

# Time delay factor can be used as a key factor for preventing the outbreak of a disease—Results drawn from a mathematical study of a one season eco-epidemiological model

Samrat Chatterjee<sup>a,1</sup>, Kalyan Das<sup>b</sup>, J. Chattopadhyay<sup>a,\*</sup>

<sup>a</sup>*Agricultural and Ecological Research Unit, Indian Statistical Institute, Kolkata 700108, India*

<sup>b</sup>*Centre for Mathematical Biology and Ecology, Department of Mathematics, Jadavpur University, Kolkata 700 032, India*

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## Abstract

Chatterjee and Chattopadhyay [Role of migratory bird population in an simple eco-epidemiological model, *Math. Comp. Model. Dyn. Syst.*, in press] proposed and analyzed a one season eco-epidemiological model of susceptible and infective prey together with their predators. In such systems, time lags due to the gestation of the infective prey are of importance. In this paper we modify and analyze their model by taking this factor into consideration. Our analysis shows that the outbreak of the disease can be controlled by a careful and suitable increment of the time lag factor. Moreover, to preserve the stability of the coexisting equilibrium, the time lag factor plays an important role. To substantiate our analytical results, extensive numerical simulations are performed for a hypothetical set of parameter values.

*Keywords:* Eco-epidemiological system; Time-delay; Hopf bifurcation; Global stability; Permanence

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## 1. Introduction

The study of ecological systems with the influence of epidemiological parameters is termed as eco-epidemiology. The relevant literature on this field is now very rich [26,5,18,27,6,19] etc. Probably Haderl and Freedman [14] were the first who described a predator–prey model where the prey is infected by a parasite, and the prey in turn infects the predator with the parasite. Xiao and Chen [29] claimed that they were the first to formulate and analyze an eco-epidemiological model with time delay. But, as far as our knowledge goes, very little attention has been paid so far to understand the role played by a migratory prey in the spread of a disease. Such dynamics has its own importance. Migration may introduce a new disease to a new place, or it can even re-introduce a disease which was totally washed away from that place. For example, the 1962 epidemic of EEE in Jamaica resulted from the transport of the virus by birds from the continental United States, see [24]. In another example, West Nile virus (WNV) is introduced in the Middle East by migrating white storks, see [23]. It is observed that a predator can become infected by predation of a prey infected by WNV, see [12] (for other examples, see [4]).

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\* Corresponding author. Fax: +91 33 25753049.

E-mail address: joydev@isical.ac.in (J. Chattopadhyay).

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Chatterjee and Chattopadhyay [4] proposed and analyzed a one-season mathematical model of such a situation, where the prey population migrates from one place to another and carries a disease. They divided the migratory prey population into two groups, namely, the susceptible prey and the infective prey. They assumed that the predator population is present in the system, and its growth rate is governed by an alternative source along with the migratory prey population. Since most of the diseases that are spread by migratory birds, like salmonella [3], WNV [28] etc., are seasonally dependent, so they were mainly interested to see the behavior of the dynamical system for the period when the migratory birds were present in the considered system. Their analytical and numerical results showed that the introduction of a disease through a migratory population destabilizes an otherwise stable system around the interior equilibrium. They also observed that proper predation may be used as a suitable control measure for preventing the extinction of the species. These findings are in accordance with some experimental results [25,21].

However, in the above case the effects due to time lags have been neglected. It seems reasonable to assume that the predator population will not die instantaneously after eating the infective prey population. Rather, there is some time lag for gestation of the infective prey by the predator. Keeping this in mind, we have modified the model proposed by Chatterjee and Chattopadhyay [4] by incorporating a delay in the term involving the gestation of infective prey by the predator. We find that the solutions of the system are positively invariant and bounded. We have analytically studied the model and derived the conditions for the permanence of the positive steady state and global stability of the disease free steady state. We have estimated length of delay for which the system preserves its stability around the positive steady state. We have also derived the conditions for instability of the system around the interior equilibrium and Hopf bifurcation. We have observed that in our model system the stability of the disease free equilibrium does not depend on the value of  $\tau$ , i.e., the time delay. Numerical simulations for a hypothetical set of parameter values have been performed to support our analytical findings. It has been observed that a time delay can drive the system to sustained oscillations. Moreover, a time delay effect produced by a delay in gestation of the infective prey population may prevent the outbreak of the disease and may be a worthy candidate for a control programme implementation.

The paper is organized as follows. In Section 2, we outline the basic mathematical model with the positivity (see Section 2.1) and the boundedness of solutions (see Section 2.2). We recapitulate the main results obtained by Chatterjee and Chattopadhyay [4] in Section 3. In Section 4 we find conditions for the local stability (see Section 4.1), the time delay is estimated for which local stability is preserved (see Section 4.2), and bifurcation results (see Section 4.3) is studied both analytically and numerically. The global stability results and the permanence of the system are given in Section 5. The paper ends with a discussion.

## 2. Mathematical model

Chatterjee and Chattopadhyay [4] considered a predator–prey system, where the predator population  $P$  is present in the system and the prey population  $N$  is migrating into the system. Before formulating the model equations, we would like to recall the basic assumption made by Chatterjee and Chattopadhyay [4] on the predator and the migratory prey populations present in the system:

(A1) The model considered by them is for one season and so, instead of taking a logistic growth in the prey population, they considered a growth term known as constant immigration with exponential deaths [20] for the migratory prey population. Let  $A$  be the constant rate of recruitment of the prey population (including newborns and migration) [1,9] and  $d$  be the natural death rate of the prey population. Then the growth rate of the migratory prey population is given by,

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

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

(A2) The infective prey population  $i$  is generated through infection of the susceptible prey  $s$ . It was assumed that 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 may be 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 natural death and death due to the disease. Obviously,  $e \geq d$ .

(A3) In their paper they were studying the dynamics of the system for the season when the migratory prey is present. But in the absence of the migratory prey it was assumed that the predator population is present in the system. So, in the absence of migratory prey, i.e., outside the considered season, there must exist some alternative resource for the growth of the predator population. Depending on that alternative resource the predator population is assumed to grow in 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

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

Now, in the presence of the migratory prey it was reasonable to assume that the predator population would not switch over its predation totally from that alternative resource to the newly entered migratory prey population. So, in their model formulation they assumed that the growth rate of the predator population was governed by both the alternative source and the migratory prey population.

(A4) They also assumed that the predator population becomes infected after the predation of the infective prey. For example, the cats which predate on the song birds infected by salmonella can pick up the illness and die [<http://www.gov.nf.ca/agric/pubfact/salmonella.htm>], the same is seen for WNV [12]. So, the predation of the infected prey population is included in the predator's growth equation with a negative sign, as done by Chattopadhyay et al. [7] in an eco-epidemiological context. But it was assumed that the infection does not spread among the predator population because either the infected predators die out immediately after becoming infected by the disease and thus are removed from the system [27], or they are the dead end host of the disease like mammals in the case of WNV [8].

(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 [4] proposed the following system of differential equations for their problem:

$$\left. \begin{aligned}\frac{ds}{dt} &= A - bsi - k_1sp - ds + fi \\ \frac{di}{dt} &= bsi - k_2pi - (e + f)i \\ \frac{dp}{dt} &= rp \left( 1 - \frac{p}{k} \right) + k'_1sp - k'_2pi\end{aligned}\right\}, \quad (2.1)$$

where  $s(t)$  is the density of the susceptible prey population,  $i(t)$  is the density of the 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 (2.1),  $k_1$  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 populations. Obviously,  $k_1 \geq k'_1$  and  $k_2 \geq k'_2$ . Also,  $k_1 \leq k_2$  which is quite natural. For example, in the case of salmonella, the sick birds often appear uncomfortable, with heads drooped, wings out, feathers fluffed up, and breathing heavily and these types of behavior leave them more susceptible to predation by large birds or cats [<http://www.gov.nf.ca/agric/pubfact/salmonella.htm>], [8].

In the above model given by system (2.1), it was assumed that the infective prey has a negative effect on the growth rate of the predator population. But it seems reasonable to assume that the death of the predator population is not instantaneous, i.e., there is an elapsed time for the gestation of infected prey and hence a delay arises in the system. In this paper we are mainly interested to see how the predation process influences the epidemics. It is a well-known fact

(also mentioned in the above paragraph) that an infective prey is less active and can be caught more easily (see [29]), and thus the epidemic in a system is mainly monitored by the infective prey, especially if it has a negative effect on the growth of all other species as in our case. So, here we have assumed the delay effect only on the gestation of the infective prey. With this new assumption model Eq. (2.1) becomes

$$\left. \begin{aligned} \frac{ds}{dt} &= A - bsi - k_1sp - ds + fi \\ \frac{di}{dt} &= bsi - k_2pi - (e + f)i \\ \frac{dp}{dt} &= rp \left(1 - \frac{p}{k}\right) + k'_1sp - k'_2p(t - \bar{\tau})i(t - \bar{\tau}) \end{aligned} \right\}, \tag{2.2}$$

where  $\bar{\tau}(\bar{\tau} > 0)$  is the time required for the gestation of infective prey by the predator.

For simplicity, we write model (2.2) in dimensionless form as follows, by using the same variable transformations  $P = p/k, S = s, I = i, T = rt$ :

$$\left. \begin{aligned} \frac{dS}{dT} &= B - \sigma SI - \alpha SP - \delta S + \mu I \\ \frac{dI}{dT} &= \sigma SI - \beta IP - (\gamma + \mu)I \\ \frac{dP}{dT} &= P(1 - P) + \alpha' SP - \beta' P(t - \tau)I(t - \tau) \end{aligned} \right\}, \tag{2.3}$$

where  $\sigma = \frac{b}{r}, B = \frac{A}{r}, \delta = \frac{d}{r}, \gamma = \frac{e}{r}, \mu = \frac{f}{r}, \alpha = \frac{kk_1}{r}, \beta = \frac{kk_2}{r}, \alpha' = \frac{k'_1}{r}, \beta' = \frac{k'_2}{r}$  and  $\tau = r\bar{\tau}$ , with initial conditions  $\phi = (\phi_1, \phi_2, \phi_3)$  defined in the Banach space

$$C_+ = \{\phi \in C([-\tau, 0], R_{0,+}^3) : \phi_1(\theta) = S(\theta), \phi_2(\theta) = I(\theta), \phi_3(\theta) = P(\theta)\}, \tag{2.4}$$

where  $S(\theta) > 0, I(\theta) > 0, P(\theta) > 0, \theta \in C[-\tau, 0]$ . For convenience, in the following we replace  $T$  by  $t$  for the dimensionless time.

### 2.1. Positive invariance

Let us put Eq. (2.3) in a vector form by setting

$$X = \text{col}(S, I, P) \in R^3, \tag{2.5}$$

$$F(X) = \begin{bmatrix} F_1(X) \\ F_2(X) \\ F_3(X) \end{bmatrix} = \begin{bmatrix} B - \sigma SI - \alpha SP - \delta S + \mu I \\ \sigma SI - \beta IP - (\gamma + \mu)I \\ P(1 - P) + \alpha' SP - \beta' P(t - \tau)I(t - \tau) \end{bmatrix}, \tag{2.6}$$

where  $F : C_+ \rightarrow R^3$  and  $F \in C^\infty(R^3)$ . Then Eq. (2.3) becomes

$$\dot{X} = F(X), \tag{2.7}$$

with  $X(\theta) = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C_+$  and  $\phi_i(\theta) > 0 (i = 1, 2, 3)$ . It is easy to check in Eq. (2.6) that whenever choosing  $X(\theta) \in C_+$  such that  $X_i = 0$ , then  $F_i(x)|_{x_i(t)=0, x(t) \in C_+} \geq 0 (i = 1, 2, 3)$ . Due to lemma in [30], any solution of Eq. (2.7) with  $X(\theta) \in C_+$ , i.e.,  $X(t) = X(t, X(\theta))$ , is such that  $X(\theta) \in R^3$  for all  $t > 0$ .

