

## ROLE OF MIGRATORY BIRDS UNDER ENVIRONMENTAL FLUCTUATION — A MATHEMATICAL STUDY

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Chatterjee and Chattopadhyay<sup>1</sup> proposed and analyzed a simple one season eco-epidemiological model to understand the role of migratory birds in an eco-epidemiological scenario. In this paper, we have modified their model taking into account standard incidence as horizontal incidence. We have studied the proposed model both in the absence and in the presence of environmental disturbances. From our analysis, we observe that in the absence of the environmental fluctuations, the disease spread by migratory birds may be controlled by either decreasing the contact rate, or increasing the recovery or predation rates. Further, we also observe that if the artificial eutrophication (or intensity of white noise) is kept under a certain threshold value, the control procedure proposed in the deterministic case is also valid in the presence of environmental disturbances.

*Keywords:* Eco-epidemiological System; Migratory Birds; Standard Incidence Rate; Stochastic Perturbations; Control.

### 1. Introduction

One of the unsolved problems in today's world is the emerging infectious diseases that are spread by the migratory birds. But unfortunately no real efforts have been made so far by the researchers to study this. This problem needs special attention as the migratory birds are mainly responsible for the introduction of a new disease to a new place, and also responsible for the re-introduction of a disease to a place that was totally washed away from that place. For example, the 1962 epidemic of Eastern equine encephalomyelitis (EEE) in Jamaica resulted from transport of the virus by birds from the continental United States.<sup>2</sup> In another example, the West Nile virus (WNV) is introduced to the Middle East by migrating white storks.<sup>3</sup> It is observed that a predator becomes infected by predation of a prey infected by WNV.<sup>4</sup> The same problem arises in the case of Salmonella bacteria. It was observed that some wild migrating birds are responsible for the spread of this bacteria.<sup>5</sup> In

another example, the highly pathogenic avian influenza virus which is suspected to be re-introduced into Japan by some wild migrating birds from South Korea.<sup>6</sup>

Chatterjee and Chattopadhyay<sup>1</sup> proposed and analyzed a predator-prey model, where the prey population migrates and carries disease from one place to another. They divided the migratory prey population into two groups, namely the susceptible and the infective prey. Since, most of the diseases that are spread by the migratory birds, like Salmonella,<sup>7,8</sup> are season dependent, so their model is a one-season model. Their analytical and numerical results showed that the introduction of a disease through migratory population destabilizes the otherwise stable system around the co-existence steady state. They also observed that proper predation may be used as a suitable control measure for preventing the extinction of the species. Their findings are in accordance with some earlier experimental results.<sup>9,10</sup>

But in their paper they have ignored the following two important points:

- (1) First they assumed that the transmission of the disease follows simple mass action incidence. It is seen that in the case of constant total population, if the disease is not fatal and the model does not address vital dynamics (the normal birth and death dynamics), then the infection term  $bxy$  may be justified (since  $\frac{b}{x+y}$  is now a constant). Then the meaning of  $b$  becomes the encounter infection rate. But, for large populations like migratory birds, individual's finite and often slow movements prevent it to make contact to a large number of individuals in a unit time. Such a mechanism is better described by  $b\frac{x}{x+y}y$  than  $bxy$ .<sup>11-15</sup> As migratory birds always appear in a large population, so standard incidence should be taken as horizontal incidence to get an appropriate and correct result.
- (2) The second point which is more vital and the main focus of the present paper is the role of environmental fluctuation in such systems. The paper of Chatterjee and Chattopadhyay<sup>1</sup> rests on the assumption that the environmental parameters involved with the model system are all constant irrespective of time and environmental fluctuations. But, environmental fluctuation is an important component of an ecosystem. Within deterministic environment we seek the constant equilibrium population and then investigate their stability which follows from the dynamics of the interactions between and within the species. But, most natural phenomena do not follow strictly deterministic laws; they oscillate randomly about some average value so that the deterministic equilibrium is no longer an absolutely fixed state.<sup>16,17</sup> May<sup>18</sup> pointed out the fact that due to environmental fluctuation, the birth rates, carrying capacity, competition coefficients and other parameters involved with the model system exhibit random fluctuation to a greater or lesser extent. Consequently the equilibrium population distribution fluctuates randomly around some average values.

In this paper, we have modified their model taking into account the above-mentioned factors and studied the modified model both in the absence and presence of environmental fluctuation. Our analysis shows that the control method proposed

by us in the absence of the environmental disturbances, are also valid in the presence of environmental stochasticity, provided the intensity of the environmental fluctuation is under certain threshold value.

We have organized the paper as follows. In Sec. 2, we introduce the basic deterministic model with some preliminary results. The stability of different equilibrium points is discussed in Sec. 3. In Sec. 4, we outline the stochastic analogue of the deterministic model discussed in the above sections. The stochastic stability of the interior equilibrium point is discussed in Sec. 5. The numerical simulation is performed for both the models and the results are given in Sec. 6. The paper ends with a discussion.

## 2. The Deterministic Model

Chatterjee and Chattopadhyay<sup>1</sup> considered a predator-prey system, where the predator population  $P$  is present in the system and the prey population  $N$  migrates into the system. Before formulating the model equation, we would like to recall the basic assumptions made by Chatterjee and Chattopadhyay<sup>1</sup> on the predator and the migratory prey populations present in the system:

(A1) They considered the growth rate of the migratory prey population as,

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

where  $A$  is the constant rate of recruitment of the prey population (including newborns and migration) and  $d$  is the natural death rate of the prey population. In this case the population approaches  $\frac{A}{d}$  as  $t$  goes to infinity.

(A2) It was assumed that the infective prey population  $i$  is generated through infection of susceptible prey  $s$ , and also the infective prey population is not in a state of reproduction. But as time passes, some of them recover from the disease and become susceptible again. So, the dynamics of the prey population was written as

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

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 the natural death and the death due to the disease. Obviously,  $e \geq d$ .

(A3) They assumed that in the absence of the migratory prey, there exists some alternative resource for the growth of the predator population. Depending on that alternative resource, the predator population is assumed to grow in logistic fashion with carrying capacity  $k > 0$  and an intrinsic growth rate constant  $r > 0$ . Hence,

in the absence of the migratory prey, the growth equation of the predator is given by

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

They assumed that, in the presence of the migratory prey populations, the growth rate of the predator population was governed by both the alternative source and the migratory prey population.

(A4) It was observed that in the case of WNV, Salmonella, the predator population becomes infected after the predation of the infective prey. So, the predation of the infected prey population was included in the predator's growth equation with a negative sign. Moreover it was also observed that the infection does not spread among the predator population.<sup>1</sup>

(A5) Further for mathematical simplicity, they assumed 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.

With the above assumptions, Chatterjee and Chattopadhyay<sup>1</sup> proposed the following system of differential equations:

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

where  $s(t)$  is the density of susceptible prey population,  $i(t)$  is the density infective prey population, and  $p(t)$  is the density of their predator population at any time  $t$ . Moreover,  $s(0) \geq 0$ ,  $i(0) \geq 0$ ,  $p(0) \geq 0$ .

In system (1),  $k_1$  and  $k_2$  are the searching efficiency constants or the predation rate on the susceptible and infective prey population, respectively.  $k_1'$  and  $k_2'$  are the growth rates of the predator due to predation of the susceptible and infective prey population. Obviously,  $k_1 \geq k_1'$ ,  $k_2 \geq k_2'$  and  $k_1 \leq k_2$ .<sup>1</sup>

We modify their model by taking standard incidence as the mode of disease transmission. The modified model is as follows:

$$\begin{aligned} \frac{ds}{dt} &= A - \frac{bsi}{s+i} - k_1 sp - ds + fi \\ \frac{di}{dt} &= \frac{bsi}{s+i} - k_2 pi - (e + f)i \\ \frac{dp}{dt} &= rp \left(1 - \frac{p}{k}\right) + k_1' sp - k_2' pi. \end{aligned} \quad (2)$$

### 2.1. Positive invariance

Let us put Eq. (2) in a vector form by setting

$$X = \text{col}(s, i, p) \in R^3. \quad (3)$$

$$F(X) = \begin{bmatrix} F_1(X) \\ F_2(X) \\ F_3(X) \end{bmatrix} = \begin{bmatrix} A - \frac{bsi}{s+i} - k_1sp - ds + fi \\ \frac{bsi}{s+i} - k_2pi - (e+f)i \\ rp \left(1 - \frac{p}{k}\right) + k_1'sp - k_2'pi \end{bmatrix}, \quad (4)$$

where  $F : C_+ \rightarrow R^3$ . Then Eq. (2) becomes

$$\dot{X} = F(X), \quad (5)$$

with  $X(0) = X_0 \in R_+^3$ . It is easy to check in Eq. (4) that whenever choosing  $X(0) \in R_+^3$  such that  $X_i = 0$ , then  $F_i(x) |_{X_i=0} \geq 0$ , ( $i = 1, 2, 3$ ). Due to lemma,<sup>19</sup> any solution of Eq. (5) with  $X_0 \in R_+^3$ , say  $X(t) = X(t; X_0)$ , is such that  $X(t) \in R_+^3$  for all  $t > 0$ .

