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Stability and Hopf Bifurcation of a Delay Eco-Epidemiological Model with Nonlinear Incidence Rate^{*}

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Abstract. In this paper, a three-dimensional eco-epidemiological model with delay is considered. The stability of the two equilibria, the existence of Hopf bifurcation and the permanence are investigated. It is found that Hopf bifurcation occurs when the delay τ passes a sequence of critical values. Moreover, by applying Nyquist criterion, the length of delay is estimated for which the stability continues to hold. Numerical simulation with a hypothetical set of data has been done to support the analytical results.

Keywords: predator-prey model, eco-epidemiology, delay, Hopf bifurcation.

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1 Introduction

The mathematical modelling of epidemics has become a very important subject of research after the pioneering work of Kermac and Mckendrick (1927) on SIRS

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(susceptible-infected-removed-susceptible) systems, in which the evolution of a disease which gets transmitted upon contact is described. Important studies in the next decades have been carried out, with the aim of controlling the effects of diseases and of developing suitable vaccination strategies (Hethcote, [17]; Wang and Ma, [34]; Liu et al., [27]; Bhunu and Garira, [4]; Magombedze et al., [28]; Samanta, [31]). After the seminal models of Vito Volterra and Alfred James Lotka in the mid 1920s for predator-prey interactions, mutualist and competitive mechanisms have been studied extensively in the recent years by researchers (Hsu et al., [20]; Kuang et al., [24]; Hwang,[23]).

In the natural world, however, species do not exist alone. It is of more biological significance to study the persistence-extinction threshold of each population in systems of two or more interacting species subjected to parasitism. Mathematical biology, namely predator-prey systems and models for transmissible diseases are major fields of study in their own right. But little attention has been paid so far to merge these two important areas of research (Haque M. and Venturino E., [14]; Haque M. and Venturino E., [15]; Greenhalgh D. and Haque M., [11]; Liu M.X., Jin Z. and Haque M., [26]; Venturino E. [33]; Xiao and Chen, [35, 36]; Hethcote, Wang et al., [18]). In order to study the influence of disease on an environment, two or more interacting species should be presented. In this paper, we shall focus on such an eco-epidemiological system consisting of three species, namely, the sound prey (which is susceptible), the infected prey (which becomes infective by some viruses) and the predator population. We will be concerned with the effect of time delay and a nonlinear incidence rate on the dynamics of an eco-epidemiological model.

We have two populations: the prey, whose total population density is denoted by N(t) and the predator, whose population density is denoted by P(t). We make the following assumptions:

We make the following assumptions:

 (A_1) In the absence of infection and predation, the prey population density grows logistically with carrying capacity K > 0 and an intrinsic birth rate constant (r > 0) (Greenhalgh D. and Haque M., [11]; Haque M. and Venturino E., [14])

$$\frac{dS(t)}{dt} = rS(t)\left(1 - \frac{S(t)}{K}\right). \tag{1.1}$$

- (A₂) In the presence of disease, the total prey population N(t) is divided into two distinct classes, namely, susceptible population S(t) and infected population I(t) (Greenhalgh D. and Haque M., [11]; Haque M. and Venturino E., [14]). Therefore, at any time t, the total density of prey population is N(t) = S(t) + I(t).
- (A_3) We assume that only susceptible prey S(t) are capable of reproducing with logistic law (1.1); i.e., the infected prey I(t) are removed by death (say its death rate is a positive constant μ), or by predation if only susceptible preys can reproduce, then infected prey have no possibility of reproducing. However, the infective population I(t) still contributes with S(t) to population growth toward the carrying capacity (Greenhalgh D. and Haque M., [11]; Haque M. and Venturino E., [14]).