## 2.2. Boundedness of solutions

**Lemma 2.1.** Assume that the initial conditions of Eq. (2.3) satisfy  $\phi_1(0) + \phi_2(0) \geq \frac{B}{\delta}$ ,  $\theta \in [-\tau, 0]$ . Then either (i)  $S(t) + I(t) \geq \frac{B}{\delta}$  for all  $t \geq 0$  and therefore as  $t \rightarrow +\infty$ ,  $(S(t), I(t), P(t)) \rightarrow E_1 = (\frac{B}{\delta}, 0, 0)$  or (ii) there exists a  $t_0 > 0$  such that  $S(t) + I(t) < \frac{B}{\delta}$  for all  $t > t_0$ . Finally, if  $\phi_1(0) + \phi_2(0) < \frac{B}{\delta}$ ,  $\theta \in [-\tau, 0]$ , then  $S(t) + I(t) < \frac{B}{\delta}$  for all  $t \geq 0$ .

**Proof.** We consider first  $S(t) + I(t) \geq \frac{B}{\delta}$  for all  $t \geq 0$ . From the first two equations of (2.3) we get

$$\begin{aligned} \frac{d}{dt}(S + I) &= B - \alpha SP - \delta S - \beta IP - \gamma I \\ &< [B - \delta(S(t) + I(t))] \quad (\because \delta < \gamma) \\ &\leq 0. \end{aligned} \tag{2.8}$$

Hence, for all  $t \geq 0$ , we have that  $\frac{dS}{dt} + \frac{dI}{dt} \leq 0$ . Let

$$\lim_{t \rightarrow \infty} S(t) + I(t) = \eta. \tag{2.9}$$

If  $\eta > \frac{B}{\delta}$ , then by the Barbalat Lemma [2], we have

$$\begin{aligned} 0 &= \lim_{t \rightarrow \infty} \frac{d}{dt}(S(t) + I(t)) = \lim_{t \rightarrow \infty} [B - \alpha S(t)P(t) - \delta S(t) - \beta I(t)P(t) - \gamma I(t)] \\ &\leq \lim_{t \rightarrow \infty} [B - \delta(S(t) + I(t))] \\ &= B - \delta \lim_{t \rightarrow \infty} (S(t) + I(t)) < 0. \end{aligned}$$

This contradiction shows that  $\eta = \frac{B}{\delta}$ , i.e.,

$$\lim_{t \rightarrow \infty} (S(t) + I(t)) = \frac{B}{\delta}. \tag{2.10}$$

Let us denote by  $g(t) = S(t) + I(t)$  for  $t \in [0, \infty)$ . Of course,  $g(t)$  is differentiable and  $\dot{g}(t)$  is uniformly continuous for  $t \in (0, +\infty)$ . Thus, with Eq. (2.10) all the assumptions of the Barbalat Lemma holds true, and therefore

$$\lim_{t \rightarrow \infty} \frac{d}{dt}(S(t) + I(t)) = 0. \tag{2.11}$$

Since from the first two equations of Eq. (2.3)

$$\frac{d}{dt}(S(t) + I(t)) = B - \alpha S(t)P(t) - \delta S(t) - \beta I(t)P(t) - \gamma I(t), \tag{2.12}$$

then Eq. (2.10) implies that

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{d}{dt}(S(t) + I(t)) &= \lim_{t \rightarrow \infty} [B - \alpha S(t)P(t) - \delta S(t) - \beta I(t)P(t) - \gamma I(t)] \\ &= \lim_{t \rightarrow \infty} [(\delta - \gamma)I(t) - \alpha S(t)P(t) - \beta I(t)P(t)] \\ &= - \lim_{t \rightarrow \infty} [(\gamma - \delta)I(t) + (\alpha S(t) + \beta I(t))P(t)]. \end{aligned} \tag{2.13}$$

Hence Eqs. (2.11) and (2.13) are in agreement if and only if  $\lim_{t \rightarrow \infty} I(t) = 0$  and  $\lim_{t \rightarrow \infty} P(t) = 0$ , which jointly from Eq. (2.10) implies  $\lim_{t \rightarrow \infty} S(t) = \frac{B}{\delta}$ . This completes case (i).

Suppose that assumption (i) is violated. Then there exists  $t_0 > 0$  at which for the first time  $S(t_0) + I(t_0) = \frac{B}{\delta}$ . According to Eq. (2.12) we have

$$\left. \frac{d}{dt}(S(t) + I(t)) \right|_{t=t_0} = B - \alpha S(t_0)P(t_0) - \delta S(t_0) - \beta I(t_0)P(t_0) - \gamma I(t_0) < 0.$$

This implies that once a solution with  $S + I$  has entered into the interval  $(0, \frac{B}{\delta})$ , then it remains bounded there for all  $t > t_0$ , i.e.,  $S(t) + I(t) < \frac{B}{\delta}$  for all  $t > t_0$ .

Finally, if  $\phi_1(\theta) + \phi_2(\theta) < \frac{B}{\delta}$ ,  $\theta \in [-\tau, 0]$ , then applying the previous argument it follows that  $S(t) + I(t) < \frac{B}{\delta}$  for all  $t > 0$ , i.e., (iii) holds true. This completes the proof.  $\square$

**Lemma 2.2.** *There is a  $M > 0$  such that for any positive solution  $(S(t), I(t), P(t))$  of system (2.3)  $P(t) < M$  for all large  $t$ , where*

$$M = \frac{\bar{k}}{\delta}, \quad \bar{k} = B + \frac{(1 + \delta)^2}{4}.$$

**Proof.** Lemma 2.1 implies that for any  $(\phi_1, \phi_2, \phi_3) \in C_+$  such that  $\phi_1(\theta) + \phi_2(\theta) \geq \frac{B}{\delta}$ ,  $\theta \in [-\tau, 0]$ , either a time  $t_0 > 0$  exists for which  $S(t) + I(t) \leq \frac{B}{\delta}$  for all  $t > t_0$ , or  $\lim_{t \rightarrow \infty} S(t) = \frac{B}{\delta}$ ,  $\lim_{t \rightarrow \infty} I(t) = 0$ . Furthermore, if  $\phi_1(\theta) + \phi_2(\theta) < \frac{B}{\delta}$ ,  $\theta \in [-\tau, 0]$  then  $S(t) + I(t) \leq \frac{B}{\delta}$  for all  $t > 0$ . Hence in any case a non-negative time, say  $t^*$ , exists such that  $I(t) < \frac{B}{\delta}$ ,  $S(t) < \frac{B}{\delta} + \varepsilon$ , for all  $t > t^*$ .

Set  $W = S(t) + I(t) + P(t)$ .

Calculating the derivative of  $W$  along the solutions of system (2.3), we find for  $t > t^* + \tau$

$$\begin{aligned} \dot{W} &= B - \alpha S(t)P(t) - \delta S(t) - \beta I(t)P(t) - \gamma I(t) + P(t)(1 - P(t)) - \beta' P(t - \tau)I(t - \tau) + \alpha' S(t)P(t) \\ &\leq B - \delta(S(t) + I(t)) + P(t)(1 - P(t)) \quad (\because \alpha' < \alpha) \\ &= B - \delta(S(t) + I(t) + P(t)) + P(t)(1 + \delta - P(t)) \\ &\leq B - \delta W + \frac{(1 + \delta)^2}{4}, \end{aligned}$$

where  $\frac{(1 + \delta)^2}{4}$  is the maximum value of the function  $P(t)(1 + \delta - P(t))$ . Therefore

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

where

$$\bar{k} = B + \frac{(1 + \delta)^2}{4}.$$

Thus, there exists a positive constant  $M$ , such that  $W(t) < M$  for all large  $t$ . The assertion of Lemma 2.2 now follows and the proof is completed.  $\square$

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

$$\Omega = \left\{ (S, I, P) \in R_{0,+}^3 : S + I \leq \frac{B}{\delta}, P \leq M \right\}. \quad (2.14)$$

**Theorem 2.1.** *The set  $\Omega$  is a global attractor in  $R_{0,+}^3$  and it is positively invariant.*

**Proof.** Due to Lemmas 2.1 and 2.2 for all initial conditions in  $C_+$  such that  $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta))$  do not belong to  $\Omega$  for  $\theta \in [-\tau, 0]$ , 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  $(S(t), I(t), P(t)) \rightarrow E_1(\frac{B}{\delta}, 0, 0)$  as  $t \rightarrow +\infty$ . But  $E_1 \in \partial\Omega$ . Hence the global attractivity of  $\Omega$  in  $R_{0,+}^3$  has been proved.  $\square$

Assume now that  $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in \text{int } \Omega$ . Then Lemma 2.1 implies that  $S(t) + I(t) < \frac{B}{\delta}$  for all  $t > 0$  and also by Lemma 2.2 we know that  $P(t) < M$  for all large  $t$ . Let us remark that if  $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in \partial\Omega$ ,  $\theta \in [-\tau, 0]$ , because  $\phi_1(\theta) + \phi_2(\theta) = \frac{B}{\delta}$  or  $\phi_3(\theta) = M$  or both, then the corresponding solutions  $(S(t), I(t), P(t))$  immediately enter  $\text{int } \Omega$  or coincide with  $E_1$ .

### 3. Qualitative analysis of the model without time delay

In this section we recall the main results obtained by Chatterjee and Chattopadhyay [4]. The model without time delay i.e., the system (2.3) (with  $\tau = 0$ ), possesses the following biological feasible equilibria.

$E_1 \equiv (\frac{B}{\delta}, 0, 0)$ ,  $E_2 \equiv (S_2, 0, P_2)$ , where  $S_2 = \frac{P_2 - 1}{\alpha}$  and  $P_2$  is given by the positive root of the quadratic equation  $\alpha P^2 + (\delta - \alpha)P - (\delta + B\alpha') = 0$ , and  $E_3 \equiv (S_3, I_3, 0)$ , where  $S_3 = \frac{\gamma + \mu}{\lambda}$ ,  $I_3 = \frac{B\lambda - \delta(\gamma + \mu)}{\gamma\lambda}$ . Based on their results we make the following two remarks.

**Remark 3.1.** The equilibria  $E_1$  and  $E_2$  exist for any parametric values, while  $E_3$  exists if  $\lambda > \frac{(\gamma + \mu)\delta}{B}$ .

**Remark 3.2 (LAS).** The axial equilibrium  $E_1$  is a saddle point with S-axis as stable manifold and the IP-plane as an unstable manifold if  $\lambda > \frac{(\gamma + \mu)\delta}{B}$  or the P-axis as an unstable manifold and the SI-plane as stable manifold if  $\lambda < \frac{(\gamma + \mu)\delta}{B}$ .

If  $\lambda > \frac{\alpha'[\beta(\delta - \alpha - Q) - 2\alpha(\gamma + \mu)]}{\delta + \alpha - Q}$ , where  $Q = \sqrt{(\delta - \alpha)^2 + 4\alpha(\delta + B\alpha')} > 0$  holds, then  $E_2$  is locally asymptotically stable (LAS).

If  $\lambda > \frac{(\gamma + \mu)(\alpha'\gamma + \beta'\delta)}{\beta'B - \gamma}$  holds, then  $E_3$  is LAS.

The regions of parameter space for which the model system (2.3) (with  $\tau = 0$ ) admits feasible interior equilibrium (s) must correspond to a positive root  $S^*$  of the quadratic equation

$$w_1 S^2 + w_2 S + w_3 = 0, \tag{3.1}$$

where  $w_1, w_2, w_3$  are given by

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

for which additionally,

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

They described the range of possibilities for which the interior positive equilibrium (s) exists. They found that if there is exactly one interior stationary solution  $E^*(S^*, I^*, P^*)$ ,  $S^* > 0$ ,  $I^* > 0$ ,  $P^* > 0$ , then it is a sink provided  $w_1 > 0$  and  $w_2 < 0$ . If there are two interior solutions, then one is saddle point and the other is a sink. They also found other possibilities for which a unique interior equilibrium exists like (i)  $\lambda < \beta\alpha'$  and (ii)  $\beta\alpha' < \lambda < \beta\alpha' + \alpha\beta'$ , but for these parametric conditions (given by (i) and (ii)), the interior equilibrium is saddle point in nature.

Finally, they concluded that predation can be used as a key factor for controlling the disease.