### 2.2. Equilibrium points and conditions for their existence

The system (2) has the following equilibrium points, the axial equilibrium point

$$E_1 \equiv \left( \frac{A}{d}, 0, 0 \right),$$

the planar equilibrium point

$$E_2 \equiv \left( \frac{A(e+f)}{e(b-e-f) + d(e+f)}, \frac{A(b-e-f)}{e(b-e-f) + d(e+f)}, 0 \right),$$

and

$$E_3 \equiv \left( \bar{s}, 0, \frac{k(r + k_1'\bar{s})}{r} \right),$$

where

$$kk_1k_1'\bar{s}^2 + r(d + kk_1)\bar{s} - rA = 0$$

and the interior equilibrium point  $E^* \equiv (s^*, i^*, p^*)$ , where

$$s^* = \frac{r(k_2p^* + e + f)(p^* - k)}{B},$$

$$i^* = \frac{r(k_2p^* + e + f - b)(k - p^*)}{B},$$

where,

$$B = k(k_2p^* + (e + f))\{(k_1' + k_2') - kk_2'b\},$$

and  $p^*$  satisfies the relation

$$w_1 p^{*3} + w_2 p^{*2} + w_3 p^* + w_4 = 0, \quad (6)$$

where

$$\begin{aligned} w_1 &= k_1 k_2 r - r k_2^2, \\ w_2 &= (k_1 - k_2) [r(e + f) - k k_2 r] + k_2 r(b + d - e) \\ w_3 &= -A k k_2 (k_1' + k_2') + (k_2 - k_1) r k (e + f) + (e - k_2 k) r b \\ &\quad + (e - d) r (k k_2 - (e + f)), \\ w_4 &= -A k (e + f) (k_1' + k_2') + b k (A k_2' - e r) + (e - d) r k (e + f). \end{aligned}$$

The axial equilibrium point  $E_1 \equiv (\frac{A}{d}, 0, 0)$  and the planar equilibrium point  $E_3 \equiv (\bar{s}, 0, \frac{k(r+k_1'\bar{s})}{r})$  exist for all parameter values, while the steady state  $E_2$  exists if

$$b > e + f. \quad (7)$$

**Remark 2.2.1.** From the relation (7), we observe that if the contact rate is greater than the death rate and the recovery rate of the infective prey population, then it may lead to the extinction of the predator population.

We have a unique positive value of  $p^*$  that satisfies the relation (6) if

$$b_1 < b < b_2, \quad (8)$$

where,

$$b_2 = \frac{k_1 k k_2 + (e + f)(k_2 - k_1) + (e - d)k_2 - k k_2^2}{k_2} \quad \text{and,} \quad b_1 = \max\{l_1, l_2\}$$

where,

$$\begin{aligned} l_1 &= \frac{r(e + f)(k(k_2 - k_1) + d - e) + (e - d) r k k_2 - A k k_2 (k_1' + k_2')}{(k k_2 - e) r} \\ l_2 &= \frac{A k (e + f) (k_1' + k_2') + r k (d - e) (e + f)}{k (A k_2' - e r)}. \end{aligned}$$

Additionally, for the existence of a unique positive equilibrium point  $E^*$ , the parameter  $b$  must also satisfy the following condition,

$$b > \frac{(k_1' + k_2')(k_2 p^* + e + f)}{k_2'} \equiv b_3. \quad (9)$$

**Remark 2.2.2.** From relation (8) and (9), we observe that for the existence of a unique positive equilibrium point  $E^*$ , the contact rate must lie within certain range whose upper bound is  $b_2$  and the lower bound is the maximum between  $b_1$  and  $b_3$ .

### 2.3. Boundedness of the solutions

Let us first recall (without proof) the following lemma (2.3.1) due to Barbalat.<sup>20</sup>

**Lemma 2.3.1.** *Let  $g$  be a real valued differential function defined on some half line  $[a, +\infty)$ ,  $a \in (-\infty, +\infty)$ . If (i)  $\lim_{t \rightarrow +\infty} g(t) = \alpha$ ;  $|\alpha| < +\infty$ , and (ii)  $g'(t)$  is uniformly continuous for  $t > a$ , then  $\lim_{t \rightarrow +\infty} g'(t) = 0$ .*

**Lemma 2.3.2.** *Assume that the initial condition of Eq. (2) satisfies  $s_0 + i_0 \geq \frac{A}{d}$ . Then either (i):  $s(t) + i(t) \geq \frac{A}{d}$  for all  $t \geq 0$  and therefore as  $t \rightarrow +\infty$ ,  $(s(t), i(t), p(t)) \rightarrow E_1 = (\frac{A}{d}, 0, 0)$  or (ii): there exists a  $t_1 > 0$  such that  $s(t) + i(t) < \frac{A}{d}$  for all  $t > t_1$ . Finally if  $s_0 + i_0 < \frac{A}{d}$ , then  $s(t) + i(t) < \frac{A}{d}$  for all  $t \geq 0$ .*

**Proof.** Using the Barbalat lemma given in Lemma 2.3.1, one can prove the above Lemma 2.3.2 and hence the proof is omitted.  $\square$

**Lemma 2.3.3.** *There is a  $M > 0$  such that for any positive solution  $(s(t), i(t), p(t))$  of the system (2),  $p(t) < M$  for all large  $t$ , where*

$$M = \frac{\bar{k}}{d}, \quad \bar{k} = A + \frac{r(1+d)^2}{4k}.$$

**Proof.** See Appendix A.  $\square$

Let  $\Omega$  be the following subset of  $R_{0,+}^3$ :

$$\Omega = \left\{ (s, i, p) \in R_{0,+}^3 : s + i \leq \frac{A}{d}, p \leq M \right\}. \quad (10)$$

**Theorem 2.3.1.** *The set  $\Omega$  is a global attractor in  $R_{0,+}^3$  and, of course, it is positively invariant.*

**Proof.** Due to Lemmas 2.3.2 and 2.3.3 for all initial conditions in  $R_{+,0}^3$  such that  $(s(t_0), i(t_0), p(t_0))$  does not belong to  $\Omega$ , either there exists a positive time, say  $T$ ,  $T = \max\{t_0, t^*\}$ , such that the corresponding solution  $(s(t), i(t), p(t)) \in \text{int } \Omega$  for all  $t > T$ , or the corresponding solution is such that  $(s(t), i(t), p(t)) \rightarrow E_1(\frac{A}{d}, 0, 0)$  as  $t \rightarrow +\infty$ . But,  $E_1 \in \partial\Omega$ . Hence the global attraction of  $\Omega$  in  $R_{0,+}^3$  has been proved.  $\square$

Assume now that  $(s(t_0), i(t_0), p(t_0)) \in \text{int } \Omega$ . Then Lemma 2.3.2 implies that  $s(t) + i(t) < \frac{A}{d}$  for all  $t > 0$  and also by Lemma 2.3.3 we know that  $p(t) < M$  for all large  $t$ . Let us remark that if  $(s(t_0), i(t_0), p(t_0)) \in \partial\Omega$ , because  $s(t_0) + i(t_0) = \frac{A}{d}$  or  $p(t_0) = M$  or both, then still the corresponding solutions  $(s(t), i(t), p(t))$  must immediately enter  $\Omega$  or coincide with  $E_1$ .