 (A_4) Capasso and Serio [6] introduced a saturated incidence rate q(I(t))S(t)into epidemic models, where q(I(t)) tends to a saturation level when I(t)gets large, i.e., $q(I(t)) = \beta I(t)/(1 + \alpha I(t))$, where $\beta I(t)$ measures the infection force of the disease and $1/(1 + \alpha I(t))$ measures the inhibition effect from the behavioural change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate $\beta S(t)I(t)$, because it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters. And we are also concerned with the effect of time delay and a nonlinear incidence rate on the dynamics of the SI epidemic model. Hence, we assume that the force of infection at time t is given by $\beta S(t)I(t-\tau)/(1+\alpha I(t-\tau))$, where β is the average number of contacts per infective per day, α is half saturation constant for infection and $\tau > 0$ is a fixed time during which the infectious agents develop in the vector and it is only after the time that the infected vector can infect a susceptible prey (Xu and Ma, [37, 38]). Hence, the SI model of the infected prey is:

$$\begin{cases} \frac{dS(t)}{dt} = rS(t)\left(1 - \frac{S(t)}{K}\right) - \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}\\ \frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)} - dI(t). \end{cases}$$

 (A_5) Numerous field studies show that infected prey are more vulnerable to predation compared with their non-infected counterpart (Hudson et al., [21]; Lafferty et al., [25]; Murray et al., [29]). Lafferty and Morris [25] quantified that the predation rates on infected prey may be 31 times higher compared to that on susceptible prey. Thus, we consider the case when the predator mainly eats the infected prey with Leslie-Gower ratiodependent schemes (Song and Li, [32]; Nindjin et al., [30]; Aziz-Alaoui and Daher Okiye, [2]; Hsu and Hwang, [19]; Aziz-Alaoui, [1]; Greenhalgh D. and Haque M., [11]). That is to say, the predator consumes the prey according to the ratio-dependent functional response and the predator grows logistically with intrinsic growth rate δ and carrying capacity proportional to the prey populations size I(t).

From the above assumptions we obtain the following model:

$$\begin{cases} \frac{dS(t)}{dt} = rS(t)(1 - \frac{S(t)}{K}) - \frac{\beta S(t)I(t - \tau)}{1 + \alpha I(t - \tau)}, \\ \frac{dI(t)}{dt} = \frac{\beta S(t)I(t - \tau)}{1 + \alpha I(t - \tau)} - \frac{cP(t)I(t)}{mP(t) + I(t)} - dI(t), \\ \frac{dP(t)}{dt} = \delta P(t)(1 - \frac{hP(t)}{I(t)}), \end{cases}$$
(1.2)

where S(t) is the density of the susceptible prey population, I(t) is the density of the infective prey population, P(t) is the density of their predator population at any time t. In system (1.2), r is the intrinsic birth rate of the sound prey,

K is the carrying capacity of the sound prey, β is the infection rate, α is the half saturation constant of infection, d is the death rate of the infected prey, c is the search rate, m is the half capturing saturation constant, δ is the intrinsic growth rate of the predator, h is the maximum value of the per capita reduction rate of prey due to predator. All the parameters are positive.

Although there are several eco-epidemiological models with the disease in the prey (Liu M.X. Jin Z. and Haque M., [26]; Xiao Y.N. and Chen L.S., [36]), the novelty of the model (1.2) is that it includes both the delay and the non-linear incidence rate. In addition, there are some common diseases in popular vegetables where infected vegetables are avoided by their predator (Haque M. and Greenhalgh D., [13]). The initial conditions for system (1.2) take the form

$$S(\theta) = \varphi_1(\theta), \quad I(\theta) = \varphi_2(\theta), \quad P(\theta) = \varphi_3(\theta),$$

$$\varphi_1(\theta) \ge 0, \quad \varphi_2(\theta) \ge 0, \quad \varphi_3(\theta) \ge 0, \quad \theta \in [-\tau, 0],$$

$$\varphi_1(0) > 0, \quad \varphi_2(0) > 0, \quad \varphi_3(0) > 0,$$

(1.3)

where $(\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta)) \in C([-\tau, 0], R^3_{+0})$, the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into R^3_{+0} , where $R^3_{+0} = \{(x_1, x_2, x_3) : x_i \geq 0, i = 1, 2, 3\}$. It is well known by the fundamental theorem of functional differential equations that system (1.2) has a unique solution (S(t), I(t), P(t)) satisfying initial conditions (1.3).

The paper is organised as follows. In Section 2, we prove the positivity and the boundedness of solutions. We find conditions for local stability and present bifurcation results in Section 3. In Section 4, the time delay is estimated for which local stability is preserved. The permanence of system is proved in Section 5. Some numerical simulations are performed for hypothetical values of parameters and results are given in Section 6.