#### 4. Qualitative analysis of the model with time delay

##### 4.1. Local stability analysis

The variational matrix  $L$  of system (2.3) about any arbitrary point  $(S, I, P)$  is given by

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

**Remark 4.1.** The characteristic equation for the variational matrix  $L_1$  about the steady state  $E_1$  and the characteristic equation for the variational matrix  $L_2$  about the disease free steady state  $E_2$  remains the same as obtained for the non-delayed system (2.1) (in the dimensionless form). Thus, in our model system, the delay has no effect on the stability nature of the system about  $E_1$  and  $E_2$ .

The characteristic equation for the variational matrix  $L^*$  about  $E^*$  takes the following form:

$$D(\lambda, \tau) = \lambda^3 + \bar{l}\lambda^2 + \bar{m}\lambda + \bar{n} - e^{-\lambda\tau}(\bar{f}\lambda^2 + \bar{g}\lambda + \bar{h}) = 0, \quad (4.1)$$

where

$$\begin{aligned} \bar{l} &= 2P^* + \delta + \sigma I^* + \alpha P^* - 1 - \alpha' S^*, \\ \bar{m} &= -\sigma I^* \alpha' S^* - \delta \alpha' S^* + 2\alpha P^{*2} - \alpha P^* - \delta - \sigma I^* \mu + 2\delta P^* - \sigma I^* + 2\sigma I^* P^* + \sigma^2 I^* S^*, \\ \bar{n} &= 2\sigma^2 I^* S^* P^* + \sigma I^* \mu - 2\sigma I^* \mu P^* - \sigma I^* \beta P^* \alpha' S^* + \sigma I^* \mu \alpha' S^* + \alpha' P^* \beta I \mu - \sigma^2 I^* S^{*2} \alpha' - \sigma^2 I^* S^*, \\ \bar{f} &= -\beta I^*, \\ \bar{g} &= \beta \beta' I^* P^* - \alpha P^* \beta' I^* - \delta \beta' I^* + \sigma \beta', \\ \bar{h} &= \alpha \sigma \beta' S^* I^* P^* - \sigma \mu \beta' - \sigma \beta \beta' P^* + \delta \beta \beta' I^* P^* + \sigma^2 \beta' S^* + \alpha \beta \beta' I^* P^{*2}. \end{aligned}$$

To find conditions for the local asymptotic stability of system (2.3), we use the following theorem of Gopalsamy [13].

**Theorem 4.1.** A set of necessary and sufficient conditions for the equilibrium(s) to be asymptotically stable for all  $\tau \geq 0$  is the following:

- (i) The real parts of all the roots of  $D(\lambda, 0) = 0$  are negative.
- (ii) For all real  $\omega$  and any  $\tau \geq 0$ ,  $D(i\omega, \tau) \neq 0$  where  $i = \sqrt{-1}$ .

Here  $D(\lambda, 0) = 0$  has roots with negative real parts provided system (2.1) is locally asymptotically stable about different equilibria (for the conditions see Section 3).

For  $\omega = 0$ ,  $D(0, \tau) = \bar{n} - \bar{h} \neq 0$ .

Now for  $\omega \neq 0$ ,

$$D(i\omega, \tau) = -i\omega^3 - \bar{l}\omega^2 + \bar{m}i\omega + \bar{n} - e^{-\omega\lambda\tau}(-\bar{f}\omega^2 + \bar{g}i\omega + \bar{h}).$$

Let  $D(i\omega, \tau) = 0$  and separating the real and imaginary parts we get

$$-\bar{l}\omega^2 + \bar{n} = -\bar{f}\omega^2 \cos \omega\tau + \bar{h} \cos \omega\tau + \bar{g}\omega \sin \omega\tau,$$

$$-\omega^3 + \bar{m}\omega = \bar{f}\omega^2 \sin \omega\tau - \bar{h} \sin \omega\tau + \bar{g}\omega \cos \omega\tau.$$

Squaring and adding the above two equations we get

$$\omega^6 + Q_1\omega^4 + Q_2\omega^2 + Q_3 = 0,$$



where

$$\begin{aligned} Q_1 &= \bar{l}^2 - \bar{f}^2 - 2\bar{m}, \\ Q_2 &= \bar{m}^2 - 2\bar{l}\bar{n}^2 + 2\bar{f}\bar{h} - \bar{g}^2, \\ Q_3 &= \bar{n}^2 - \bar{h}^2. \end{aligned}$$

Sufficient conditions for the non-existence of a real number  $\omega$  satisfying  $D(i\omega, \tau) = 0$  can be written as

$$\omega^6 + Q_1\omega^4 + Q_2\omega^2 + Q_3 > 0,$$

which can be transformed to

$$\omega^6 + Q_1 \left[ \omega^2 + \frac{Q_2}{2Q_1} \right]^2 + Q_3 - \frac{Q_2^2}{4Q_1} > 0.$$

Therefore, a sufficient condition for  $E^*$  to be stable is (i)  $Q_1 > 0$  and (ii)  $Q_3 > \frac{Q_2^2}{4Q_1}$ .

#### 4.2. Estimation of the length of delay to preserve stability

In this section we assume that in the absence of delays,  $E^*$  is locally asymptotically stable. By continuity and for sufficiently small  $\tau > 0$ , all eigenvalues of (4.1) have negative real parts provided one can guarantee that no eigenvalue with positive real part bifurcates from infinity (which could happen since it is a retarded system). For the stability analysis we use the Nyquist criterion [10]. To do this, we consider the space of real valued continuous functions defined on  $[\tau, \infty)$  satisfying the initial conditions and linearize system (2.3) near its interior equilibrium  $E(x^*, y^*, z^*)$ . Let  $u(t)$ ,  $v(t)$  and  $w(t)$  be the respective linearized variables of this model. Then system (2.3) becomes

$$\left. \begin{aligned} \frac{du}{dt} &= \bar{A}_1 u(t) + \bar{A}_2 v(t) + \bar{A}_3 w(t) \\ \frac{dv}{dt} &= \bar{B}_1 u(t) + \bar{B}_2 w(t) \\ \frac{dw}{dt} &= \bar{C}_1 u(t) + \bar{C}_2 w(t) + \bar{C}_3 v(t - \tau) + \bar{C}_4 w(t - \tau) \end{aligned} \right\}, \tag{4.2}$$

where

$$\left. \begin{aligned} \bar{A}_1 &= -\sigma I^* - \alpha P^* - \delta, \bar{A}_2 = -\sigma S^* + \mu, \bar{A}_3 = -\alpha S^* \\ \bar{B}_1 &= \sigma I^*, \bar{B}_2 = -\beta I^* \\ \bar{C}_1 &= \alpha' P^*, \bar{C}_2 = 1 - 2P^* + \alpha' S^*, \bar{C}_3 = -\beta' P^*, \bar{C}_4 = -\beta' I^* \end{aligned} \right\}. \tag{4.3}$$

Let  $\bar{u}(L)$ ,  $\bar{v}(L)$  and  $\bar{w}(L)$  be the Laplace transform of  $u(t)$ ,  $v(t)$  and  $w(t)$ , respectively. Taking the Laplace transformation of system (4.2), we have

$$\begin{aligned} (L - \bar{A}_1)\bar{u}(L) &= \bar{A}_2\bar{v}(L) + \bar{A}_3\bar{w}(L) + u(0), \\ L\bar{v}(L) &= \bar{B}_1\bar{u}(L) + \bar{B}_2\bar{w}(L) + v(0), \\ (L - \bar{C}_2)\bar{w}(L) &= \bar{C}_1\bar{u}(L) + \bar{C}_3\bar{v}(L)e^{-L\tau} + \bar{C}_3K_1(L)e^{-L\tau} + \bar{C}_4\bar{w}(L)e^{-L\tau} + \bar{C}_4K_2(L)e^{-L\tau} + w(0), \end{aligned}$$

where

$$\begin{aligned} K_1(L) &= \int_{-\tau}^0 e^{-Lt} v(t) dt, \\ K_2(L) &= \int_{-\tau}^0 e^{-Lt} w(t) dt. \end{aligned}$$

The inverse Laplace transform of  $\bar{u}(L)$  will have terms which exponentially increase with time, if  $\bar{u}(L)$  has poles with positive real parts. Since  $E^*$  needs to be locally asymptotically stable, it is necessary and sufficient that all poles of  $\bar{u}(L)$  have negative real parts. We shall employ the Nyquist criterion which states that if  $L$  is the arc length of a curve encircling the right half-plane, the curve  $\bar{u}(L)$  will encircle the origin a number of times equal to the difference between the number of poles and the number of zeroes of  $\bar{u}(L)$  in the right half-plane.

Let  $F(L) = L^3 + \bar{l}L^2 + \bar{m}L + \bar{n} - e^{-L\tau}(\bar{f}L^2 + \bar{g}L + \bar{h})$  (from (4.1)). Then conditions for local asymptotic stability of  $E^*$  as given by Freedman et al. [10] are

$$\operatorname{Re} F(iv_0) = 0, \quad (4.4)$$

$$\operatorname{Im} F(iv_0) > 0, \quad (4.5)$$

where  $v_0$  is the smallest positive root of Eq. (4.4).

Now (4.4) and (4.5) become

$$-\bar{l}v_0^2 + \bar{n} = -\bar{f}v_0^2 \cos v_0\tau + \bar{h} \cos v_0\tau + \bar{g}v_0 \sin v_0\tau,$$

$$-v_0^3 + \bar{m}v_0 > \bar{f}v_0^2 \sin v_0\tau - \bar{h} \sin v_0\tau + \bar{g}v_0 \cos v_0\tau.$$

To get an estimation on the length of delay, we utilize the following conditions,

$$-\bar{l}v^2 + \bar{n} = -\bar{f}v^2 \cos v\tau + \bar{h} \cos v\tau + \bar{g}v \sin v\tau, \quad (4.6)$$

$$-v^3 + \bar{m}v > \bar{f}v^2 \sin v\tau - \bar{h} \sin v\tau + \bar{g}v \cos v\tau. \quad (4.7)$$

Therefore,  $E^*$  will be stable if inequality (4.7) holds at  $v = v_0$ , where  $v_0$  is the first positive root of Eq. (4.6). We shall now estimate an upper bound  $v_+$  of  $v_0$ , which would be independent of  $\tau$ . Then we estimate  $\tau$  so that (4.7) holds for all values of  $v$ ,  $0 \leq v \leq v_+$ , and hence in particular at  $v = v_0$ .

Maximizing  $-\bar{f}v^2 \cos v\tau + \bar{h} \cos v\tau + \bar{g}v \sin v\tau$ , subject to  $|\sin v\tau| \leq 1$ ,  $|\cos v\tau| \leq 1$  we obtain

$$-\bar{l}v^2 + \bar{n} \leq \bar{f}v^2 + \bar{h} + \bar{g}v. \quad (4.8)$$

Thus the unique positive solution of  $(\bar{f} + \bar{l})v^2 + \bar{g}v - (\bar{n} - \bar{h}) = 0$  denoted by  $v_+$  is always greater than or equal to  $v_0$ .

Hence if  $v_+ = \frac{-\bar{g} + \sqrt{\bar{g}^2 + 4(\bar{f} + \bar{l})(\bar{n} - \bar{h})}}{2(\bar{f} + \bar{l})}$  then from (4.8) we have  $v_0 \leq v_+$ .

Here we see that  $v_+$  is independent of  $\tau$ . Now we need to estimate  $\tau$  so that (4.7) holds for all  $0 \leq v \leq v_+$ . Now rearranging (4.7) by  $|\sin v\tau| \leq \tau v$  and  $|1 - \cos v\tau| \leq \frac{1}{2}\tau^2 v^2$ , we get

$$\frac{\bar{g}}{2}\tau^2 v^2 + (\bar{h} - \bar{f}v^2)\tau + (\bar{m} - \bar{g} - v^2) > 0. \quad (4.9)$$

Thus (4.7) will be satisfied if

$$A_0\tau^2 + B_0\tau + C_0 > 0,$$

where  $A_0 = \frac{\bar{g}}{2}v_+^2$ ,  $B_0 = \bar{h} - \bar{f}v_+^2$ ,  $C_0 = (\bar{m} - \bar{g} - v_+^2)$ .