### 3. Local Stability of Different Equilibrium Points

**Theorem 3.1.** *The axial equilibrium  $E_1(\frac{A}{d}, 0, 0)$  is a saddle point with  $s$ -axis as stable manifold and  $ip$ -plane as an unstable manifold if  $b > e + f$  or  $p$ -axis as an unstable manifold and  $si$ -plane as stable manifold if  $b < e + f$ . The planar equilibrium  $E_2$  is locally stable or unstable accordingly  $h_1 >$  or  $< 0$  respectively, where*

$$h_1 = (b - e - f)(re - k_2'A) + (e + f)(rd + k_1'A).$$

*The planar equilibrium  $E_3$  is locally stable or unstable accordingly*

$$rb - k_2k(r + k_1'\bar{s}) - r(e + f) > \quad \text{or} \quad < 0,$$

*respectively.*

**Proof.** The proof is obvious (using the variational matrix approach) and hence omitted.  $\square$

**Theorem 3.2.** *The positive steady state  $E^*(s^*, i^*, p^*)$  is locally asymptotically stable if  $F_1, F_2, G_1, G_2$  all are greater than 1, where*

$$F_1 = \frac{\frac{r}{k} \left\{ L + \frac{bs^*i^*}{(s^*+i^*)^2} \right\} + k_1k_2s^*}{k_1k_2i^*}$$

$$F_2 = \frac{fk_1k_2}{\left\{ k_1k_2L + \frac{frbi^*}{k(s^*+i^*)^2} + \frac{bk_1k_2s^{*2}}{(s^*+i^*)^2} \right\}}$$

$$G_1 = \frac{LMs^* + M^2s^* + s^{*2}p^*k_1k_2 + s^*i^*p^*k_1k_2}{\left\{ L + \frac{bs^*i^*}{(s^*+i^*)^2} \right\} \left\{ fi^* + \frac{rp^*}{k}s^* \right\} + s^{*2}p^*k_1k_2}$$

$$G_2 = \frac{k_1^2(L + M)s^*}{k_2(Mk_2 + fk_1)i^*},$$

*where,*

$$L = \frac{bi^{*2}}{(s^* + i^*)^2} + k_1p^* + d, \quad \text{and} \quad M = \frac{bs^*i^*}{(s^* + i^*)^2} + \frac{rp^*}{k}.$$

**Proof.** See Appendix B.  $\square$

The above discussion rests on the assumption that the environmental parameters involved with the model system are all constants irrespective of time and environmental fluctuations. In the next section, we will consider the effect of environmental fluctuation on the model system (2) and the stochastic stability of the co-existing equilibrium point associated with the model system.



#### 4. The Stochastic Model

The study of the dynamical behavior of the migratory bird population under variable environmental conditions is of great interest,<sup>21</sup> specially since their mobility depends on the environmental conditions.<sup>22</sup> The relation between the environment and the population dynamics in the birds has been studied by various researchers.<sup>23-26</sup> In the present article, we also like to observe the dynamics of system (2) under the influence of stochastic perturbation.

There are two ways to develop the stochastic model corresponding to an existing deterministic one to study the effect of the fluctuating environment. Firstly, one can replace the environmental parameters involved with the deterministic model system by some random parameters (e.g. the growth rate parameter “ $r$ ” can be replaced by  $r_0 + \epsilon\gamma(t)$ , where  $r_0$  is the average growth rate,  $\gamma(t)$  is the noise function and  $\epsilon$  is the intensity of fluctuation), secondly, one can add the randomly fluctuating driving force directly to the deterministic growth equations of prey and predator populations without altering any particular parameter.<sup>27</sup> A widely used way to incorporate the effect of environmental fluctuation in a system is to add the stochastic perturbation terms into the growth equation of the populations.<sup>28,29</sup> Thus we introduce stochastic perturbation terms into the growth equations of both prey and predator population to incorporate the effect of the randomly fluctuating environment. We assume that stochastic perturbations of the state variables around their steady-state values  $E^*$  are of Gaussian white noise type which are proportional to the distances of  $s, i, p$  from their steady-state values  $s^*, i^*, p^*$ , respectively.<sup>30</sup> Gaussian white noise is extremely useful to model rapidly fluctuating phenomena. Specially for terrestrial system, where the environmental variability is large at both short-periods and long-term periods and could be expected to develop internal mechanisms to the system which would cope with short-term variability and minimize the effects of long-term variations.<sup>31</sup> Hence, analysis of the system with white noise gives better results. So the deterministic model system (2) results in the following stochastic model system

$$\begin{aligned} ds &= F_1(s, i, p)dt + \sigma_1(s - s^*)d\xi_t^1 \\ di &= F_2(s, i, p)dt + \sigma_2(i - i^*)d\xi_t^2 \\ dp &= F_3(s, i, p)dt + \sigma_3(p - p^*)d\xi_t^3, \end{aligned} \quad (11)$$

where  $\sigma_1, \sigma_2$  and  $\sigma_3$  are real constants and known as intensity of environmental fluctuation,  $\xi_t^i = \xi_i(t)$ ,  $i = 1, 2, 3$  are standard Wiener processes independent from each other,<sup>32</sup> and  $F_1, F_2$ , and  $F_3$  are given by

$$\begin{aligned} F_1(s, i, p) &= A - \frac{bsi}{s+i} - k_1sp - ds + fi \\ F_2(s, i, p) &= \frac{bsi}{s+i} - k_2pi - (e+f)i \\ F_3(s, i, p) &= rp \left(1 - \frac{p}{k}\right) + k_1'sp - k_2'pi. \end{aligned}$$

In the rest of the work, we consider (11) as an Itô stochastic differential system of the type

$$dX_t = f(t, X_t)dt + g(t, X_t)d\xi_t, \quad X_{t_0} = X_0, \quad (12)$$

where the solution  $(X_t, t > 0)$  is an Itô process, “ $f$ ” is a slowly varying continuous component or *drift coefficient*, “ $g$ ” is the rapidly varying continuous random component or diffusion coefficient, and  $\xi_t$  is a three-dimensional stochastic process having scalar Wiener process components with increments  $\Delta\xi_t^j = \xi_j(t + \Delta t) - \xi_j(t)$  are independent Gaussian random variables  $\mathbf{N}(0, \Delta t)$ .

In the case of system (11),

$$X_t = (s, i, p)^T, \quad \xi_t = (\xi_t^1, \xi_t^2, \xi_t^3)^T, \quad f = \begin{bmatrix} F_1(s, i, p) \\ F_2(s, i, p) \\ F_3(s, i, p) \end{bmatrix}, \quad (13)$$

$$g = \begin{bmatrix} \sigma_1(s - s^*) & 0 & 0 \\ 0 & \sigma_2(i - i^*) & 0 \\ 0 & 0 & \sigma_3(p - p^*) \end{bmatrix}.$$

Since the diffusion matrix “ $g$ ” depends upon the solution of  $X_t$ , system (11) is said to have multiplicative noise.

## 5. Stochastic Stability of Interior Equilibrium

The stochastic differential system (11) can be centered at its positive equilibrium point  $E^*(s^*, i^*, p^*)$  by introducing the variables  $u_1 = s - s^*$ ,  $u_2 = i - i^*$ ,  $u_3 = p - p^*$ . It looks to be a very difficult problem to derive asymptotic stability in mean square sense by Lyapunov functions method working on the complete nonlinear Eqs. (11). For simplicity of mathematical calculations, we deal with the stochastic differential equations obtained by linearizing the vector function “ $f$ ” in (13) about the positive equilibrium point  $E^*$ . The linearized version of (12) around  $E^*$  is given by

$$dU(t) = F(U(t))dt + g(U(t))d\xi(t), \quad (14)$$

where  $U(t) = \text{col}(u_1(t), u_2(t), u_3(t))$  and

$$F(U(t)) = \begin{bmatrix} -a_{11}u_1 - a_{12}u_2 - a_{13}u_3 \\ a_{21}u_1 - a_{22}u_2 - a_{23}u_3 \\ a_{31}u_1 - a_{32}u_2 - a_{33}u_3 \end{bmatrix}, \quad (15)$$

$$g(U(t)) = \begin{bmatrix} \sigma_1u_1 & 0 & 0 \\ 0 & \sigma_2u_2 & 0 \\ 0 & 0 & \sigma_3u_3 \end{bmatrix},$$

with

$$\begin{aligned}
 a_{11} &= \frac{bi^{*2}}{(s^* + i^*)^2} + k_1 p^* + d, & a_{12} &= \frac{bs^{*2}}{(s^* + i^*)^2} - f, & a_{13} &= k_1 s^* \\
 a_{21} &= \frac{bi^{*2}}{(s^* + i^*)^2}, & a_{22} &= \frac{bs^* i^*}{(s^* + i^*)^2}, & a_{23} &= k_2 i^*, \\
 a_{31} &= k_1' p^*, & a_{32} &= k_2' p^*, & a_{33} &= \frac{rp^*}{k}.
 \end{aligned}$$

Note that, in (14) the positive equilibrium  $E^*$  corresponds to the trivial solution  $(u_1, u_2, u_3) = (0, 0, 0)$ . Let  $\Omega$  be the set defined by  $\Omega = [(t \geq t_0) \times \mathbb{R}^3, t_0 \in \mathbb{R}^+]$ . Let  $V \in C^3(\Omega)$  be a differentiable function of time  $t$  and twice differentiable function of  $X$ . We define the following theorem due to Afanasév *et al.*<sup>33</sup>

**Theorem 5.1.** *Suppose there exists a function  $V(U, t) \in C^3(\Omega)$  satisfying the inequalities*

$$K_1|U|^\alpha \leq V(U, t) \leq K_2|U|^\alpha. \quad (16)$$

$$LV(U, t) \leq -K_3|U|^\alpha, \quad K_i > 0, \quad i = 1, 2, 3, \quad \alpha > 0. \quad (17)$$

Then the trivial solution of (14) is exponentially  $\alpha$ -stable for all time  $t \geq 0$ .