2 Positivity and Boundedness of Solutions

It is important to show the positivity and boundedness of the solution of system (1.2) as it represents populations. Positivity implies not only that the population survives, but also that the population extinguishes when the density decreases to 0. Boundedness may be interpreted as a natural restriction to growth as a consequence of limited resources. The model system can be put into the matrix form $\dot{X} = G(X)$, where $X = (S, I, P)^{\top} \in \mathbb{R}^3$ and G(X) is given by

$$G(X) = \begin{pmatrix} G_1(X) \\ G_2(X) \\ G_3(X) \end{pmatrix} = \begin{pmatrix} rS(1 - \frac{S}{K}) - \frac{\beta SI(t - \tau)}{1 + \alpha I(t - \tau)} \\ \frac{\beta SI(t - \tau)}{1 + \alpha I(t - \tau)} - \frac{cPI}{mP + I} - dI \\ \delta P(1 - hP/I) \end{pmatrix}$$

Let $R_+^3 = [0, +\infty) \times [0, +\infty) \times [0, +\infty)$ be the nonnegative octant in R^3 , the $G: R_+^3 \to R^3, G \in C^{\infty}(R^3)$ (where G is a function of the variable $X \in R_+^3$) is locally Lipschitz and satisfies the condition $G_i(X)|_{X_i(t)=0, X \in R_+^3} \ge 0$, where $X_1 = S, X_2 = I, X_3 = P$.

Due to Lemma 2 in Yang et al., [39], any solution of (1.2) with positive initial conditions exist uniquely and each component of X remains in the interval $[0, \bar{b})$ for some $\bar{b} > 0$. Furthermore, if $\bar{b} < +\infty$ then

$$\lim_{t \to +\infty} \sup_{t \to +\infty} [S(t) + I(t) + P(t)] = +\infty.$$

Next, the boundedness of solutions will be presented. Since

$$\frac{dS}{dt} \le rS\Big(1 - \frac{S}{K}\Big),$$

by a standard comparison theorem, we have $\limsup_{t \to +\infty} S(t) \leq M_1$, where $M_1 = \max\{S(0), K\}$. Define the function W(t) = S(t) + I(t). For any positive constant ϵ , the time derivative of W(t) along a solution of (1.2) is

$$\frac{dW}{dt} = rS\left(1 - \frac{S}{K}\right) - \frac{cPI}{mP + I} - dI$$
$$\leq S\left(r + \epsilon - \frac{rS}{K}\right) - qW(t) \leq \frac{K(r + \epsilon)^2}{4r} - qW(t),$$

where $q = \min\{\varepsilon, d\}$. Thus, $\frac{dW}{dt} + qW \leq \frac{K(r+\epsilon)^2}{4r}$. Applying a theorem in differential inequalities, we obtain

$$W(t) \le \frac{K(r+\epsilon)^2}{4rq} + \left[W(S(0), I(0)) - \frac{K(r+\epsilon)^2}{4rq}\right]e^{-qt}.$$

Hence, $\limsup_{t \to \infty} W(t) \leq \frac{K(r+\epsilon)^2}{4r}$. Therefore, there exist $M_2 > 0$ and some $T_1 > 0$ such that $I(t) \leq M_2, t \geq T_1$.

Lastly, we consider the boundedness of P(t). From the third equation of system (1.2), we get

$$\frac{dP}{dt} \le \delta P \left(1 - \frac{hP}{M_2} \right).$$

By a standard comparison theorem, we have $\limsup_{t\to+\infty} P(t) \leq M_3$, where $M_3 = \max\{P(0), M_2/h\}$. So, all solutions of system (1.2) with initial condition enter the region

$$B = \{ (S(t), I(t), P(t)) : 0 \le S(t) \le M_1, 0 \le I(t) \le M_2, 0 \le P(t) \le M_3 \}.$$