Then the Nyquist criterion holds for  $0 \leq \tau \leq \tau_+$ , where  $\tau_+ = \frac{1}{2A_0}(-B_0 + \sqrt{B_0^2 + 4A_0C_0})$  and  $\tau_+$  gives an estimate for the length of delay for which stability is preserved. Thus we are now in a position to state the following theorem.

**Theorem 4.2.** *If there exists a  $\tau$  in  $0 \leq \tau \leq \tau_+$  such that  $A_0\tau^2 + B_0\tau + C_0 > 0$ , then  $\tau_+$  is the maximum value (length of delay) of  $\tau$  for which  $E^*$  is asymptotically stable.*

4.3. Bifurcation results

Let us consider  $\tau \neq 0$  and assume  $\lambda = \bar{\mu} + i\bar{\nu}$  in (4.1). Then separating the real and imaginary parts, we get the system of transcendental equations

$$\begin{aligned} &\bar{\mu}^3 - 3\bar{\mu}\bar{\nu}^2 - \bar{l}(\bar{\mu}^2 + \bar{\nu}^2) + \bar{m}\bar{\mu} + \bar{n} \\ &= \bar{f}(\bar{\mu}^2 - \bar{\nu}^2) \cos \bar{\nu}\tau e^{-\bar{\mu}\tau} + 2\bar{f}\bar{\nu}\bar{\mu} \sin \bar{\nu}\tau e^{-\bar{\mu}\tau} + \bar{g}\bar{\mu} \cos \bar{\nu}\tau e^{-\bar{\mu}\tau} + \bar{h} \cos \bar{\nu}\tau e^{-\bar{\mu}\tau} + \bar{g}\bar{\nu} \sin \bar{\nu}\tau e^{-\bar{\mu}\tau}, \end{aligned} \tag{4.10}$$

$$\begin{aligned} &-\bar{\nu}^3 + 3\bar{\mu}^2\bar{\nu} + 2\bar{l}\bar{\mu}\bar{\nu} + \bar{m}\bar{\nu} \\ &= -\bar{f}(\bar{\mu}^2 - \bar{\nu}^2) \sin \bar{\nu}\tau e^{-\bar{\mu}\tau} + 2\bar{f}\bar{\nu}\bar{\mu} \cos \bar{\nu}\tau e^{-\bar{\mu}\tau} - \bar{g}\bar{\mu} \sin \bar{\nu}\tau e^{-\bar{\mu}\tau} - \bar{h} \sin \bar{\nu}\tau e^{-\bar{\mu}\tau} + \bar{g}\bar{\nu} \cos \bar{\nu}\tau e^{-\bar{\mu}\tau}. \end{aligned} \tag{4.11}$$

Let us consider  $\lambda$  and hence  $\bar{\mu}$  and  $\bar{\nu}$  as functions of  $\tau$ . We are interested to know the change of stability of  $E^*$  which will occur at the values of  $\tau = \hat{\tau}$  for which  $\bar{\mu} = 0$  and  $\bar{\nu} \neq 0$ .

Then the Eqs. (4.10) and (4.11) become

$$\left. \begin{aligned} -\bar{l}\hat{\nu}^2 + \bar{n} &= -\bar{f}\hat{\nu}^2 \cos \hat{\nu}\hat{\tau} + \bar{h} \cos \hat{\nu}\hat{\tau} + \bar{g}\hat{\nu} \sin \hat{\nu}\hat{\tau} \\ -\hat{\nu}^3 + \bar{m}\hat{\nu} &= \bar{f}\hat{\nu}^2 \sin \hat{\nu}\hat{\tau} - \bar{h} \sin \hat{\nu}\hat{\tau} + \bar{g}\hat{\nu} \cos \hat{\nu}\hat{\tau} \end{aligned} \right\}. \tag{4.12}$$

Eliminating  $\hat{\tau}$ , we have

$$\hat{\nu}^6 + (\bar{l}^2 - \bar{f}^2 - 2\bar{m})\hat{\nu}^4 + (\bar{m}^2 - 2\bar{l}\bar{n}^2 + 2\bar{f}\bar{h} - \bar{g}^2)\hat{\nu}^2 + \bar{n}^2 - \bar{h}^2 = 0. \tag{4.13}$$

In order to establish Hopf bifurcation at  $\tau = \hat{\tau}$ , we need to show that  $\frac{d\bar{\mu}}{d\tau} \neq 0$  at  $\tau = \hat{\tau}$ . We differentiate (4.10) and (4.11) with respect to  $\tau$  and setting  $\tau = \hat{\tau}$ ,  $\bar{\mu} = 0$  and  $\bar{\nu} = \hat{\nu}$  we get

$$\left. \begin{aligned} \bar{L} \frac{d\bar{\mu}}{d\tau}(\hat{\tau}) + \bar{M} \frac{d\bar{\nu}}{d\tau}(\hat{\tau}) &= X \\ -\bar{M} \frac{d\bar{\mu}}{d\tau}(\hat{\tau}) + \bar{L} \frac{d\bar{\nu}}{d\tau}(\hat{\tau}) &= Y \end{aligned} \right\}, \tag{4.14}$$

where  $\bar{L} = -3\hat{\nu}^2 + \bar{m} - \bar{g} \cos \hat{\nu}\hat{\tau} - 2\bar{f}\hat{\nu} \sin \hat{\nu}\hat{\tau}$ ,  $\bar{M} = -2\bar{l}\hat{\nu} + 2\bar{f}\hat{\nu} \cos \hat{\nu}\hat{\tau} - \bar{g} \sin \hat{\nu}\hat{\tau}$ ,  $X = \bar{f}\hat{\nu}^3 \sin \hat{\nu}\hat{\tau} - \bar{h}\hat{\nu} \sin \hat{\nu}\hat{\tau} + \bar{g}\hat{\nu}^2 \cos \hat{\nu}\hat{\tau}$ ,  $Y = -\bar{g}\hat{\nu}^2 \sin \hat{\nu}\hat{\tau} + \bar{f}\hat{\nu}^3 \cos \hat{\nu}\hat{\tau} + \bar{h}\hat{\nu} \cos \hat{\nu}\hat{\tau}$ .

Solving (4.14), we get

$$\frac{d\bar{\mu}}{d\tau}(\hat{\tau}) = \frac{\bar{L}X - \bar{M}Y}{\bar{L}^2 + \bar{M}^2},$$

where  $\frac{d\bar{\mu}}{d\tau}(\hat{\tau})$  has the same sign as that of  $\bar{L}X - \bar{M}Y$ .

Substituting the values of  $\bar{L}$ ,  $\bar{M}$ ,  $X$ ,  $Y$  and using (4.12), we get

$$\bar{L}X - \bar{M}Y = \hat{\nu}^2 \left[ 3\hat{\nu}^4 + 2(\bar{l}^2 - \bar{f}^2 - 2\bar{m}) + (\bar{m}^2 - 2\bar{l}\bar{n}^2 + 2\bar{f}\bar{h} - \bar{g}^2) \right].$$

Let

$$F(z) = z^3 + A_1z^2 + A_2z + A_3,$$

where  $A_1 = \bar{l}^2 - \bar{f}^2 - 2\bar{m}$ ,  $A_2 = \bar{m}^2 - 2\bar{l}\bar{n}^2 + 2\bar{f}\bar{h} - \bar{g}^2$ ,  $A_3 = (\bar{n}^2 - \bar{h}^2)$  which is left hand side of (4.13) with  $\hat{\nu}^2 = z$ .

Then  $F(\hat{\nu}^2) = 0$  and we note that

$$\frac{d\bar{\mu}}{d\tau}(\hat{\tau}) = \frac{\hat{\nu}^2}{\bar{L}^2 + \bar{M}^2} \frac{dF}{dz}(\hat{\nu}^2). \tag{4.15}$$

Hence we can describe criteria for the preservation of stability (instability) geometrically as follows:

- (1) If the polynomial  $F(z)$  has no positive roots, there is no change of stability.
- (2) If  $F(z)$  is decreasing (increasing) at all its positive roots, stability (instability) is preserved.

We note the following fact:

- (i) For the existence of  $\hat{v} > 0$ ,  $F(z)$  must have at least one positive real root.  
 (ii) Since  $F(z)$  is cubic in  $z$ ,

$$\lim_{z \rightarrow \infty} F(z) = \infty.$$

(iii) If  $F(z)$  has a unique positive root, then it must increase at that point to satisfy (ii).

(iv) If  $F(z)$  has two or three distinct positive real roots, then it must decrease at one root and increase at the other, hence (ii) is not satisfied.

(v) If  $A_3 < 0$ , then  $F(z)$  has only one root.

(vi) If  $A_1 > 0$ ,  $A_3 > 0$  and  $A_2 < 0$ , then (i) will be satisfied.

Now, if  $A_1 > 0$ ,  $A_3 > 0$  and  $A_2 < 0$ , then the minimum of  $F(z)$  will exist at

$$z_{min} = \frac{-A_1 + \sqrt{A_1^2 - 3A_2}}{3}$$

and (i) will be satisfied if  $F(z_{min}) > 0$ , i.e.,

$$2A_1^3 - 9A_1A_2 + 27A_3 > 2(A_1^2 - 3A_2)^{3/2} \quad (4.16)$$

or

$$2A_1(A_1^2 - 3A_2) + 27A_3 - 3A_1A_2 > 2(A_1^2 - 3A_2)^{3/2}.$$

Since  $27A_3 - 3A_1A_2 > 27A_3$  (since  $A_1 > 0$ ,  $A_3 > 0$  and  $A_2 < 0$ ), and  $A_1^2 - 3A_2 > A_1^2$ , hence  $2A_1(A_1^2 - 3A_2) + 27A_3 - 3A_1A_2 > 27A_3 + 2A_1^3$ .

Thus, for Eq. (4.16) to hold it is sufficient that

$$27A_3 + 2A_1^3 > 2(A_1^2 - 3A_2)^{3/2} \Rightarrow A_2 > \frac{1}{3} \left[ A_1^2 - \left( \frac{27A_3 + 2A_1^3}{2} \right)^{2/3} \right].$$

Now we can state the following theorem.

**Theorem 4.3.** *If  $A_1 > 0$ ,  $A_3 > 0$  and relation (4.16) holds, then the stable positive equilibrium  $E^*$  remains stable for all  $\tau > 0$ .*

**Example 4.1.** Let us consider the following hypothetical set of parameter values:

$A = 7$  individuals  $\text{ha}^{-1} \text{day}^{-1}$ ,  $r = 5.4 \text{ day}^{-1}$ ,  $k = 40$  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  $\text{day}^{-1}$ ,  $d = 0.009 \text{ day}^{-1}$ ,  $f = 0.7 \text{ day}^{-1}$ ,  $e = 0.0097 \text{ day}^{-1}$ ,  $b = 0.05$  ha per individuals  $\text{day}^{-1}$ ,  $k_1 = 0.002$  ha per individuals  $\text{day}^{-1}$ , and  $k_2 = 0.003$  ha per individuals  $\text{day}^{-1}$ .

For this set of parameter values there exists a unique interior equilibrium  $E^*(16.5683, 43.1378, 39.5721)$ . Moreover, this positive steady state  $E^*$  is locally asymptotically stable, since the eigenvalues associated with the variational matrix of system (2.2) (given in the paper of Chatterjee and Chattopadhyay [4]) at  $E^*$  are given by  $(-1.6278, -0.0055, -0.9898)$ . With these parameter values and taking the initial population of the susceptible prey, infective prey and the predator as 3.3 individuals  $\text{ha}^{-1}$ , 1.6 individuals  $\text{ha}^{-1}$ , 1 individual  $\text{ha}^{-1}$ , respectively, we get Fig. 1.

With this set of parameter values one can easily see that  $A_1 = 0.1853 > 0$ , and  $A_3 = 8.5661 \times 10^{-5} > 0$ . Thus, according to Theorem 4.3, this interior equilibrium  $E^*$  remain stable for all  $\tau > 0$ . For  $\tau = 250$ , we observe that all three populations remain stable (see Fig. 2).

From Fig. 2 it is clear that system (2.2) is stable for  $\tau = 250$ . Moreover, we have also checked for other values of  $\tau > 250$  and found that the system remains stable for any  $\tau > 0$ .

