100% effective) to the migratory birds with a rate above a critical value, then the use of a predator as a controlling agent will be meaningful and applicable to the real world situations. Before ending our article, we would like to mention that the proposed model system (11) needs modification and further in depth analysis. We are leaving this study for our future work. Moreover, the above article rests on the assumption that the environmental parameters involved with the model system are all constants irrespective to time and environmental fluctuations. But, in reality all such parameters exhibit random variations to a greater or lesser extent [26]. Thus, the model taken in the article may sometime give incorrect results if the densities of the populations involved are low and the fluctuations play a dominant role. In our next study we shall take into account the environmental effects on the system.

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Appendix A

Proof (Lemma 2.1): We define a function

$$W = s + i + r + p \tag{A.1}$$

The time-derivative of (A.1) along the solutions of (2) is

$$\begin{aligned}\frac{dW}{dt} &= A - d(s+i+r) - \epsilon i - k_1(s+r)p - k_2 ip + p(e-fp) + k_1'(s+r)p - k_2' pi \\ &\leq A - d(s+i+r) + p(e-fp), \quad (\text{since, } k_1' \leq k_1).\end{aligned}$$

Now if we take $\eta > o$ such that $\eta \leq d$, then

$$\begin{aligned}\frac{dW}{dt} &\leq A - \eta(s+i+r+p) + p(e-fp+\eta) \\ &\leq A - \eta W + \frac{(e+\eta)^2}{4f} \\ \frac{dW}{dt} + \eta W &\leq A + \frac{(e+\eta)^2}{4f}.\end{aligned}$$

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

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

Applying the theory of differential inequalies [27], we obtain

$$0 < W(s,i,r,p) < \frac{l}{\eta}(1 - e^{-\eta t}) + W(s(0), i(0), r(0), p(0))e^{-\eta t}.$$

For $t \to \infty$, we have $0 < W < l/\eta$. Hence, all the solution $(s(t), i(t), r(0), p(t))$ of (2) that initiate at $(s(0), i(0), r(0), p(0)) \varepsilon \Re^4_{0,+}$ are confined in the region

$$G = \{(s,i,r,p)\varepsilon \Re^4_{0,+} : W = \frac{l}{\eta} + \theta$$

for any $\theta > 0\}$ for all $t \geq T$, where T depends on the initial values $(s(0), i(0), r(0), p(0))$.

Therefore, the set G is an invariant set which contains the Ω-limit set of all the paths of the system (2) that initiate in the positive octant.

Appendix B

We shall first state a lemma relating to the characteristic polynomial of a matrix [28] which we have used to prove the theorems of section (4).

Lemma 4.1: *Let M be an $n \times n$ matrix which is symmetrically partitioned into upper or lower triangular block matrices labelled*

$$M = \begin{bmatrix} M_1 & M_2 \\ 0 & M_3 \end{bmatrix}$$

or

$$M = \begin{bmatrix} M_1 & 0 \\ M_2 & M_3 \end{bmatrix}$$

Then the characteristic polynomial of the matrix M is equal to the product of the characteristic polynomials of M_1 and M_3.

Using the above lemma we shall prove the theorems given in section 4. To begin our proof we have to first find the variational matrix of the system (2). The variational matrix J of the system (2) is:

$$J = \begin{bmatrix} -\frac{\beta\bar{i}(i+r)}{N^2} - d - k_1 p & -\frac{\beta s(s+r)}{N^2} & \frac{\beta s i}{N^2} & -k_1 s \\ \frac{\beta\bar{i}(i+r)}{N^2} & \frac{\beta s(s+r)}{N^2} - k_2 p - \gamma - \epsilon - d & -\frac{\beta s i}{N^2} & -k_2 i \\ 0 & \gamma & -d - k_1 p & -k_1 r \\ p k_1' & -p k_2' & p k_1' & e - 2fp + k_1'(s+r) - k_2' i \end{bmatrix}.$$

Now we shall prove the theorems one by one.

Proof (Theorem 4.1): After computing the variational matrix J_1 associated with $E_1(A/d, 0, 0, 0)$, we find the following eigenvalues: $-d$, $-d$, $\beta - (\gamma + \epsilon + d)$, $(ed + k_1' A)/d$. Since $(ed + k_1' A)/d > 0$, so E_1 is always unstable.

Proof (Theorem 4.2): The variational matrix associated with $E_2(\bar{s}, 0, 0, \bar{p})$ is given below

$$J_2 = \begin{bmatrix} -(d + k_1\bar{p}) & -\beta & 0 & -k_1\bar{s} \\ 0 & \beta - (\gamma + \epsilon + d) - k_2\bar{p} & 0 & 0 \\ 0 & \gamma & -(d + k_1\bar{p}) & 0 \\ k_1'\bar{p} & -k_2'\bar{p} & k_1'\bar{p} & -f\bar{p} \end{bmatrix}.$$

After applying congruence ope ations on the above matrix, it reduces to the following matrix

$$J_2' = \begin{bmatrix} -(d+k_1\bar{p}) & -\beta & 0 & 0 \\ 0 & \beta-(\gamma+\epsilon+d)-k_2\bar{p} & 0 & 0 \\ 0 & \gamma & -(d+k_1\bar{p}) & 0 \\ k_1'\bar{p}+\dfrac{k_1A}{d+k_1\bar{p}} & -k_2'\bar{p}+\dfrac{\beta k_1A}{d+k_1\bar{p}} & k_1'\bar{p} & \dfrac{-k_1k_1'A\bar{p}}{d+k_1\bar{p}}-f\bar{p} \end{bmatrix}.$$

Thus, the eigenvalues of the variational matrix associated with $E_2(\bar{s},0,0,\bar{p})$ are given by $-(d+k_1\bar{p})$, $-(d+k_1\bar{p})$, $\beta-(\gamma+\epsilon+d)-k_2\bar{p}$, $-((k_1k_1'A\bar{p})/(d+k_1\bar{p}+f\bar{p}))$ (using Theorem 4.1).