Note that, if in (16), (17),  $\alpha = 2$ , then the trivial solution of (14) is exponentially mean square stable. Furthermore, the trivial solution of (14) is globally asymptotically stable in probability.

For definitions of stability again we refer to Afanasév *et al.*<sup>33</sup>

Remark that, with reference to (14),

$$LV(t, u) = \frac{\partial V(t, u(t))}{\partial t} + f^T(u(t)) \frac{\partial V(t, u)}{\partial u} + \frac{1}{2} \text{Tr} \left[ g^T(u(t)) \frac{\partial^2 V(t, u)}{\partial u^2} g(u(t)) \right] \quad (18)$$

where

$$\frac{\partial V}{\partial u} = \text{col} \left( \frac{\partial V}{\partial u_1}, \frac{\partial V}{\partial u_2}, \frac{\partial V}{\partial u_3} \right), \quad \frac{\partial^2 V(t, u)}{\partial u^2} = \left( \frac{\partial^2 V}{\partial u_j \partial u_i} \right)_{i,j=1,2,3},$$

and  $T$  means transposition.

We can prove the following:

**Theorem 5.2.** *Assume that for some positive real value  $\omega_2$  and  $\omega_3$  the following inequality holds true:*

$$[2bs^* i^* - \sigma_2^2 (s^* + i^*)^2] (2rp^* - k\sigma_3^2) > \left( \frac{k_2 i^*}{\omega_3} + \frac{k_2' p^*}{\omega_2} \right) (s^* + i^*)^2 k. \quad (19)$$

Then if

$$\sigma_1^2 < \frac{2bi^{*2}}{(s^* + i^*)^2} + 2k_1p^* + 2d, \quad \sigma_2^2 < \frac{2bs^*i^*}{(s^* + i^*)^2}, \quad \sigma_3^2 < \frac{2rp^*}{k}, \quad (20)$$

where,  $\omega_2 = \frac{bs^{*2} - f(s^* + i^*)^2}{bi^{*2}}$ , (where,  $\frac{bs^{*2}}{(s^* + i^*)^2} > f$ ) and  $\omega_3 = \frac{k_1s^*}{k_1'p^*}$  the zero solutions of system (11) exponentially 2-stable.

**Proof.** See Appendix C. □

Thus the internal parameters of the model system and the intensities of environmental fluctuation have the ability to maintain the stability of the stochastic model system and exhibit a balanced dynamics at any future time within a bounded domain of the parametric space. Here it is difficult to compare between the deterministic system (2) and the stochastic system (11) in terms of the system parameters because the conditions obtained for the stability of the two systems are very complicated. So, in order to compare these two systems we shall adopt the use of numerical simulation.

## 6. Numerical Analysis

The following set of parameter values  $A = 10$  individuals  $\text{ha}^{-1} \text{day}^{-1}$ ,  $r = 0.01 \text{ day}^{-1}$ ,  $k = 1$  individuals  $\text{ha}^{-1}$ ,  $k_1' = 0.6 \times k_1$  ha per individual  $\text{day}^{-1}$ ,  $k_2' = 0.6 \times k_2$  ha per individual  $\text{day}^{-1}$ ,  $d = 0.09 \text{ day}^{-1}$ ,  $f = 0.7 \text{ day}^{-1}$ ,  $e = 0.16 \text{ day}^{-1}$ ,  $b = 1.7 \text{ day}^{-1}$ ,  $k_1 = 0.03$  ha per individual  $\text{day}^{-1}$ , and  $k_2 = 0.03$  ha per individual  $\text{day}^{-1}$  are chosen for the numerical experiments of the models described in (2) and (11). For these values, Eq. (6) has only one positive root, i.e.  $p^* = 19.41$ . Substituting this value in the expressions of  $s^*$  and  $i^*$ , we get  $E^* = (12.45, 2.22, 19.41)$ . The eigenvalues associated with the characteristic Eq. (21) is given by  $-0.5247 \pm 0.3317i$ , and  $-0.0747$ . Thus, from our analytical result we can say that for the above set of hypothetical parameter values, system (2) possesses an unique positive steady state  $E^*$  which is locally asymptotically stable (see Fig. 1). We shall observe the dynamical behavior of system (2) around this positive steady state  $E^*$ .

Presence of infection in an ecological system plays a vital role in determining the dynamical behavior of the system. Anderson and May<sup>34</sup> showed that invasion of a resident predator-prey system by a new strain of parasites could cause destabilization and exhibit limit cycle oscillation. Haderler and Freedman<sup>35</sup> observed a similar type of phenomenon. Beltrami and Carroll<sup>36</sup> observed the role of viral disease in recurrent phytoplankton blooms and showed that introduction of virus-contaminated cells, even in very small numbers, has the effect of destabilization. Another important parameter in our model system is the recovery rate  $f$ . Chatterjee et al.<sup>37</sup> already showed that by increasing the recovery rate  $f$  it is possible to control the outbreak of the disease caused by migratory birds. So, we begin our analysis with these two parameters, i.e. the contact rate  $b$  and the recovery rate  $f$ .

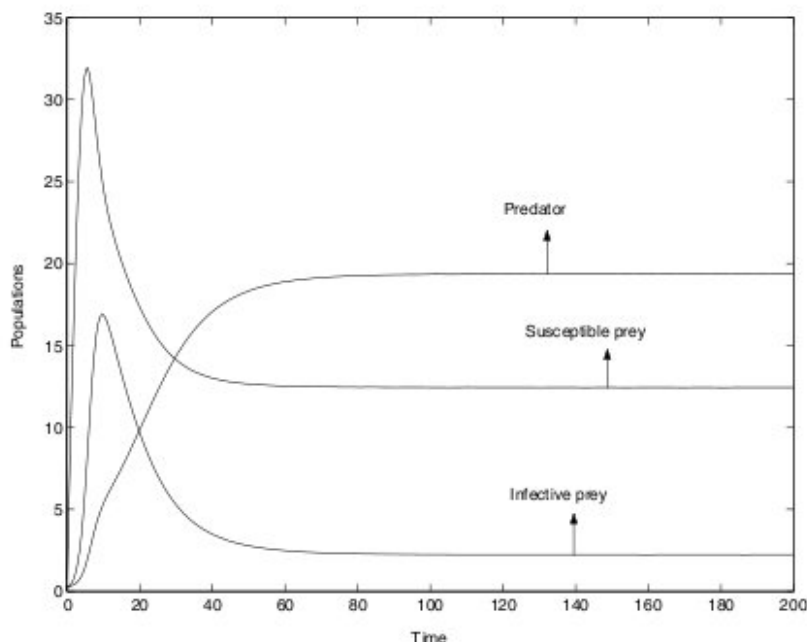


Fig. 1. Solution of ODEs for the above hypothetical set of parameter values, showing that  $E^*$  is (locally) asymptotically stable.

We shall discuss the role of  $b$  and  $f$  in the stability of the system around the positive steady state with the help of the bifurcation diagram produced by XPPAUT. It gives the values for variables  $(s, i, p)$  at different points on the bifurcation diagram, so that one can identify the particular steady states by grabbing the points. It also helps in identifying the position in the bifurcation diagram, where the stability switches from one steady state to the other (bifurcation point or BP) and the possibility for the co-existence of the steady states (limit points or LP).<sup>38</sup> From Fig. 2, we observe that the first bifurcation point (BP1) occur at  $b = 1.567$  ( $\equiv b_{b1}$ ), the second bifurcation point (BP2) occur at  $b = 1.732$  ( $\equiv b_{b2}$ ) and the LP occur at  $b = 1.93$  ( $\equiv b_l$ ). At BP1 the stability switches from the steady state  $E_3$  to the positive steady state  $E^*$ . At BP2 the positive steady state  $E^*$  and the steady state  $E_2$  are both stable. Finally, after crossing the LP the steady state  $E_2$  is only stable steady state. Similarly Fig. 3 also has two BPs and an LP. The LP is at  $f = 0.593$  ( $\equiv f_l$ ) and the first BP is at  $f = 0.684$  ( $\equiv f_{b1}$ ). From the LP to the first BP (BP1), both the positive steady state and the steady state  $E_2$  are stable. At BP1, the stability switches from the steady state  $E_2$  to the co-existence steady state  $E^*$ . Then at the second BP (BP2), which is at  $f = 0.833$  ( $\equiv f_{b2}$ ), the stability switches from the positive steady state  $E^*$  to the steady state  $E_3$ .