**Theorem 4.4.** *If  $A_1 > 0$ ,  $A_3 < 0$  and if the positive equilibrium  $E^*$  is unstable for  $\tau = \tau_0$ , then it will remain unstable for all  $\tau > \tau_0$ .*

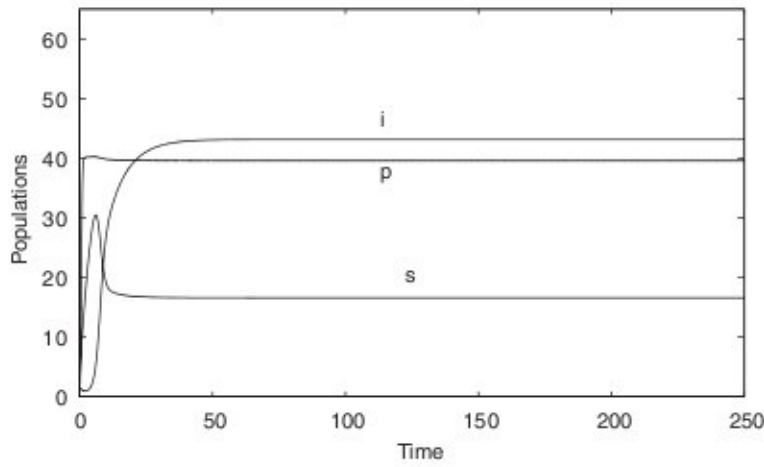


Fig. 1. Time evolution of all the population for the model system (2.1).

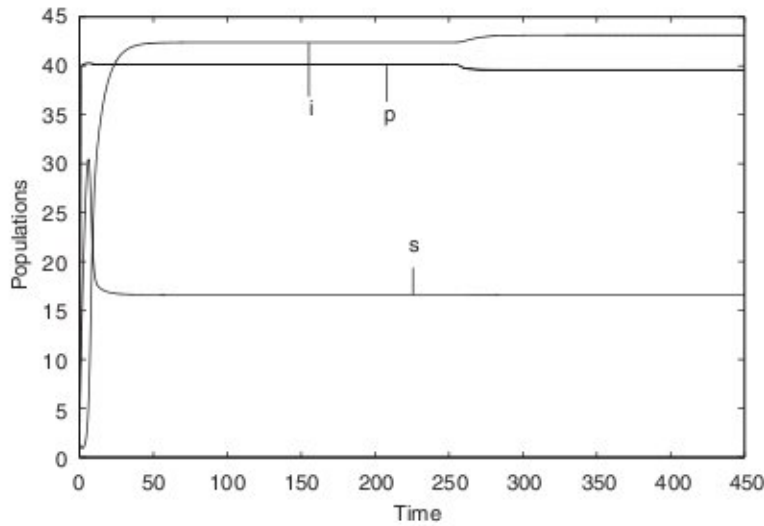


Fig. 2. This figure depicts the time evolution of all populations for the model system (2.2),  $\tau = 250$ .

Before proceeding further we state the following lemma due to G.J. Butler (see Appendix 2 in [11]).

**G. J. Butler’s Lemma:** Let

$$\Delta(\lambda, \tau) = \lambda^2 - (A + D)\lambda + (AD - BC) - BEe^{-\lambda\tau}.$$

If  $A + D < 0$ ,  $AD - BC > BE$ , then the real parts of the solutions of the above equation are negative for  $\tau < \tau_0$ , where  $\tau_0 > 0$  is the smallest value for which there is a solution to the above equation with real part zero.

The existence of unique  $\hat{v}$  is given by (v) and hence from (4.12) we have

$$\hat{\tau} = \frac{1}{\hat{v}} \tan^{-1} \left\{ \frac{\hat{v}(\bar{f}\hat{v}^4 - \bar{f}\bar{m}\hat{v}^2 - \bar{h}\hat{v}^2 - \bar{l}\bar{g}\hat{v}^2 + \bar{h}\bar{m} - \bar{n}\bar{g})}{\bar{g}\hat{v}^4 - \bar{g}\bar{m}\hat{v}^4 + \bar{f}\bar{n}\hat{v}^2 - \bar{h}\bar{l}\hat{v}^2 + \bar{f}\bar{l}\hat{v}^4 - \bar{h}\bar{n}} \right\} + \frac{n\pi}{\hat{v}}, \tag{4.17}$$

where  $n = 0, 1, 2, \dots$ . Our required  $\hat{\tau}$  is given by  $n = 0$  in (4.17). Since,  $\frac{d\hat{\tau}}{d\tau}(\hat{\tau}) > 0$ , it is clear that at least one eigenvalue of

$$\lambda^3 + \bar{l}\lambda^2 + \bar{m}\lambda + \bar{n} - e^{-\lambda\tau}(\bar{f}\lambda^2 + \bar{g}\lambda + \bar{h}) = 0,$$

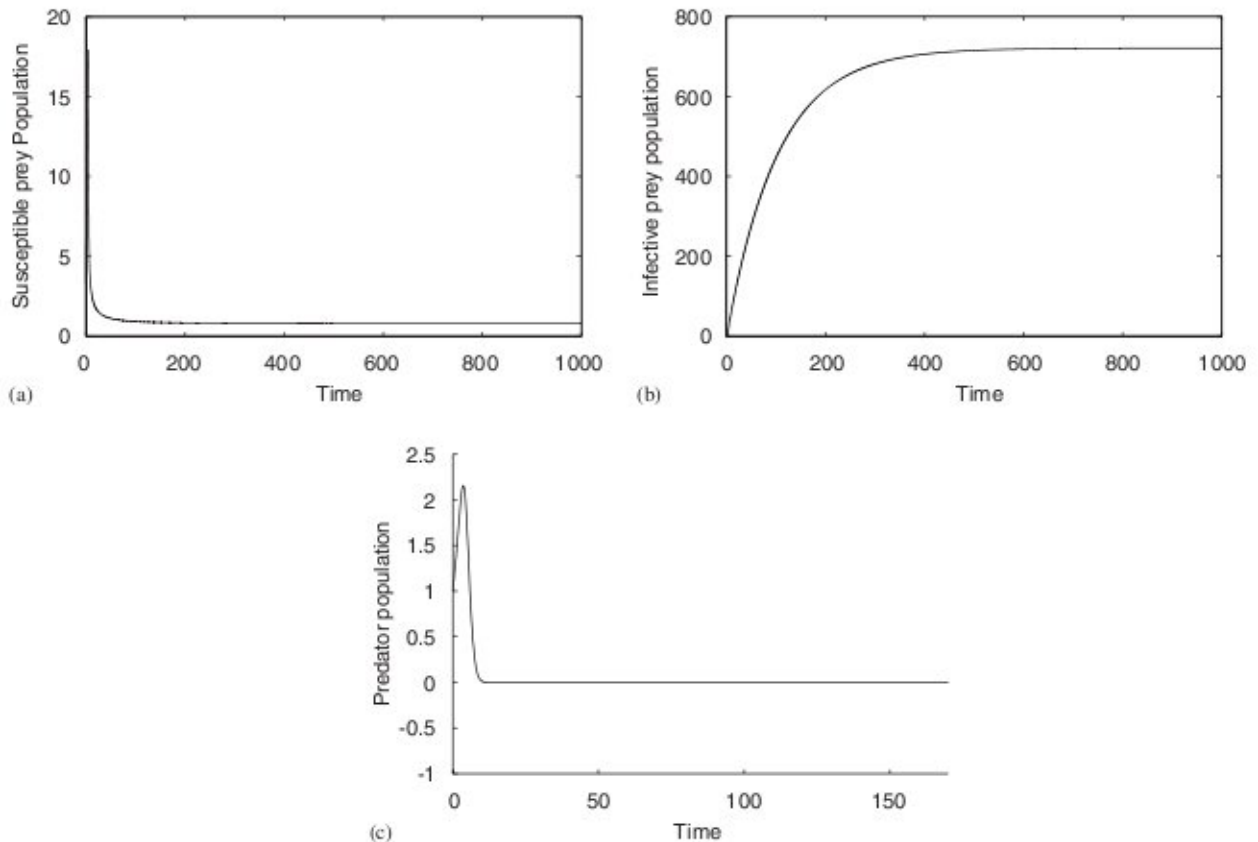


Fig. 3. Time evolution of different populations for model system (2.1).

with negative real part at  $\tau = 0$  which will become positive for  $\tau > \hat{\tau}$ , where  $\hat{\tau}$  is the smallest positive value of  $\hat{\tau}$  given by (4.17). The negative real part of the eigenvalue will continue to be negative for  $\tau < \hat{\tau}$  which is guaranteed by the direct extension of G.J. Butler's Lemma for the three dimensional problem. Therefore, the Hopf bifurcation criterion is satisfied and consequently there exists a small amplitude periodic solution as  $\tau$  passes through  $\hat{\tau}$ .

**Remark 4.2.** It must be pointed out that from the above analysis we can not determine the stability of the bifurcating periodic orbits, that is, the periodic solutions may exist either for  $\tau > \hat{\tau}$  or for  $\tau < \hat{\tau}$ , near  $\hat{\tau}$ .

**Remark 4.3.** If (v) is violated, that is,  $A_1 < 0$ ,  $A_2 < 0$  and  $A_3 > 0$  then by Descartes' rule of signs there exists two positive roots  $v_1$  and  $v_2$  satisfying Eq. (4.13). According to Theorem 3.1 of Kuang [22], the stability of  $E^*$  changes a finite number of times as  $\tau$  is increased and eventually becomes unstable for sufficiently large values of  $\tau$ . This will be clear from the following example.

**Example 4.2 (An epidemic case).** Let us consider the following hypothetical set of parameter values:  $A = 7$  individuals  $\text{ha}^{-1} \text{day}^{-1}$ ,  $r = 0.33 \text{ day}^{-1}$ ,  $k = 40$  individuals  $\text{ha}^{-1}$ ,  $k'_1 = 0.6 \times k_1 \text{ ha per individual day}^{-1}$ ,  $k'_2 = 0.6 \times k_2 \text{ ha per individuals day}^{-1}$ ,  $d = 0.009 \text{ day}^{-1}$ ,  $f = 0.03 \text{ day}^{-1}$ ,  $e = 0.0097 \text{ day}^{-1}$ ,  $b = 0.05 \text{ ha per individual day}^{-1}$ ,  $k_1 = 0.002 \text{ ha per individuals day}^{-1}$ , and  $k_2 = 0.05 \text{ ha per individuals day}^{-1}$ . For this set of parameter values, the coexistence equilibrium  $E^*$  is given by (40.3775, 1.7296, 39.5835). We find that for these parameter values system (2.1), that is, the system without the time lag, becomes unstable about the coexistence equilibrium (the eigenvalues associated with the variational matrix of system (2.2) at  $E^*$  are  $(-21.14, 0.55, -1.62)$ ). Moreover, for this set of parameter values the predator population is washed away from the system (see Fig. 3), and so this is a typical example for occurrence of an epidemic in the system.

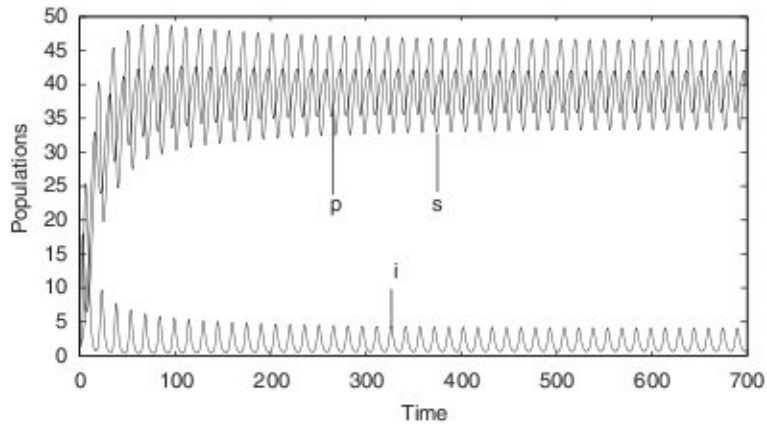


Fig. 4. The figure depicts the time evolution of all populations for system (2.2) for  $\tau = 11$ .