3 Stability Analysis and Hopf Bifurcation

In this section, we focus our analysis on the stability of the equilibria and Hopf bifurcation of the positive equilibrium of the system (1.2). System (1.2) has the boundary equilibrium $E_1(S_1, I_1, 0)$, where $S_1 = \frac{d}{\beta}(1 + \alpha I_1)$ and I_1 is the positive root of

$$rd\alpha^2 I^2 + (K\beta^2 + 2rd\alpha - Kr\alpha\beta)I + rd - Kr\beta = 0.$$

Obviously, $I_1 > 0$ if $K\beta > d$. And

$$I_1 = \frac{-(K\beta^2 + 2rd\alpha - Kr\alpha\beta) + \sqrt{(K\beta^2 + 2rd\alpha - Kr\alpha\beta)^2 - 4rd\alpha^2(rd - Kr\beta)}}{2rd\alpha^2}$$

System (1.2) has the positive equilibrium $E_2(S_2, I_2, P_2)$, where $P_2 = I_2/h$, $S_2 = (d+c/m+h)(1+\alpha I_2)/\beta$, and I_2 is the positive equilibrium which is given by the following equation $\varpi_1 I^2 + \varpi_2 I + \varpi_3 = 0$, where $\varpi_1 = r(d+c/m+h)\alpha^2$,

$$\varpi_2 = K\beta^2 + 2r(d + \frac{c}{m+h})\alpha - Kr\alpha\beta, \quad \varpi_3 = r(d + \frac{c}{m+h}) - Kr\beta$$

Clearly, E_2 is positive if $\varpi_3 > 0$, i.e.,

$$\beta > \frac{1}{K} \left(d + \frac{c}{m+h} \right)$$
 and $I_2 = \frac{-\overline{\omega}_2 + \sqrt{\overline{\omega}^2 - 4\overline{\omega}_1 \overline{\omega}_3}}{2\overline{\omega}_1}$

Let $E^{\ast}(S^{\ast},I^{\ast},P^{\ast})$ be any arbitrary equilibrium. Then the characteristic equation about E^{\ast} is given by

$$\begin{vmatrix} r - \frac{2rS^*}{K} - \frac{\beta I^*}{1 + \alpha I^*} - \lambda & -\frac{\beta S^* e^{-\lambda \tau}}{(1 + \alpha I^*)^2} & 0\\ \frac{\beta I^*}{1 + \alpha I^*} & \frac{\beta S^* e^{-\lambda \tau}}{(1 + \alpha I^*)^2} - \frac{cmP^{*2}}{(mP^* + I^*)^2} - d - \lambda & -\frac{cI^{*2}}{(mP^* + I^*)^2}\\ 0 & \frac{\delta hP^{*2}}{I^{*2}} & \delta - \frac{2\delta hP^*}{I^*} - \lambda \end{vmatrix} = 0.$$
(3.1)

For equilibrium E_1 , (3.1) reduces to

$$\begin{vmatrix} -\frac{rS_1}{K} - \lambda & -\frac{\beta S_1}{(1+\alpha I_1)^2} e^{-\lambda\tau} & 0\\ \frac{\beta I_1}{1+\alpha I_1} & \frac{\beta S_1}{(1+\alpha I_1)^2} e^{-\lambda\tau} - d - \lambda & -c\\ 0 & 0 & \delta - \lambda \end{vmatrix} = 0.$$
(3.2)

It is clear that (3.2) has the characteristic root $\lambda = \delta > 0$. Hence, the equilibrium E_1 is unstable.

$$\begin{aligned} \text{Theorem 1. If } (d + \frac{c}{m+h})(1 - \frac{1}{1+\alpha I_2}) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} > 0 \ and \\ & \Big[\Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) \Big(\delta + \frac{rS_2}{K} \Big) - \frac{rS_2}{K} \frac{ch}{(m+h)^2} \Big] \Big[\Big(d + \frac{c}{m+h} \Big) \\ & \times \Big(1 - \frac{1}{1+\alpha I_2} \Big) + \Big(\delta + \frac{rS_2}{K} \Big) - \frac{ch}{(m+h)^2} \Big] + \frac{r\delta S_2}{K} \Big(\delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \Big) \\ & + \frac{\beta^2 S_2 I_2}{(1+\alpha I_2)^3} \Big[\Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \Big] > 0, \end{aligned}$$

then the positive equilibrium E_2 is locally asymptotically stable when $\tau = 0$.