Thus, biologically we may conclude that if  $b$  is below  $b_{b1}$  or  $f$  is above  $f_{b2}$  then the disease is washed away from the system. But if  $b$  is above  $b_l$  or  $f$  is below  $f_l$

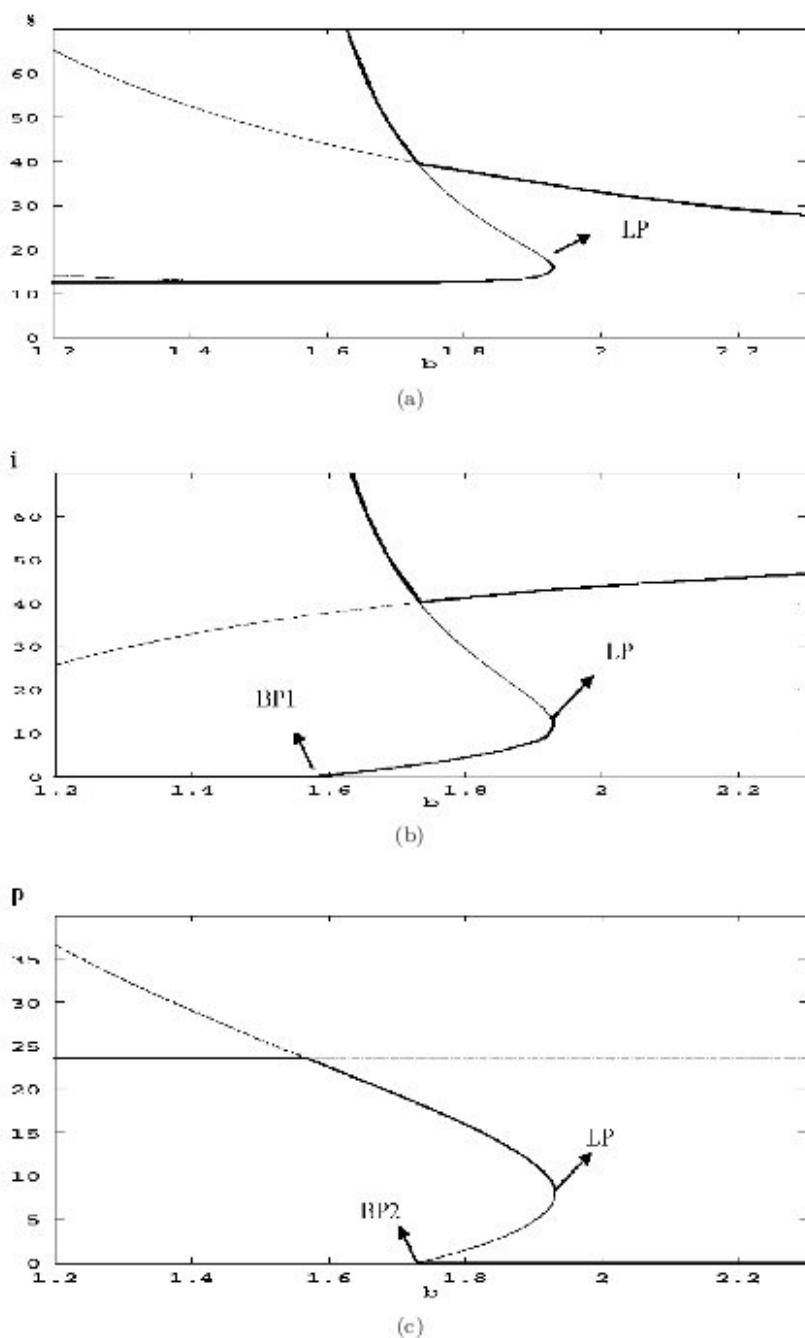


Fig. 2. Figure depicting population behavior for different values of contact rate, "b". (a) Susceptible prey vs. "b", (b) infective prey vs. "b", and (c) predator vs. "b". The solid deep line is stable steady state and the light line and the dashed line is the unstable steady state.

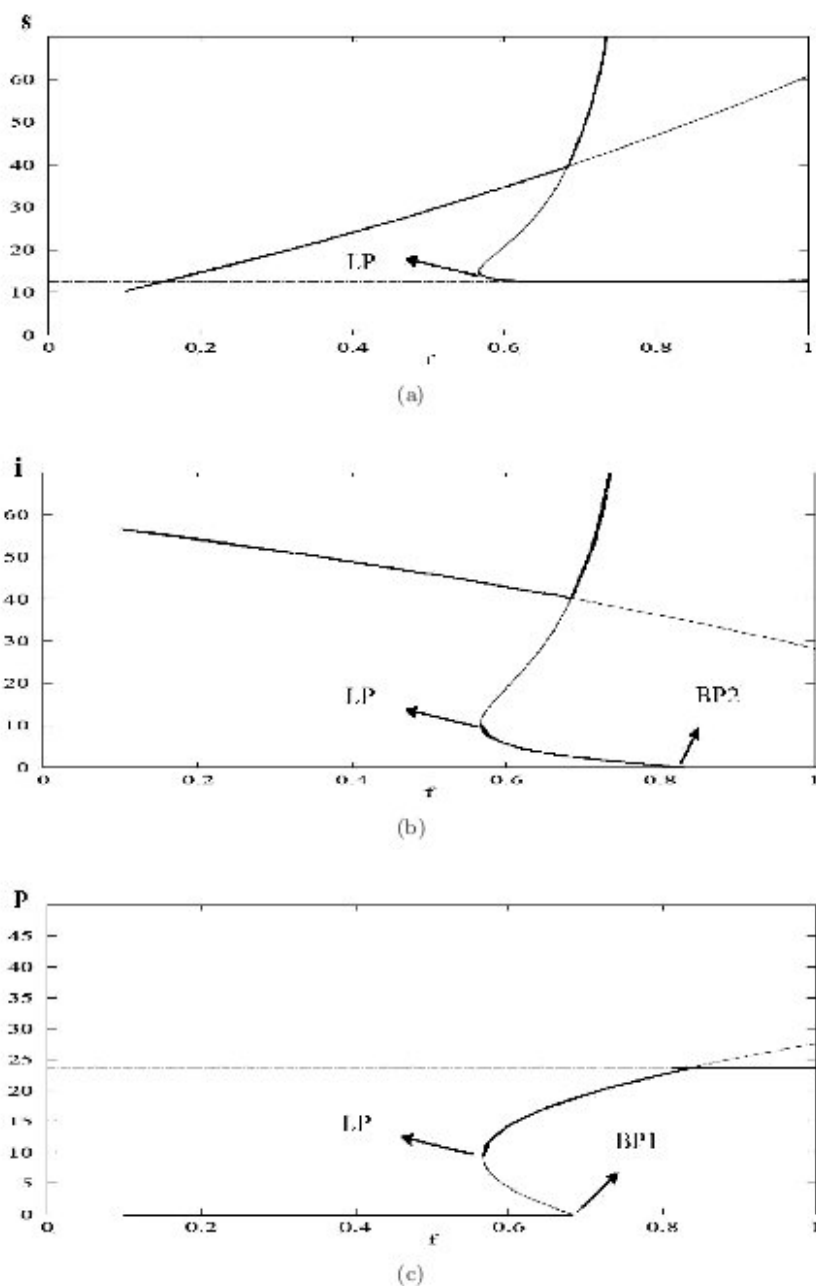


Fig. 3. Figure depicting population behavior for different values of recovery rate, "f". (a) Susceptible prey vs. "f", (b) infective prey vs. "f", and (c) predator vs. "f". The solid deep line is stable steady state and the light line and the dashed line is the unstable steady state.