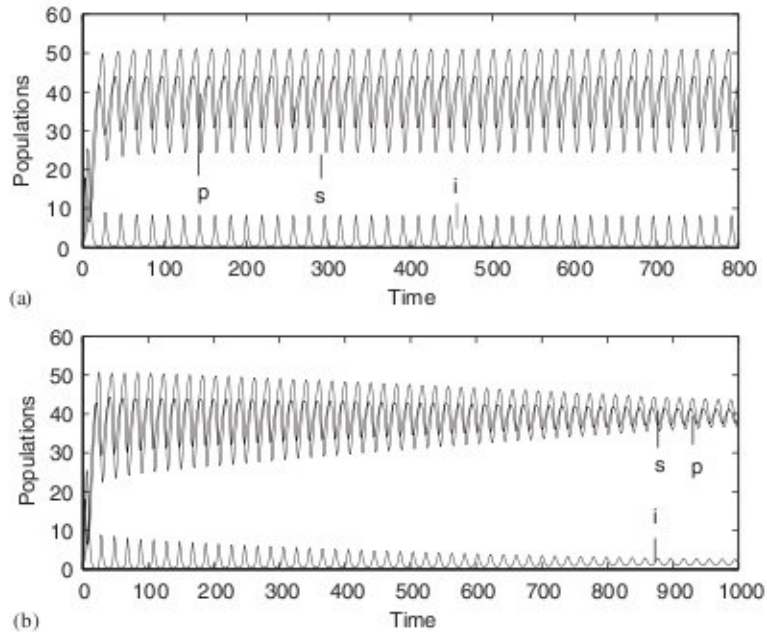


Fig. 5. Time evolution of all populations for model system (2.2). (a) Depicts the populations for  $\tau = 16$  and (b) depicts the populations for  $\tau = 17$ .

From Fig. 3 we observed that there is a remarkable variation in the susceptible and infective prey population level. The susceptible population decline from the initial population of 3.3 individuals  $ha^{-1}$  to 0.794 individuals  $ha^{-1}$ . On the other hand the infective population increases from the initial population of 1.6 individuals  $ha^{-1}$  to 720.87 individuals  $ha^{-1}$ . The huge increase in the infective prey population leads to the extinction of the predator population.

Now we shall find the role of the time lag  $\tau$ . For this we first substitute these values in Eq. (4.13). Substituting these values in Eq. (4.13), we obtain

$$\hat{v}^6 - 112.1430\hat{v}^4 - 9.9616\hat{v}^2 + 0.9488 = 0. \tag{4.18}$$

Solving Eq. (4.18), we get two positive values of  $\hat{v}$ , that is,  $\hat{v}_1 = 0.24028$  and  $\hat{v}_2 = 10.5939$ , and so according to Remark 4.3 there exists two critical values of  $\tau$ ,  $\hat{\tau}_1$  and  $\hat{\tau}_2$ , such that the positive equilibrium  $E^*$  bifurcates to periodic solutions when  $\tau$  lies near  $\hat{\tau}_1$  and  $\hat{\tau}_2$ . We have already seen that the positive steady state  $E^*$  is unstable for  $\tau = 0$ . When we increase the value of  $\tau$  from  $\tau = 0$  to  $\tau = 11$  we observe that the positive steady state  $E^*$  bifurcates into a periodic

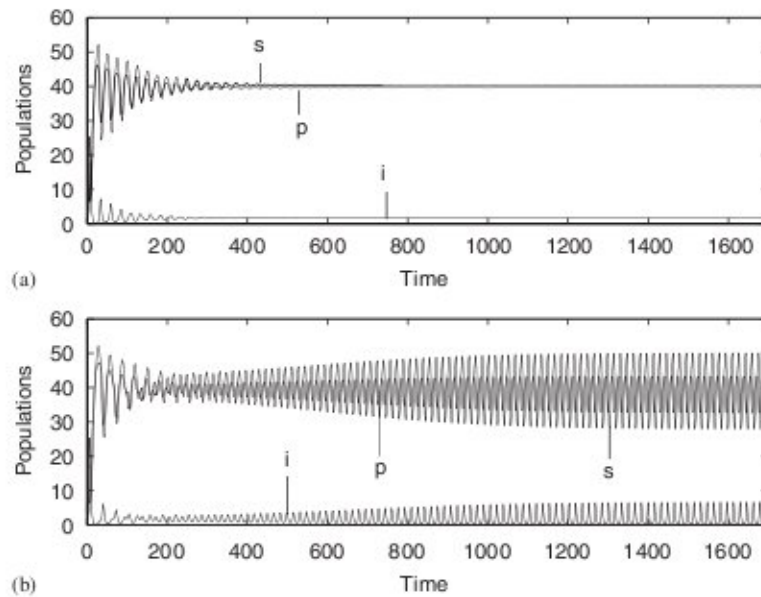


Fig. 6. Time evolution of all populations for model system (2.2). (a) Depicts the populations for  $\tau = 25$  and (b) depicts the populations for  $\tau = 26$ .

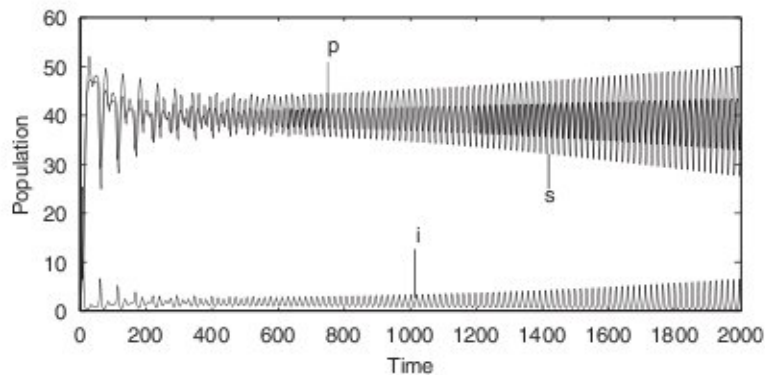


Fig. 7. Unstable solution (with growing oscillations) of model system (2.2) for  $\tau > 30$ .

solution, see Fig. 4. If  $\tau$  is further increased from  $\tau = 16$  to  $\tau = 17$  the positive steady state  $E^*$  becomes a stable focus (with decaying oscillations), see Fig. 5. Hence we see that when  $\tau$  passes through  $\hat{\tau}_1 \simeq 12$ , a small amplitude periodic solution occurs, and the unstable positive steady state  $E^*$  becomes a stable focus. If  $\tau$  is further increased from  $\tau = 25$  to 26 the positive steady state  $E^*$  bifurcates from the stable focus into another periodic solution, (see Fig. 6). Finally, when  $\tau$  is increased beyond  $\tau = 30$  the positive steady state  $E^*$  becomes unstable (by means of a growing oscillation) (Fig. 7). Hence we observe that when  $\tau$  passes through  $\hat{\tau}_2 \simeq 28$  a small amplitude periodic solution occurs and  $E^*$  loses its stability. This suggests that the delay in the gestation of the infective prey population plays an important role in maintaining the stability of the system about the coexistence equilibrium, and helps in preventing an outbreak of the disease. However, the stability of the coexistence equilibrium becomes weaker as  $\tau$  increases and eventually, it becomes unstable for sufficiently large  $\tau$ .

*Probable control of the epidemic:* A control of the epidemic by a careful increase of the value of  $\tau$ , i.e., the time-lag for the gestation of the infective prey, can be a very useful policy, since it works even if we increase the value of the parameter  $b$  (the force of infection), see Fig. 8.



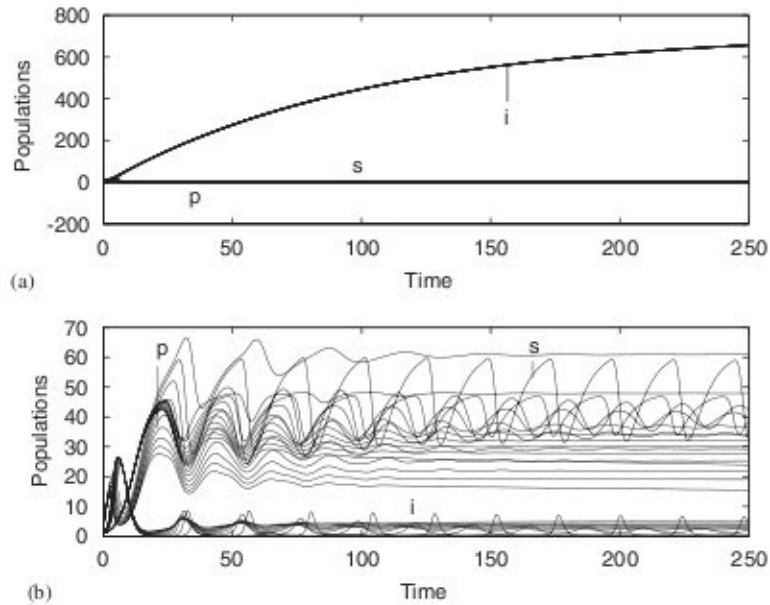


Fig. 8. Time evolution for different values of  $b$  (a) for model system (2.1), (b) for model system (2.2).

In Fig. 8, we vary the value of the parameter  $b$  in the range  $[0.03, 0.09]$ , keeping the other parameters, including  $\tau$ , fixed. In Fig. 8(a) time evolution for system (2.1) is plotted for different values of  $b$  taken from the above range starting from 0.03 to 0.09 with a step size of 0.005. In Fig. 8(b) time evolution for system (2.2) is plotted for same values of  $b$ , as taken for Fig. 8(a). We observe that the gestation of the infective prey population plays an important role in controlling the epidemic in the system.

## 5. Global stability results, permanence

In this section we shall prove the global stability result of the disease free equilibrium and the permanence of the coexistence equilibrium. We begin with the global stability of the disease free equilibrium  $E_2$ . We proved that any solution of Eq. (2.3) starting outside  $\Omega$  (in  $R_+^3$ ) either enters into  $\Omega$  at some finite time, say  $t_0 > 0$ , and then it remains in its interior  $\Omega$  for all  $t > t_0$  or tends to the boundary equilibrium  $E_1$ . But the boundary equilibrium  $E_1$  is always a repeller, see Remark (4.1). It is therefore sufficient to prove that the disease free equilibrium is asymptotically stable (under certain conditions) with respect to  $\text{int } \Omega$  to prove the global stability in  $R_+^3$ .

**Theorem 5.1.** *If the following conditions hold, then all solutions with initial conditions (2.4) of system (2.3) starting in  $\Omega$  approach the disease free equilibrium  $E_2(S_2, 0, P_2)$  as  $t \rightarrow +\infty$ :*

- (i)  $B(\delta + \mu) = \delta^2 S_2$ .
- (ii)  $\sigma B = \delta(\mu + \gamma)$ .
- (iii)  $4\delta > \alpha^2$ .