Proof. For equilibrium E_2 , (3.1) reduces to

$$\begin{vmatrix} -\frac{rS_2}{K} - \lambda & -\frac{\beta S_2 e^{-\lambda \tau}}{(1 + \alpha I_2)^2} & 0\\ \frac{\beta I_2}{1 + \alpha I_2} & \frac{\beta S_2 e^{-\lambda \tau}}{(1 + \alpha I_2)^2} - \frac{cmP_2^2}{(mP_2 + I_2)^2} - d - \lambda & -\frac{cI_2^2}{(mP_2 + I_2)^2}\\ 0 & \delta/h & -\delta - \lambda \end{vmatrix} = 0,$$

i.e.,

$$\Delta(\lambda,\tau) = \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 + (B_1\lambda^2 + B_2\lambda + B_3)e^{-\lambda\tau} = 0, \quad (3.3)$$

where

$$\begin{split} A_1 &= \delta + d + \frac{cmP_2^2}{(mP_2 + I_2)^2} + \frac{rS_2}{K} = \delta + d + \frac{cm}{(m+h)^2} + \frac{rS_2}{K}, \\ A_2 &= \delta \Big(d + \frac{cmP_2^2}{(mP_2 + I_2)^2} \Big) + \frac{rS_2}{K} \Big(\delta + d + \frac{cmP_2^2}{(mP_2 + I_2)^2} \Big) + \frac{\delta}{h} \frac{cI_2^2}{(mP_2 + I_2)^2} \\ &= \delta \Big(d + \frac{cm}{(m+h)^2} \Big) + \frac{rS_2}{K} \Big(\delta + d + \frac{cm}{(m+h)^2} \Big) + \frac{\delta ch}{(m+h)^2}, \\ A_3 &= \frac{rS_2}{K} \Big[\delta \Big(d + \frac{cmP_2^2}{(mP_2 + I_2)^2} \Big) + \frac{\delta}{h} \frac{cI_2^2}{(mP_2 + I_2)^2} \Big] + \frac{rS_2}{K} \frac{\delta ch}{(m+h)^2} \\ &= \frac{rS_2}{K} \Big[\delta \Big(d + \frac{cm}{(m+h)^2} \Big) + \frac{\delta ch}{(m+h)^2} \Big] + \frac{rS_2}{K} \frac{\delta ch}{(m+h)^2}, \\ B_1 &= -\frac{\beta S_2}{(1+\alpha I_2)^2}, \quad B_2 &= -\frac{\delta \beta S_2}{(1+\alpha I_2)^2} - \frac{r\beta S_2^2}{K(1+\alpha I_2)^2} + \frac{\beta^2 S_2 I_2}{(1+\alpha I_2)^3}, \\ B_3 &= -\frac{r\delta \beta S_2^2}{K(1+\alpha I_2)^2} + \frac{\delta \beta^2 S_2 I_2}{(1+\alpha I_2)^3}. \end{split}$$

For $\tau = 0$, the transcendental equation (3.3) reduces to

$$\lambda^3 + (A_1 + B_1)\lambda^2 + (A_2 + B_2)\lambda + A_3 + B_3 = 0.$$
(3.4)

We can easily get

$$A_{1} + B_{1} = \left(d + \frac{c}{m+h}\right) \left(1 - \frac{1}{1+\alpha I_{2}}\right) + \delta + \frac{rS_{2}}{K} - \frac{ch}{(m+h)^{2}},$$

$$A_{2} + B_{2} = \left(d + \frac{c}{m+h}\right) \left(1 - \frac{1}{1+\alpha I_{2}}\right) \left(\delta + \frac{rS_{2}}{K}\right)$$

$$+ \frac{r\delta S_{2}}{K} - \frac{rS_{2}}{K} \frac{ch}{(m+h)^{2}} + \frac{\beta^{2}S_{2}I_{2}}{(1+\alpha I_{2})^{3}},$$

$$A_{3} + B_{3} = \frac{r\delta S_{2}}{K} \left(d + \frac{c}{m+h}\right) \left(1 - \frac{1}{1+\alpha I_{2}}\right) + \frac{\delta\beta^{2}S_{2}I_{2}}{(1+\alpha I_{2})^{3}}.$$