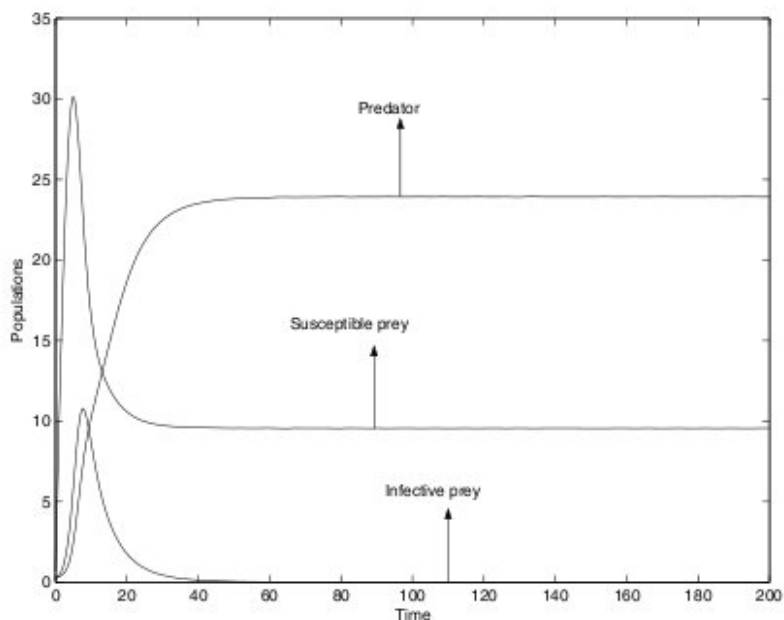
then there is a chance for the outbreak of the disease that leads to the extinction of the predator population. Thus, decrease in the value of  $b$  and increase in the value of  $f$ , prevents the outbreak of the disease and helps in the co-existence of all the species.

We have already seen that rate of infection and the recovery rate play vital roles in determining the dynamical behavior of our system (2). But, in an eco-epidemiological system along with infection rate, predation rate also plays an important role in controlling the dynamical behavior of the system.<sup>39,40</sup> Packer *et al.*<sup>39</sup> suggested that predation actually helps to reduce the infection load in the prey population. According to them, it may be possible to control infection among the prey population by predation. They cited several experimental observations also in support of their claim, e.g. Sih *et al.*<sup>9</sup> reviewed predator-removal experiments, where they removed the predator population from the system and observed its effect on the prey population that are infected by some transmissible disease. They found 54 of 135 systems in which prey populations subsequently declined. The same result was obtained by Hudson<sup>10</sup> when they examined the interaction between red grouse, the parasite nematode, *Trichostrongylus tenuis*, and their predators. In the above two experiments the author showed that the predator population actually helps in the existence of the prey population that are infected by some transmissible disease. So, next we shall study the role of the predation rate on both the susceptible and the infective prey. Keeping all other parameters fixed, if the predation rate on both the susceptible and infective prey is slightly increased from 0.03 to 0.04, it is observed that the system becomes disease free (see Fig. 4a). But, the predation rate must be monitor carefully, otherwise excessive predation on the infective population may result in the extinction of the predator population itself (see Fig. 4b).

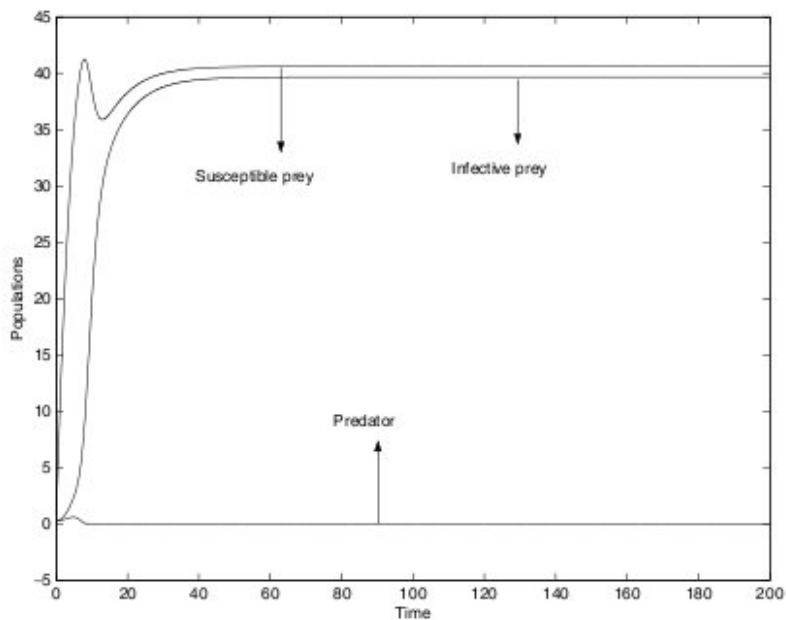
All the above results were obtained for the system (2), which is under constant environment. Next we shall observe the dynamical behavior of the system in the presence of the environmental disturbances. For the numerical simulation of the stochastic differential Eq. (11) we use the EulerMaruyama (EM) and Milstein methods with the help of MATLAB software, for the numerical scheme see Carletti.<sup>41</sup>

For the stochastic version of the model system (2), we have obtained the condition for asymptotic stability of equilibrium point  $E^*$  in mean square sense by using a suitable Lyapunov function (22). These conditions depend upon  $\sigma_1$ ,  $\sigma_2$ ,  $\sigma_3$  and the parameters involved with the model system. Now substituting the above set of parameter values, in the inequality (20) we get  $\sigma_1 < 1.19$ ,  $\sigma_2 < 0.66$  and  $\sigma_3 < 0.62$ . Moreover, for the above set of parameter values one can see that relation (19) is also satisfied. With the above set of parameter values and taking the values of  $\sigma_1 = 0.5$ ,  $\sigma_2 = 0.2$  and  $\sigma_3 = 0.1$ , we observe that 90% or more of the population distribution will lie within a said neighborhood (see Fig. 5). Hence we conclude that for the above set of parameter values, the system (11) is stochastically stable around





(a)



(b)

Fig. 4. Figure depicting population behavior for (a)  $k_1 = k_2 = 0.04$  and (b)  $k_1 = 0.04, k_2 = 0.6$ .

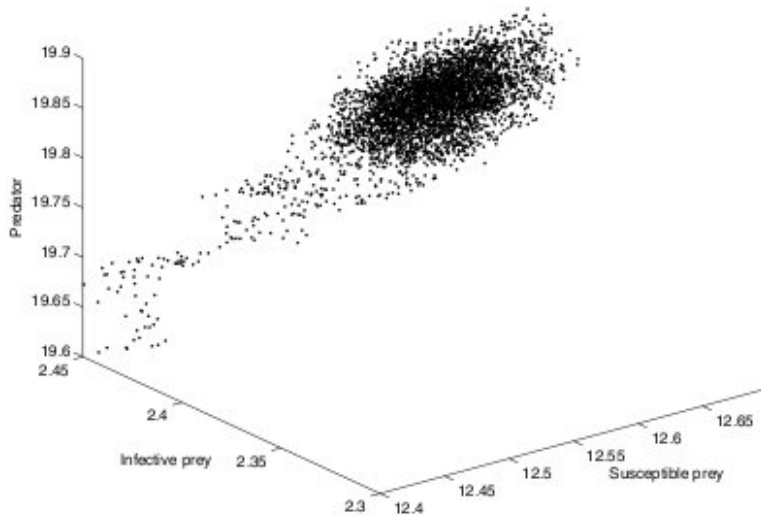


Fig. 5. The figure depicts the population distribution or probability cloud around the interior equilibrium point.

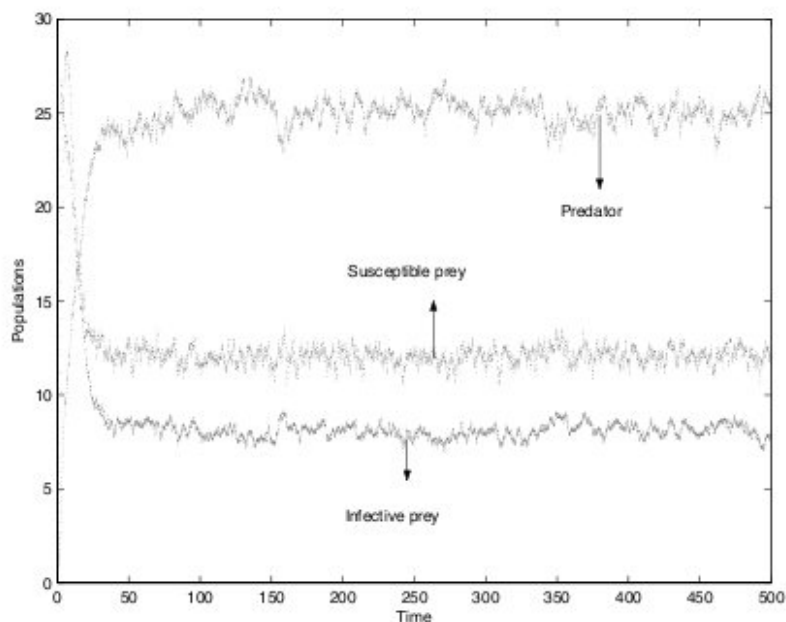
the positive steady state  $E^*$ . For the definition of stochastic stability in terms of probabilistic cloud, we refer to Bandyopadhyay and Chattopadhyay.<sup>42</sup>

Next, we have fixed the values of the environmental fluctuations at  $\sigma_1 = 0.7$ ,  $\sigma_2 = 0.4$  and  $\sigma_3 = 0.4$ , which is much below the respective threshold values. To observe the behavior of the system under the variable environment, we vary the three key parameters, namely the contact rate, the recovery rate and the predation rate.