**Proof.** From Theorem 2.1, we see that the set  $\Omega$  is a global attractor in  $R_{0+}^3$  and, of course it is positively invariant. Let us consider the following Lyapunov function:

$$V(t) = \frac{1}{2}(S(t) - S_2)^2 + I(t) - I_2 - I_2 \log \frac{I(t)}{I_2} + P(t) - P_2 - P_2 \log \frac{P(t)}{P_2}.$$

Taking the time derivative of  $V(t)$  along the solution of (2.3) and because of the positivity of the solutions, we have

$$\begin{aligned}\dot{V} &< (S - S_2)\{B - \delta S + \mu I\} + (P - P_2)\{1 - P + \alpha' S\} + (I - I_2)\{\sigma S - \gamma - \mu\} \\ &= (S - S_2)\{B - \delta(S - S_2) - \delta S_2 + \mu I\} + (P - P_2)\{1 - (P - P_2) - P_2 \\ &\quad + \alpha'(S - S_2) + \alpha' S_2\} + (I - I_2)\{\sigma S - \gamma - \mu\}.\end{aligned}$$

For all the solutions of (2.3) starting in  $\Omega$ , we know that  $S(t) + I(t) \leq \frac{B}{\delta}$  and  $P(t) \leq M$ . Hence, using the existence condition  $1 - P_2 + \alpha' S_2 = 0$ , we have

$$\begin{aligned}\dot{V} &\leq (S - S_2) \left\{ -\delta(S - S_2) + B - \delta S_2 + \frac{\mu B}{\delta} \right\} + (P - P_2)\{-(P - P_2) + \alpha'(S - S_2)\} \\ &\quad + (I - I_2) \left\{ \frac{\sigma B}{\delta} - \gamma - \mu \right\}.\end{aligned}$$

Assuming  $B(\delta + \mu) = \delta^2 S_2$  and  $\sigma B = \delta(\mu + \gamma)$ , we get

$$\dot{V} \leq -\delta(S - S_2)^2 - (P - P_2)^2 + \alpha'(S - S_2)(P - P_2).$$

The right hand side of the above expression can be written as  $-Z^T R Z$ , where  $Z = (S - S_2, P - P_2)$  and the symmetric matrix  $R$  is given by

$$R = \begin{bmatrix} \delta & -\frac{\alpha'}{2} \\ -\frac{\alpha'}{2} & 1 \end{bmatrix}.$$

Now,  $\dot{V} \leq 0$  if the symmetric matrix  $R$  is positive definite.  $R$  is positive definite if  $4\delta > \alpha'^2$ . Hence by LaSalle's extension of Lyapunov theorem (see [15]), the disease free equilibrium is asymptotically stable with respect to  $\text{int } \Omega$ , provided the conditions stated in Theorem 5.1 holds true.  $\square$

In the following we shall prove that the instability of  $E_2$  and  $E_3$  implies that system (2.3) is permanent. Before starting our theorem, we give the following two definitions from Xiao and Chen [29].

**Definition 5.1.** System (2.3) is uniformly persistent if there is an  $\eta > 0$  (independent of the initial data) such that every solution  $(S(t), I(t), P(t))$  with initial condition (2.4) satisfies

$$\liminf_{t \rightarrow +\infty} S(t) \geq \eta, \quad \liminf_{t \rightarrow +\infty} I(t) \geq \eta, \quad \liminf_{t \rightarrow +\infty} P(t) \geq \eta.$$

**Definition 5.2.** System (2.3) is said to be permanent if there exists a compact region  $\Omega_0 \in \text{int } \Omega$  such that every solution of Eq. (2.3) with initial condition (2.4) will eventually enter and remain in region  $\Omega_0$ .

Clearly for a dissipative system uniform persistence is equivalent to permanence.

**Lemma 5.1.** Consider the following equation:

$$\begin{aligned}\frac{dx}{d\tau} &= C - axy - dx, \\ \frac{dy}{d\tau} &= axy - ey.\end{aligned}$$

Existence of the interior equilibrium of the above system ensures its global asymptotic stability.

**Proof.** See [4].  $\square$

**Lemma 5.2.** Consider the following equation:

$$\dot{x}(t) = x(t)(1 - x(t)) - \alpha x(t - \tau), \tag{5.1}$$

where  $\alpha$  is a non-negative constant. Then the trivial steady state of the above system (5.1) is a repeller, provided  $\alpha > 1$ .

**Proof.** Linearizing system (5.1) about the trivial equilibrium, we get

$$\dot{y}(t) = y(t) - \alpha y(t - \tau), \tag{5.1a}$$

where  $y$  is the linearized variable. We have to show that  $u = \liminf_{t \rightarrow +\infty} y(t) = +\infty$ . We will prove this by contradiction. If possible let  $u$  be a positive finite constant. Then there is a sufficiently small  $\varepsilon > 0$ , such that

$$u + \varepsilon < \alpha(u - \varepsilon) \quad (\because \alpha > 1). \tag{5.2}$$

The definition of  $u$  implies that there exists a  $T = T(\varepsilon) > \tau$  such that

$$y(t) > u - \varepsilon \quad \text{for } t > T. \tag{5.3}$$

Let  $\bar{t} > T + \tau$  be a local minimum point of  $y(t)$  such that  $y(\bar{t}) < u + \varepsilon$ . Then  $\dot{y}(\bar{t}) = 0$ . This leads to

$$\alpha y(\bar{t} - \tau) = y(\bar{t}) < u + \varepsilon.$$

By (5.2), we have  $y(\bar{t} - \tau) < u - \varepsilon$ , a contradiction to (5.3). Then  $u = +\infty$ , that is

$$\lim_{t \rightarrow +\infty} y(t) = +\infty.$$

Hence the lemma.  $\square$

**Theorem 5.2.** System (2.3) is permanent provided

- (i)  $\frac{B}{k_1} > \alpha(P_2 + \varepsilon_1) + \delta$ , where  $k_1 = \frac{\beta(P_2 + \varepsilon_1) + \gamma + \mu}{\sigma}$  and  $P_2 = \frac{x - \delta + \sqrt{(\delta - x)^2 + 4x(\delta + Bx')}}{2x}$ ,
- (ii)  $\beta' \lambda(B + \varepsilon_2 \gamma) > \lambda \gamma + \beta' \delta(\gamma + \mu)$ ,

where  $\varepsilon_i, i = 1, 2$  is sufficiently small.

In order to prove Theorem 5.2, we present uniform persistence theory for infinite dimensional systems from [17]. Let  $X$  be a complete metric space. Suppose that  $X^0$  is open and dense in  $X$  and  $X^0 \cup X_0 = X, X^0 \cap X_0 = \Phi$ . Assume that  $T(x)$  is a  $C^0$  semigroup on  $X$  satisfying

$$T(t) : X^0 \longrightarrow X^0, \quad T(t) : X_0 \longrightarrow X_0. \tag{5.4}$$

Let  $T_b(t) = T(t)|_{X_0}$  and let  $A_b$  be the global attractor for  $T_b(t)$ .

**Lemma 5.3.** Suppose that  $T(t)$  satisfies (5.4) and we have the following:

- (i) there is a  $t_0 \geq 0$  such that  $T(t)$  is compact for  $t > t_0$ ;
- (ii)  $T(t)$  is a point dissipative in  $X$ ;
- (iii)  $A_b = \cup_{x \in A_b} \omega(x)$  is isolated and thus has an acyclic covering  $\hat{M}$ , where

$$\hat{M} = \{M_1, M_2, \dots, M_n\};$$

- (iv)  $W^s(M_i) \cap X^0 = \Phi$  for  $i = 1, 2, \dots, n$ .

Then  $X_0$  is a uniform repeller with respect to  $X^0$ , i.e., there is an  $\varepsilon > 0$  such that for any  $x \in X^0$ ,  $\lim_{t \rightarrow +\infty} \inf d(T(t)x, X_0) \geq \varepsilon$ , where  $d$  is the distance of  $T(t)x$  from  $X_0$ .

We are now able to state the proof of Theorem 5.2.

**Proof of Theorem 5.2.** We begin by showing that the boundary planes of  $R_+^3$  repel the positive solutions of system (2.3) uniformly. Let us define

$$C_1 = \{(\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], R_+^3) : \phi_1(\theta) \neq 0, \phi_2(\theta) = 0, \theta \in [-\tau, 0]\},$$

$$C_2 = \{(\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], R_+^3) : \phi_2(\theta) = 0, \phi_1(\theta), \phi_3(\theta) \neq 0, \theta \in [-\tau, 0]\},$$

$$C_3 = \{(\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], R_+^3) : \phi_3(\theta) = 0, \phi_1(\theta), \phi_2(\theta) \neq 0, \theta \in [-\tau, 0]\}.$$

If  $C_0 = C_1 \cup C_2 \cup C_3$  and  $C^0 = \text{int } C([-\tau, 0], R_+^3)$ , it suffices to show that there exists an  $\varepsilon_0 > 0$  such that for any solution  $u_t$  of system (2.3) initiating from  $C^0$ ,  $\liminf_{t \rightarrow +\infty} d(u_t, C_0) \geq \varepsilon$ . To this end, we verify below that the conditions of Lemma 5.3 are satisfied. It is easy to see that  $C^0$  and  $C_0$  are positively invariant. Moreover, conditions (i) and (ii) of Lemma 5.3 are clearly satisfied. Thus we only need to verify conditions (iii) and (iv). There are three constant solutions  $E_1, E_2$  and  $E_3$  in  $C_0$ , corresponding, respectively, to  $(S(t) = \frac{B}{\sigma}, I(t) = 0, P(t) = 0)$ ,  $(S(t) = S_2, I(t) = 0, P(t) = P_2)$  and  $(S(t) = S_3, I(t) = I_3, P(t) = 0)$ . If  $(S(t), I(t), P(t))$  is a solution of system (2.3) initiating from  $C_1$  with  $\phi_1(0) > 0$ , it follows that  $S(t) \rightarrow \frac{B}{\sigma}, I(t) \rightarrow 0, P(t) \rightarrow 0$  as  $t \rightarrow +\infty$ . If  $(S(t), I(t), P(t))$  is a solution of system (2.3) initiating from  $C_2$  with  $\phi_i(0) > 0, i = 1, 3$ , it follows that  $S(t) \rightarrow S_2, I(t) \rightarrow 0, P(t) \rightarrow P_2$  as  $t \rightarrow +\infty$ . If  $(S(t), I(t), P(t))$  is a solution of system (2.3) initiating from  $C_3$  with  $\phi_i(0) > 0, i = 1, 2$ , it follows that  $S(t) \rightarrow S_3, I(t) \rightarrow I_3, P(t) \rightarrow 0$  as  $t \rightarrow +\infty$ . This shows that if invariant sets  $E_1, E_2$  and  $E_3$  are isolated invariant,  $\{E_1, E_2, E_3\}$  are isolated and is an acyclic covering. It is obvious that  $E_1$  is isolated invariant. The isolated invariance of  $E_2$  and  $E_3$  will follow from the following proof.

We now show that  $W^s(E_1) \cap C^0 = \emptyset, W^s(E_2) \cap C^0 = \emptyset$  and  $W^s(E_3) \cap C^0 = \emptyset$ . We restrict our attention to the second and third equations, since the proof for the first is simple.

Assuming the contrary, i.e.,  $W^s(E_2) \cap C^0 \neq \emptyset$ , then there exists a positive solution  $(S(t), I(t), P(t))$  of system (2.3) such that  $(S(t), I(t), P(t)) \rightarrow (S_2, 0, P_2)$  as  $t \rightarrow +\infty$ . Let us choose  $\varepsilon_1 > 0$  small enough such that

$$\frac{B}{k_1} > \alpha(P_2 + \varepsilon_1) + \delta. \tag{5.5}$$

Let  $t_0 > 0$  be sufficiently large such that  $P_2 - \varepsilon_1 < P(t) < P_2 + \varepsilon_1$  for  $t > t_0 - \tau$ . Then we have, for  $t > t_0$

$$\left. \begin{aligned} \frac{dS(t)}{dt} &\geq [B - \sigma I(t)S(t) - (\alpha(P_2 + \varepsilon_1) + \delta)S(t)] \\ \frac{dI(t)}{dt} &\geq I(t)[\sigma S(t) - \beta(P_2 + \varepsilon_1) - \gamma - \mu] \end{aligned} \right\}. \tag{5.6}$$

Let us consider

$$\left. \begin{aligned} \dot{x}_1 &= [B - \sigma x_1(t)x_2(t) - (\alpha(P_2 + \varepsilon_1) + \delta)x_1(t)] \\ \dot{x}_2 &= x_2(t)[\sigma x_1(t) - \beta(P_2 + \varepsilon_1) - \gamma - \mu]. \end{aligned} \right\}. \tag{5.7}$$

Let  $v = (v_1, v_2)$  and let  $\zeta > 0$  be small enough such that  $\zeta v_1 < S(t_0), \zeta v_2 < I(t_0)$ .