Obviously, $A_3 + B_3 > 0$. By direct calculations, we obtain

$$b = (A_1 + B_1)(A_2 + B_2) - (A_3 + B_3) = \left[\left(d + \frac{c}{m+h} \right) \left(1 - \frac{1}{1+\alpha I_2} \right) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \right] \left[\left(d + \frac{c}{m+h} \right) \left(1 - \frac{1}{1+\alpha I_2} \right) \left(\delta + \frac{rS_2}{K} \right) \right]$$

$$+ \frac{r\delta S_2}{K} - \frac{rS_2}{K} \frac{ch}{(m+h)^2} + \frac{\beta^2 S_2 I_2}{(1+\alpha I_2)^3} \Big] - \Big[\frac{r\delta S_2}{K} \Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) \\ + \frac{\delta \beta^2 S_2 I_2}{(1+\alpha I_2)^3} \Big] = \Big[\Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) \Big(\delta + \frac{rS_2}{K} \Big) - \frac{rS_2}{K} \frac{ch}{(m+h)^2} \Big] \\ \times \Big[\Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \Big] + \frac{r\delta S_2}{K} \Big(\delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \Big) + \frac{\beta^2 S_2 I_2}{(1+\alpha I_2)^3} \Big[\Big(d + \frac{c}{m+h} \Big) \Big(1 - \frac{1}{1+\alpha I_2} \Big) + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} \Big]$$

If the conditions of the theorem are satisfied, by Routh-Hurwitz criterion (Hurwitz, [22]), we know that all the roots of equation (3.4) have negative real parts, i.e., the positive equilibrium E_2 is locally asymptotically stable provided that the above conditions are held. This completes the proof of the theorem. \Box

To show that the positive equilibrium E_2 of system (1.2) is locally asymptotically stable for all $\tau \geq 0$, we shall use the following lemma.

Lemma 1. (Gopalsamy, [10]) A set of necessary and sufficient conditions for E_2 to be locally asymptotically stable for all $\tau \ge 0$ is the following:

(I) E_2 is stable in absence of time delay τ .

(II) There is no purely imaginary root of the characteristic equation (3.3).

In the following, we apply Lemma 1 to investigate the characteristic equation (3.3).

Theorem 2. Suppose that

(i)
$$\left[\left(d + \frac{c}{m+h}\right)\left(1 - \frac{1}{1+\alpha I_2}\right)\left(\delta + \frac{rS_2}{K}\right) - \frac{rS_2}{K}\frac{ch}{(m+h)^2}\right]\left[\left(d + \frac{c}{m+h}\right)\right] \times \left(1 - \frac{1}{1+\alpha I_2}\right) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2}\right] + \frac{r\delta S_2}{K}\left(\delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2}\right) + \frac{\beta^2 S_2 I_2}{(1+\alpha I_2)^3}\left[\left(d + \frac{c}{m+h}\right)\left(1 - \frac{1}{1+\alpha I_2}\right) + \frac{rS_2}{K} - \frac{ch}{(m+h)^2}\right] > 0,$$

(ii) $\left(d + \frac{c}{m+h}\right)\left(1 - \frac{1}{1+\alpha I_2}\right) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} > 0,$

(ii)
$$\left(d + \frac{c}{m+h}\right) \left(1 - \frac{1}{1+\alpha I_2}\right) + \delta + \frac{rS_2}{K} - \frac{ch}{(m+h)^2} > 0$$

(iii) $b_2 > 0, b_3 > 0$, where

$$b_{2} = \delta^{2} \left(d + \frac{cm}{(m+h)^{2}} \right)^{2} + \frac{r^{2} S_{2}^{2}}{K^{2}} \left(\delta + d + \frac{cm}{(m+h)^{2}} \right)^{2} + \frac{\delta ch}{(m+h)^{2}} \\ + 2 \frac{\delta ch}{(m+h)^{2}} \delta \left(d + \frac{cm}{(m+h)^{2}} \right) - 2 \frac{r^{2} S_{2}^{2}}{K^{2}} \delta \left(d + \frac{cm}{(m+h)^{2}} \right) - 2 \frac{r^{2} S_{2}^{2}}{K^{2}} \frac{\delta ch}{(m+h)^{2}}, \\ b_{3} = \frac{r^{2} \delta^{2} S_{2}^{2}}{K^{2}} \left[d + \frac{cm}{(m+h)^{2}} + \frac{ch}{(m+h)^{2}} \right]^{2} - \frac{\delta^{2} \beta^{2} S_{2}^{2}}{(1+\alpha I_{2})^{4}} \left(\frac{rS_{2}}{K} - \frac{\beta I_{2}}{1+\alpha I_{2}} \right)^{2}.$$

Then the positive equilibrium E_2 of (1.2) is asymptotically stable for all $\tau \geq 0$.