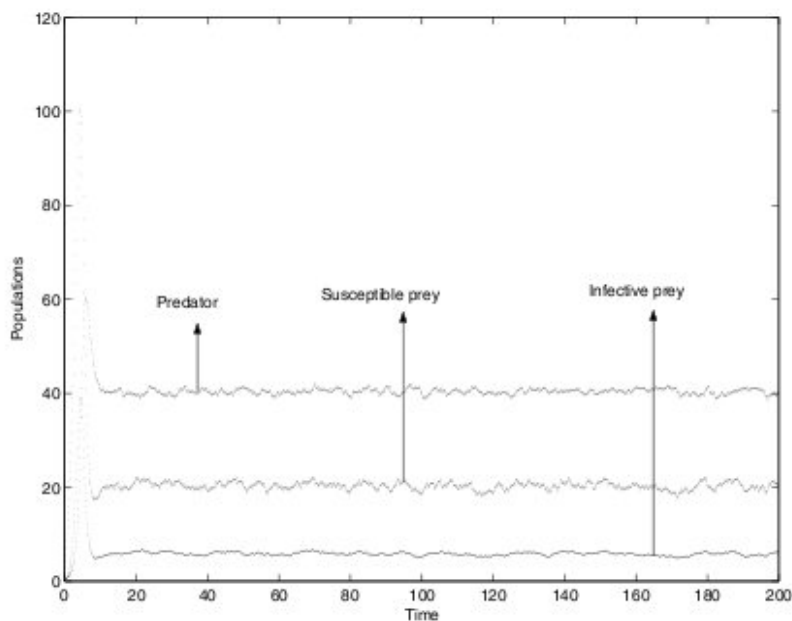
We have already seen in the deterministic situation that for certain values of the contact rate  $b$  and the recovery rate  $f$  there is an outbreak of the disease resulting in the extinction of the predator population (see Figs. 2 and 3). But it is interesting to observe that under stochastic perturbation all the three species co-exists for the same values of  $b$  and  $f$  (see Fig. 6). A similar result is obtain for the predation rate also. For example, keeping all other parameters fixed and increasing the value of  $k_2$  from 0.03 to 0.1, we observe that it is possible to control the epidemic caused by the migratory bird by increasing the predation rate even under a variable environment just by monitoring intensity of the environmental fluctuation (see Fig. 7b), which seems to be impossible under constant environment (see Fig. 7a).

## 7. Discussion

This article attempts to establish the effect of environmental stochasticity in an eco-epidemiological system, where the disease dynamic is controlled by some migratory birds. In this paper, we compare the situation for deterministic observation with stochastic perturbation around the positive equilibrium point.

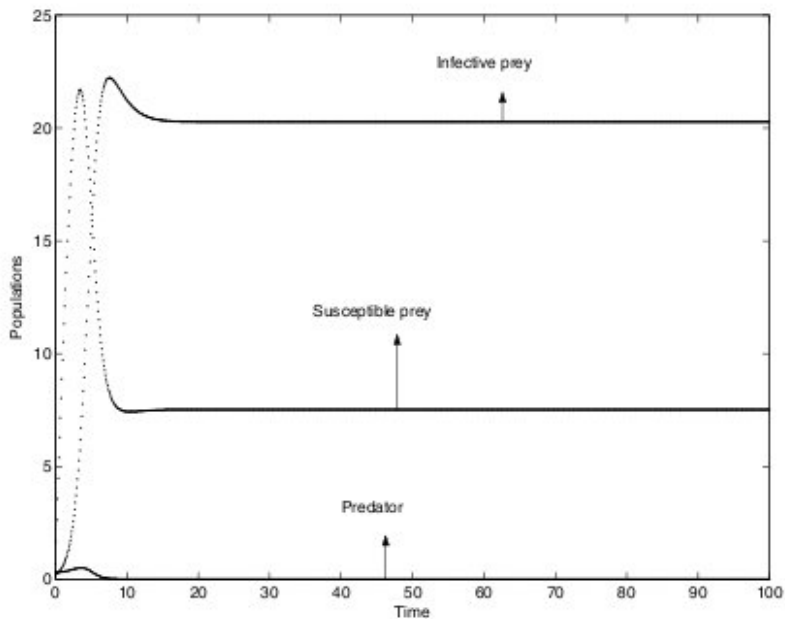


(a)

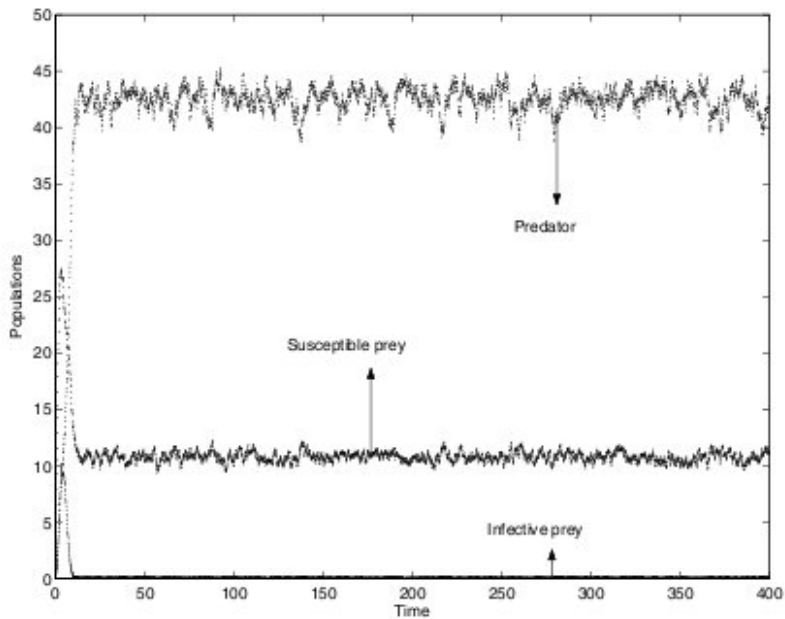


(b)

Fig. 6. (a) Solution of SDEs for  $b = 2.3$  and (b) solution of SDEs for  $f = 0.5$ , with  $\sigma_1 = 0.7$ ,  $\sigma_2 = 0.4$  and  $\sigma_3 = 0.4$ .



(a)



(b)

Fig. 7. (a) Solution of ODEs and (b) solution of SDEs; for  $k_1 = 0.03$ ,  $k_2 = 0.1$  with  $\sigma_1 = 0.7$ ,  $\sigma_2 = 0.4$  and  $\sigma_3 = 0.4$ .

First we have analyzed the system in the absence of the environmental fluctuation. In the deterministic case we have obtained a restriction on the contact rate  $b$  [see relations (8) and (9)] for which the positive steady state  $E^*$  exists. From the bifurcation diagram shown in Fig. 2, we observe that the steady state  $E^*$  is stable within a certain range ( $b_{b1}$ ,  $b_l$ ). Outside this range  $E^*$  may exist but is unstable and  $E_2$  or  $E_3$  is stable depending on the parameter value. We observe that careful monitoring of the contact rate can make the system disease free (see Fig. 2). We observe the same role for recovery rate  $f$  (see Fig. 3). We also observe that by increasing the predation rate one can make the system disease free, but the predation rate on the infective prey must not be high, otherwise the predator population will wash out from the system.

Further, to study the effect of environmental variability on this system, we have stochastically perturbed the system with respect to white noise around its positive equilibrium point. We observe that if the intensity of the environmental fluctuation is kept under a certain threshold value [see relation (20)], the system remains stochastically stable around the positive equilibrium. Numerical simulation establishes the fact that the steady state of the stochastic version is not absolutely fixed but a “fuzzy” value around which the population fluctuates. We observe that by monitoring the intensity of the environmental fluctuation we can make the system stable around the co-existence steady state, which was unstable for those values in the deterministic situation and so the deterministic model becomes more robust under stochastic fluctuation. Moreover, we observe from our numerical simulation that even in the presence of the environmental fluctuation we can control the outbreak of the disease caused by the migratory birds by monitoring the predation rate. Thus, we may conclude that if we can control the artificial eutrophication (or intensity of white noise), then the control method proposed in the deterministic situation remains valid under variable environment.