If  $(x_1(t), x_2(t))$  is a solution of system (5.7) satisfying  $x_i(t_0) = \zeta v_i, i = 1, 2$ , we know from comparison theorem (see [29])  $S(t) \geq x_1(t), I(t) \geq x_2(t)$  for all  $t > t_0$ . It is easy to know that system (5.7) has a unique positive equilibrium

$$(x_1^*, x_2^*) = \left( k_1, \frac{1}{\sigma} \left( \frac{B}{k_1} - \alpha(P_2 + \varepsilon_1) - \delta \right) \right), \quad k_1 = \frac{\beta(P_2 + \varepsilon_1) + \gamma + \mu}{\sigma},$$

which is globally asymptotically stable, from Lemma 5.1. Note that  $S(t) \geq x_1(t), I(t) \geq x_2(t)$  for all  $t > t_0$  and  $\lim_{t \rightarrow +\infty} x_2(t) = x_2^*$ . This is a contradiction. Hence  $W^s(E_2) \cap C^0 = \emptyset$ .

Let  $W^s(E_3) \cap C^0 \neq \emptyset$ . Then there exists a positive solution  $(S(t), I(t), P(t))$  of system (2.3) such that  $(S(t), I(t), P(t)) \rightarrow (S_3, I_3, 0)$  as  $t \rightarrow +\infty$ .

Let us choose  $\varepsilon_2 > 0$  small enough such that  $\beta'(I_3 + \varepsilon_2) > 1$ . That is,

$$\beta' \lambda (B + \varepsilon_2 \gamma) > \lambda \gamma + \beta' \delta (\gamma + \mu). \tag{5.8}$$

Let  $t_1 > 0$  be sufficiently large such that

$I_3 - \varepsilon_2 < I(t) < I_3 + \varepsilon_2$  for  $t > t_1 - \tau$ . Then we have, for  $t > t_1$

$$\frac{dP(t)}{dt} \geq P(1 - P) - \beta'(I_3 + \varepsilon_2)P(t - \tau). \tag{5.9}$$

Now let us consider

$$\dot{z} = z(1 - z) - \beta'(I_3 + \varepsilon_2)z(t - \tau). \tag{5.10}$$

Let  $u_1$  and let  $\zeta > 0$  be small enough such that  $\zeta u_1 < P(t_1)$ .

If  $z_1$  is a solution of system (5.10) satisfying  $z_1(t_1) = \zeta u_1$ , we know from comparison theorem (see [29]),  $P(t) \geq z_1(t)$  for all  $t > t_1$ . From Lemma 5.2, we observe that the trivial solution of Eq. (5.10) is a repeller ( $\because \beta'(I_3 + \varepsilon_2) > 1$ ).

Note that  $P(t) \geq z_1(t)$  for all  $t > t_1$  and so  $\lim_{t \rightarrow +\infty} P(t) \rightarrow 0$ . This is a contradiction. Hence  $W^s(E_3) \cap C^0 = \Phi$ . At this time, we are able to conclude from Lemma 5.3 that  $C_0$  repels the positive solutions of (2.3) uniformly. Incorporating the above into Lemmas 2.1 and 2.2, we know that system (2.3) is permanent.

### 6. Discussion

In this paper we have attempted to study the effect of a time lag in the model proposed by Chatterjee and Chattopadhyay [4]. It was interesting to compare the three species eco-epidemiological model analyzed here, which has a fixed delay corresponding to the gestation period, with the analogous model analyzed by Chatterjee and Chattopadhyay. Here we have also pointed out the well-known phenomenon of ‘exchange of stability’ through a simple bifurcation.

It was observed that the stability of the disease free equilibrium does not depend on the time lag due to the gestation of the infective prey (see Remark 4.1). We also found a sufficient condition for the stability of  $E^*$ , (see Section (4.1)). Moreover, we have also found the maximum value (length of delay) of  $\tau$  (i.e.,  $\tau_+$ ) for which a locally asymptotically stable interior equilibrium  $E^*$  will remain asymptotically stable, where  $\tau_+ = \frac{1}{2A_0}(-B_0 + \sqrt{B_0^2 + 4A_0C_0})$  (see Theorem 4.2).

Further, in Section 4.3 we found conditions for the bifurcation of the interior positive steady state. We observed both analytically and numerically that if  $A_1 > 0$  and  $A_3 > 0$ , then a stable interior equilibrium  $E^*$  remains stable for all  $\tau > 0$  (see Theorem 4.3). But, if the interior equilibrium  $E^*$  is unstable at  $\tau = \tau_0$  and  $A_1 > 0$  and  $A_3 < 0$ , then  $E^*$  remains unstable for all  $\tau > \tau_0$  (see Theorem 4.4). It was also observed that the positive equilibrium enters a Hopf bifurcation and consequently there exists a small amplitude periodic solution as  $\tau$  passes through  $\hat{\tau}$ , where  $\hat{\tau}$  is given by Eq. (4.17) for  $n = 0$ . However if  $A_1 < 0$ ,  $A_2 < 0$  and  $A_3 > 0$ , then there exists positive  $\hat{v}_1$  and  $\hat{v}_2$  satisfying Eq. (4.13), and consequently there exists two critical values of  $\tau$  i.e.,  $\tau_1$  and  $\tau_2$  around which small amplitude periodic solutions may occur.

We have also observed that system (2.3) is permanent provided (i)  $\frac{B}{k_1} > \alpha(P_2 + \varepsilon_1) + \delta$ , where  $k_1 = \frac{\beta(P_2 + \varepsilon_1) + \gamma + \mu}{\sigma}$  and  $P_2 = \frac{x - \delta + \sqrt{(\delta - x)^2 + 4\alpha(\delta + Bx')}}{2x}$ , (ii)  $\beta' \lambda (B + \varepsilon_2 \gamma) > \lambda \gamma + \beta' \delta (\gamma + \mu)$ , where  $\varepsilon_i$ ,  $i = 1, 2$ , is sufficiently small. Moreover if the conditions stated in Theorem 5.1 hold, then the disease free equilibrium is globally stable. It should be noted here that this is also independent of the value of  $\tau$ . Thus, we may conclude that the time lag does not play any role in making the system disease free.

Finally from our numerical simulations, we find two critical values  $\hat{\tau}_1 \simeq 12$  and  $\hat{\tau}_2 \simeq 28$  such that the positive equilibrium  $E^*$  bifurcates into small amplitude periodic solutions, supporting our analytical claim. We have observed from Figs. 4 and 5 that when  $\tau$  passes through  $\hat{\tau}_1$  the unstable positive equilibrium becomes stable through a small amplitude periodic solution, while, from Figs. 6 and 7, we have observed that when  $\tau$  passes through  $\hat{\tau}_2$ , a stable positive equilibrium bifurcates into a periodic solution and finally becomes unstable. This suggests that the delay in the gestation of the infective prey population plays an important role in maintaining the stability of the coexistence equilibrium and helps to control the outbreak of the disease, (see Fig. 8). But the stability of the positive equilibrium becomes weaker as  $\tau$  increases, and eventually it becomes unstable for sufficiently large  $\tau$ , supporting the well-known phenomenon of ‘exchange of stability’ through simple bifurcation.

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## References

- [1] M.E. Alexander, S.M. Moghadas, Periodicity in an epidemic model with a generalized non-linear incidence, *Math. Biosci.* 189 (2004) 75–96.
- [2] I. Barbalat, Systemes d'equations differentielles d'oscillation non lineares, *Rev. Math. Pures Appl.* 4 (1959) 267.
- [3] M. Biendo, G. Laurans, D. Thomas, O. Dechepy, F. Hamdad-Daoudi, B. Canarelli, F. Eb, Regional dissemination of *Salmonella enterica* serovar Enteritidis is season dependent, *Clin. Microbiol. Infect.* 9 (5) (2003) 360–369.
- [4] S. Chatterjee, J. Chattopadhyay, Role of migratory bird population in an simple eco-epidemiological model, *Math. Comp. Model. Dyn. Syst.*, in press.
- [5] J. Chattopadhyay, O. Arino, A predator–prey model with disease in the prey, *Nonlin. Anal.* 36 (1999) 747–766.
- [6] J. Chattopadhyay, N. Bairagi, Pelican at risk in Salton Sea—an ecoepidemiological model, *Ecol. Modell.* 136 (2001) 103–112.
- [7] J. Chattopadhyay, P.D.N. Srinivasu, N. Bairagi, Pelican at risk in Salton Sea—an ecoepidemiological model—II, *Ecolog. Modell.* 167 (2003) 199–211.
- [8] P.Y. D'Aoust, D.G. Busby, L. Ferns, J. Goltz, S. McBumey, C. Poppe, et al., Salmonellosis in songbirds in the Canadian Atlantic provinces during winter–summer 1997–1998, *Can. Vet. J.* 41 (2000) 54–60.
- [9] M. Fan, Y. Michael, Li., Wang, Ke., Global stability of an SEIS epidemic model with recruitment and a varying total population size, *Math. Biosci.* 170 (2001) 199–208.
- [10] H.I. Freedman, L.H. Erbe, V.S.H. Rao, Three species food chain models with mutual interference and time delays, *Math. Biosci.* 80 (1986) 57–80.
- [11] H.I. Freedman, V.S.H. Rao, The trade-off between mutual interference and time lags in predator prey systems, *Bull. Math. Biol.* 45 (1983) 991.
- [12] A.E. Garmendia, H.J.V. Kruiningen, R.A. French, J.F. Anderson, T.G. Andreadis, A. Kumar, A.B. West, Recovery and identification of West Nile Virus, *J. Clin. Microbiol.* 38 (8) (2000) 3110–3111.
- [13] K. Gopalsamy, *Stability and Oscillations in Delay Differential Equations of Population dynamics*, Kluwer Academic Publishers, MA, 1992.
- [14] K.P. Hadeler, H.I. Freedman, Predator–prey population with parasite infection, *J. Math. Biol.* 27 (1989) 609–631.
- [15] J.K. Hale, *Ordinary Differential Equations*, Wiley-Interscience, New York, 1969.
- [17] J.K. Hale, P. Waltman, Persistence in infinite-dimensional, *SIAM J. Math. Anal.* 20 (1989) 388.
- [18] H.W. Hethcote, The mathematics of infectious diseases, *SIAM Rev.* 42 (2000) 599–653.
- [19] H.W. Hethcote, W. Wang, L. Han, M. Zhién, A predator–prey model with infected prey, *Theor. Pop. Biol.* 66 (2004) 259–268.
- [20] J.M. Hethcote, J.M. Lorca, Dynamic models of infectious disease as regulators of population sizes, *J. Math. Biol.* 30 (1992) 693–716.
- [21] P.J. Hudson, *Grouse in Space and Time*, Fordingbridge, Hampshire, Game Conservancy Ltd., 1992.
- [22] Y. Kuang, *Delay Differential Equations with Application in Population Dynamics*, Academic Press, New York, 1993.
- [23] M. Malkinson, C. Banet, Y. Weisman, S. Pokamunski, R. King, Introduction of West Nile Virus in the Middle East by migrating white Storks, *Emerging Infect. Dis.* 8 (4) (2002) 392–397.
- [24] J.H. Rappole, S.R. Derrickson, Z. Hubalek, Migratory birds and spread of West Nile Virus in the Western Hemisphere, *Emerging Infect. Dis.* 6 (4) (2000) 319–328.
- [25] A. Sih, P. Crowley, M. McPeck, J. Petranka, K. Strohmeier, Predation, competition and prey communities: a review of field experiments, *Ann. Rev. Ecol. Syst.* 16 (1985) 269–311.
- [26] E. Venturino, Epidemics in predator–prey models: disease in prey, in: O. Arino, D. Axelrod, M. Kimmel, M. Langlais (Eds.), *Theory of Epidemics*, *Math. Pop. Dynm.: Analysis of Heterogeneity*, vol. 1, 1995, pp. 381–393.
- [27] E. Venturino, The effect of disease on competing species, *Math. Biosci.* 174 (2001) 111–131.
- [28] M.J. Wonham, T. de-Camino-Beck, M.A. Lewis, An epidemiological model for West Nile Virus: invasion analysis and control applications, *Proc. R. Soc. Lond. B* 271 (2004) 501–507.
- [29] Y. Xiao, L. Chen, Modeling and analysis of a predator–prey model with disease in the prey, *Math. Biosci.* 171 (2001) 59–82.
- [30] X. Yang, L.S. Chen, J.F. Chen, Permanence and positive periodic solution for the single species nonautonomous delay diffusive model, *Comp. Math. Appl.* 32 (1996) 109.