Proof. Assumptions (I) and (II) of Lemma 1 require real parts of the roots of $\Delta(\lambda, 0) = 0$ to be negative and $\Delta(i\omega, \tau) \neq 0$ (where $i = \sqrt{-1}$) for any real ω and $\tau \geq 0$. If at least one root of $\Delta(\lambda, 0) = 0$ have a positive real part then E_2 is no longer asymptotically stable in absence of time delay. Again, if there exists a real $\omega > 0$ for some $\tau \geq 0$ such that $\Delta(i\omega, \tau) = 0$ then the characteristic equation (3.3) will have at least one pair of purely imaginary roots and hence E_2 is not asymptotically stable in presence of discrete time delay.

By Theorem 1, the condition (I) of Lemma 1 is easily satisfied. We now verify the condition (II) of Lemma 1. Firstly, when $\omega_0 = 0$, we have $\Delta(0, \tau) = A_3 + B_3 \neq 0$. Secondly, when $\omega_0 \neq 0$, we have

$$\Delta(i\omega_0,\tau) = -i\omega_0^3 - A_1\omega_0^2 + iA_2\omega_0 + A_3 - B_1\omega_0^2 e^{-i\omega_0\tau} + B_2i\omega_0 e^{-i\omega_0\tau} + B_3 e^{-i\omega_0\tau} = 0.$$

Separating the real and imaginary parts, we obtain

$$A_1\omega_0^2 - A_3 = (B_3 - B_1\omega_0^2)\cos(\omega_0\tau) + B_2\omega_0\sin(\omega_0\tau), \qquad (3.5)$$

$$\omega_0^3 - A_2 \omega_0 = B_2 \omega_0 \cos(\omega_0 \tau) - (B_3 - B_1 \omega_0^2) \sin(\omega_0 \tau).$$
(3.6)

Squaring and adding (3.5) and (3.6) we get

$$(B_3 - B_1\omega_0^2)^2 + B_2^2\omega_0^2 = (A_1\omega_0^2 - A_3)^2 + (\omega_0^3 - A_2\omega_0)^2.$$

We finally have

$$\omega_0^6 + b_1 \omega_0^4 + b_2 \omega_0^2 + b_3 = 0, \qquad (3.7)$$

where

$$\begin{split} b_1 &= A_1^2 - 2A_2 - B_1^2 = \delta^2 + \left(d + \frac{cm}{(m+h)^2}\right)^2 + \frac{r^2 S_2^2}{K^2} - \frac{2\delta ch}{(m+h)^2} - \frac{\beta^2 S_2^2}{(1+\alpha I_2)^4}, \\ b_2 &= A_2^2 - B_2^2 - 2A_1 A_3 + 2B_1 B_3 = \delta^2 \left(d + \frac{cm}{(m+h)^2}\right)^2 + \frac{r^2 S_2^2}{K^2} \left(\delta + d + \frac{cm}{(m+h)^2}\right)^2 \\ &+ d\frac{\delta ch}{(m+h)^2} + 2\delta \left[\frac{\delta ch}{(m+h)^2} - \frac{r^2 S_2^2}{K^2}\right] \left(d + \frac{cm}{(m+h)^2}\right) - 2\frac{r^2 S_2^2}{K^2} \frac{\delta ch}{(m+h)^2}, \\ b_3 &= A_3^2 - B_3^2 = \frac{r^2 \delta^2 S_2^2}{K^2} \left[d + \frac{cm}{(m+h)^2} + \frac{ch}{(m+h)^2}\right]^2 - \frac{\delta^2 \beta^2 S_2^2}{(1+\alpha I_2)^2} \left(\frac{rS_2}{K} - \frac{\beta I_2}{1+\alpha I_2}\right)^2. \end{split}$$

Let $\omega_0^2 = \rho$. Thus, Eq. (3.7) becomes

$$g(\rho) = \rho^3 + b_1 \rho^2 + b_2 \rho + b_3 = 0.$$
(3.8)