Finally, we would like to mention that the stochastic model (11) is built as an analogous counterpart of the deterministic model (2) by incorporating the stochastic perturbation in the growth equation of the populations. One can also incorporate the uncertainty in the parameter values to observe the effect of environmental fluctuation in the parameters. We have ignored this in our model formulation because our model is a one-season model<sup>1</sup> and we mainly want to observe the changes in the growth rate of different populations due to variation in the environmental conditions. The introduction of the uncertainty in the parameter values are more useful where the growth rate or the death rate of a population is very sensitive to the daily change in the climatic conditions like in the case of mosquitoes.<sup>43</sup> Moreover, the stochastic model built by incorporating the uncertainty in the parameter values makes the analytical study a formidable task. Although the model (11) represents a simple stochastic analogue of the deterministic model (2), but it reveals the effect of environmental fluctuation on the spread of the disease due to migratory bird population.

## Acknowledgments

The authors gratefully acknowledge the comments of both the learned referees on the previous version of the paper. The comments have improved the content and the presentation of the manuscript. The research of Samrat Chatterjee is supported by the Department of Atomic Energy (Government of India).

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## Appendix A

### Proof of the Lemma 2.3.3.

Lemma 2.3.2 implies that for any  $(s(t_0), i(t_0), p(t_0))$  such that  $s(t_0) + i(t_0) \geq \frac{A}{d}$ , then either a time  $t_0 > 0$  exists for which  $s(t) + i(t) < \frac{A}{d}$  for all  $t > t_0$ , or  $\lim_{t \rightarrow \infty} S(t) = \frac{A}{d}$ ,  $\lim_{t \rightarrow \infty} i(t) = 0$ . Furthermore, if  $s(t_0) + i(t_0) < \frac{A}{d}$  then  $s(t) + i(t) \leq \frac{A}{d}$  for all  $t > 0$ . Hence in any case a non-negative time, say  $t^*$ , exists such that  $i(t) < \frac{A}{d}$ ,  $s(t) < \frac{A}{d} + \epsilon$ , for all  $t > t^*$ .

Let,

$$W = s(t) + i(t) + p(t).$$

Calculating the derivative of  $W$  along the solution of system (2), we find for  $t > t^*$

$$\begin{aligned} \dot{W} &\leq A - d(s(t) + i(t)) + p(t) \frac{(1 - p(t))}{k} \quad (\because k_1' < k_1) \\ &\leq A - dW + \frac{r(1+d)^2}{4k}, \end{aligned}$$

where  $\frac{r(1+d)^2}{4k}$  is the maximum value of the function  $\frac{rp(t)(1+d-p(t))}{k}$ .

$$\therefore \dot{W} \leq -dW + \bar{k},$$

where,

$$\bar{k} = A + \frac{r(1+d)^2}{4k}.$$

Thus, there exist a positive constant  $M$ , such that  $W(t) < M$  for all large  $t$ .  $\square$

## Appendix B

### Proof of the Theorem 3.2.

The variational matrix  $J^*$  of the system (2) around  $E^*(s^*, i^*, p^*)$  is given by:

$$J^* = \begin{bmatrix} -\left(\frac{bi^{*2}}{(s^*+i^*)^2} + k_1 p^* + d\right) & -\frac{bs^{*2}}{(s^*+i^*)^2} + f & -k_1 s^* \\ \frac{bi^{*2}}{(s^*+i^*)^2} & -\frac{bs^* i^*}{(s^*+i^*)^2} & -k_2 i^* \\ k_1' p^* & -k_2' p^* & -\frac{rp^*}{k} \end{bmatrix}.$$



The characteristic equation of  $J^*$  is given by

$$\zeta^3 + Q_1\zeta^2 + Q_2\zeta + Q_3 = 0, \quad (21)$$

where

$$\begin{aligned} Q_1 &= L + M \\ Q_2 &= ML + \left( \frac{bi^*}{(s^* + i^*)^2} \right) [Ms^* - fi^*] + p^*[k_1^2s^* - k_2^2i^*] \\ Q_3 &= \left[ \left( \frac{brs^*i^*p^*}{k(s^* + i^*)^2} \right) - k_2^2i^*p^* \right] L + \left( \frac{-bs^{*2}}{(s^* + i^*)^2} + f \right) \\ &\quad \times \left[ \left( \frac{-brp^*i^{*2}}{k(s^* + i^*)^2} \right) + k_1k_2p^*i^* \right] + k_1s^* \left[ \frac{-k_2bi^{*2}p^*}{(s^* + i^*)^2} + \frac{k_1bs^*i^*p^*}{(s^* + i^*)^2} \right], \end{aligned}$$

with

$$L = \frac{bi^{*2}}{(s^* + i^*)^2} + k_1p^* + d > 0, \quad \text{and} \quad M = \frac{bs^*i^*}{(s^* + i^*)^2} + \frac{rp^*}{k} > 0.$$

By the Routh-Hurwitz criterion, we observe that if the conditions stated in the theorem hold true, then the positive steady state  $E^*(s^*, i^*, p^*)$  is locally asymptotically stable.  $\square$

## Appendix C

### Proof of the Theorem 5.2.

Let us consider the Lyapunov function

$$V(u(t)) = \frac{1}{2} [u_1^2 + \omega_2 u_2^2 + \omega_3 u_3^2], \quad (22)$$

where  $\omega_i$  are real positive constants to be chosen later.

It is easy to check that inequalities (16) are true for  $p = 2$ . Furthermore

$$\begin{aligned} LV(u(t)) &= - \left( \frac{bi^{*2}}{(s^* + i^*)^2} + k_1p^* + d \right) u_1^2 - \frac{bs^*i^*}{(s^* + i^*)^2} \omega_2 u_2^2 - \frac{rp^*}{k} \omega_3 u_3^2 \\ &\quad - \left( \frac{bs^{*2}}{(s^* + i^*)^2} - f - \frac{bi^{*2}\omega_2}{(s^* + i^*)^2} \right) u_1 u_2 - (k_2i^*\omega_2 + k_2'p^*\omega_3) u_2 u_3 \\ &\quad - (k_1s^* - k_1'p^*\omega_3) u_1 u_3 + \frac{1}{2} \text{Tr} \left[ g^T(u(t)) \frac{\partial^2 V}{\partial u^2} g(u(t)) \right]. \end{aligned} \quad (23)$$

Using (15), we get

$$\frac{1}{2} \text{Tr} \left[ g^T(u(t)) \frac{\partial^2 V}{\partial u^2} g(u(t)) \right] = \frac{1}{2} [\sigma_1^2 u_1^2 + \omega_2 \sigma_2^2 u_2^2 + \omega_3 \sigma_3^2 u_3^2]. \quad (24)$$

If in (23) we choose

$$\omega_2 = \frac{bs^{*2} - f(s^* + i^*)^2}{bi^{*2}} \quad \left( \text{where, } \frac{bs^{*2}}{(s^* + i^*)^2} > f \right), \quad \omega_3 = \frac{k_1 s^*}{k_1' p^*},$$

from (24) it is easy to check that

$$\begin{aligned} LV(u(t)) &= - \left( \frac{bi^{*2}}{(s^* + i^*)^2} + k_1 p^* + d \right) u_1^2 - \frac{bs^* i^*}{(s^* + i^*)^2} \omega_2 u_2^2 - \frac{rp^*}{k} \omega_3 u_3^2 \\ &\quad - (k_2 i^* \omega_2 + k_2' p^* \omega_3) u_2 u_3 + \frac{1}{2} [\sigma_1^2 u_1^2 + \omega_2 \sigma_2^2 u_2^2 + \omega_3 \sigma_3^2 u_3^2], \\ &= -u^T Q u. \end{aligned} \quad (25)$$

where

$$Q = \begin{bmatrix} \frac{bi^{*2}}{(s^* + i^*)^2} + k_1 p^* + d - \frac{\sigma_1^2}{2} & 0 & 0 \\ 0 & \omega_2 \left( \frac{bs^* i^*}{(s^* + i^*)^2} - \frac{\sigma_2^2}{2} \right) & \frac{1}{2} (k_2 i^* \omega_2 + k_2' p^* \omega_3) \\ 0 & \frac{1}{2} (k_2 i^* \omega_2 + k_2' p^* \omega_3) & \omega_3 \left( \frac{rp^*}{k} - \frac{\sigma_3^2}{2} \right) \end{bmatrix}.$$

The relations (19) and (20) imply that  $Q$  is a real symmetric positive definite matrix and therefore all its eigenvalues  $\lambda_i(Q)$ ,  $i = 1, 2, 3$ , are positive real numbers. Let  $\lambda_m = \min\{\lambda_i(Q), i = 1, 2, 3\}$ ,  $\lambda_m > 0$ . From (25) we get

$$LV(u(t)) \leq -\lambda_m |u(t)|^2.$$

According to Theorem 5.2 the proof is completed.  $\square$