It can be easily seen that if $R_0 \le 1+k_2\bar{p}/\gamma'$, then $E_2(\bar{s},0,0,\bar{p})$ is LAS.

Proof (Theorem 4.3): The variational matrix associated with $E_3(\hat{s},\hat{i},\hat{r},0)$ is given below

$$J_3 = \begin{bmatrix} -\dfrac{\beta\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}-d & \dfrac{\beta\hat{s}(\hat{s}+\hat{r})}{\hat{N}^2} & \dfrac{\beta\hat{s}\hat{i}}{\hat{N}^2} & -k_1\bar{s} \\ \dfrac{\beta\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2} & \dfrac{\beta\hat{s}(\hat{s}+\hat{r})}{\hat{N}^2}-\gamma' & -\dfrac{\beta\hat{s}\hat{i}}{\hat{N}^2} & -k_2\hat{i} \\ 0 & \gamma & -d & -k_1\hat{r} \\ 0 & 0 & 0 & e+k_1'(\hat{s}+\hat{r})-k_2'\hat{i} \end{bmatrix}.$$

After applying congruence operations on the above matrix, it reduces to the following matrix

$$J_3' = \begin{bmatrix} -\dfrac{\beta\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}-d & -\dfrac{\beta\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}-\dfrac{\beta\hat{s}(\hat{s}+\hat{r})}{\hat{N}^2}-d & \dfrac{\beta\hat{s}\hat{i}}{\hat{N}^2} & -k_1\hat{s} \\ -d & -(\gamma+\epsilon+2d) & -\gamma & -(k_1\hat{s}+-k_2\hat{i})-\dfrac{\gamma-k_1\hat{r}}{d} \\ 0 & 0 & -d & -k_1\hat{r} \\ 0 & 0 & 0 & e+k_1'(\hat{s}+\hat{r})-k_2'\hat{i} \end{bmatrix}.$$

Hence, the eigenvalues of the variational matrix associated with $E_3(\hat{s},\hat{i},\hat{r},0)$ are given by $e+k_1'(\hat{s}+\hat{r})-k_2'\hat{i}$, $-d$ and the roots of the following equation

$$x^2+\left\{\gamma'+2d+\frac{\beta\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}\right\}x+\frac{\beta\gamma\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}+d\gamma-\frac{d\beta\hat{s}(\hat{s}+\hat{r})}{\hat{N}^2}+\frac{\gamma\beta\hat{s}\hat{i}}{\hat{N}^2}=0 \qquad \text{(B.1)}$$

If $e+k_1'(\hat{s}+\hat{r})-k_2'\hat{i}<0$ and the roots of the equation (6) have negative real parts, i.e., if

$$\frac{\beta\gamma\hat{i}(\hat{i}+\hat{r})}{\hat{N}^2}+d\gamma-\frac{d\beta\hat{s}(\hat{s}+\hat{r})}{\hat{N}^2}+\frac{\gamma\beta\hat{s}\hat{i}}{\hat{N}^2}>0$$

then $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$ is LAS. Substituting the values of $\hat{s}$, $\hat{i}$ and $\hat{r}$ in these expressions and after some algebraic calculation we get the required conditions as stated in the theorem.

Proof (Theorem 4.4): We have already proved in Theorem 4.1 that $E_1(A/d, 0, 0, 0)$ is always a repeller. Next, we observe from Theorem 4.2 that if $R_0 > 1 + k_2\bar{p}/\gamma'$, then $E_2(\bar{s}, 0, 0, \bar{p})$ becomes a repeller. Finally, from the eigenvalues of the variational matrix associated with $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$ we observe that if either

$$R_0 < \frac{d(e\beta - e\epsilon + k_1'A + k_2'A)}{(A(k_2'd - k_1'\gamma))},$$

or $\gamma(\beta + \gamma + d){R_0}^2 - 2\beta\gamma R_0 - \beta d < 0$, or both are satisfied then $E_3(\hat{s}, \hat{i}, \hat{r}, 0)$ also becomes a repeller. Hence Theorem 4.4.

Li–Muldowney criteria [29]: Consider the general autonomous ordinary differential equation

$$\frac{dX}{dt} = F(X(t)) \tag{B.2}$$

where F is a C^1 function in some open subset of R^n with values in R^n. Denote by $J^{[2]}$ the second additive compound matrix associated with the Jacobian matrix J (see [23] for details), and recall that if $X \in R^n$ then the corresponding logarithmic norm of $J^{[2]}$ (that we denote by $\mu_\infty(J^{[2]})$) endowed by the vector norm $|X|_\infty = \sup_i |X_i|$ is

$$\mu_\infty(J^{[2]}) = \sup\left\{\frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{j \neq r,s}\left(\left|\frac{\partial F_r}{\partial x_j}\right| + \left|\frac{\partial F_s}{\partial x_j}\right|\right): 1 \leq r < s \leq n\right\}$$

where $\mu_\infty(J^{[2]}) < 0$ implies the diagonal dominance by row matrix $J^{[2]}$. Then, the following holds.

Lemma 4.2: *A simple closed rectifiable curve that is invariant with respect to the system* (2) *cannot exist if* $\mu_\infty(J^{[2]}) < 0$.

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