We claim that (3.8) has no positive roots when $b_2 > 0$ and $b_3 > 0$. In fact, we notice that $\frac{dg(\rho)}{d\rho} = 3\rho^2 + 2b_1\rho + b_2$. Set

$$3\rho^2 + 2b_1\rho + b_2 = 0. ag{3.9}$$

The roots of (3.9) can be expressed as $\rho_{1,2} = \frac{-b_1 \pm \sqrt{b_1^2 - 3b_2}}{3}$. If $b_2 > 0$, then $b_1^2 - 3b_2 < b_1^2$; that is $\sqrt{b_1^2 - 3b_2} < b_1$. Hence, neither ρ_1 nor ρ_2 is positive. Thus, (3.9) does not have positive roots. If $g(0) = b_3 > 0$, it follows that (3.9) has no positive roots. Hence, $\Delta(i\omega_0, \tau) \neq 0$ for any real τ . This satisfies condition (II) of Lemma 1. Therefore, the positive equilibrium E_2 is always asymptotically stable for all $\tau \geq 0$ and the delay is harmless in this case. This completes the proof of the theorem. \Box

In the following, we wish to obtain a criterion for preservation of instability or stability. By using the time delay as τ the bifurcation parameter, the criteria for Hopf bifurcation are given.

Theorem 3. Assume that one of the following conditions holds:

- (i) $b_1 > 0$, $b_2 > 0$, $b_3 > 0$;
- (ii) $b_1 > 0$, $b_2 < 0$, $b_3 > 0$ and relation (3.17) holds, where b_1 , b_2 and b_3 are as defined in (3.7).

Then if the positive equilibrium E_2 is stable (unstable) at $\tau = 0$, then it remains stable (unstable) for all $\tau > 0$.

Proof. Let us begin with the characteristic Eq. (3.3). Note that it is the sign of the real parts of the solutions λ of Eq. (3.3) that determines the stability of the positive equilibrium E_2 . Let $\lambda = \mu + i\nu$. Substituting $\lambda = \mu + i\nu$ into Eq. (3.3) gives the following equations:

$$\mu^{3} - 3\mu\nu^{2} - A_{1}\mu^{2} + A_{1}\nu^{2} + A_{2}\mu + A_{3}$$

- $(B_{1}\mu^{2} - B_{1}\nu^{2} + B_{2}\mu + B_{3})e^{-\mu\tau}\cos(\nu\tau) - (2B_{1}\mu\nu + B_{2}\nu)e^{-\mu\tau}\sin(\nu\tau) = 0,$
- $\nu^{3} + 3\mu^{2}\nu + 2A_{1}\mu\nu + A_{2}\nu + (B_{1}\mu^{2} - B_{1}\nu^{2} + B_{2}\mu + B_{3})e^{-\mu\tau}\sin(\nu\tau)$
- $(2B_{1}\mu\nu + B_{2}\nu)e^{-\mu\tau}\cos(\nu\tau) = 0.$ (3.10)

We now investigate λ , and thus μ and ν are considered as functions of the delay τ . Since the change of stability of E_2 will occur at any values of τ for which $\mu = 0$, we let $\hat{\tau}$ be such that $\nu(\hat{\tau}) \neq 0$. Thus the Eq. (3.10) reduces to (where $\hat{\nu} = \nu(\hat{\tau})$)

$$-A_1\hat{\nu}^2 + A_3 = B_1\hat{\nu}^2\cos(\hat{\nu}\hat{\tau}) - B_3\cos(\hat{\nu}\hat{\tau}) - B_2\hat{\nu}\sin(\hat{\nu}\hat{\tau}), -\hat{\nu}^3 + A_2\hat{\nu} = -B_1\hat{\nu}^2\sin(\hat{\nu}\hat{\tau}) + B_3\sin(\hat{\nu}\hat{\tau}) - B_2\hat{\nu}\cos(\hat{\nu}\hat{\tau}).$$
(3.11)

Squaring and adding the both equations of (3.11) and simplifying, we obtain an equation for $\hat{\nu}$ of the following form

$$\hat{\nu}^6 + (A_1^2 - B_1^2 - 2A_2)\hat{\nu}^4 + (A_2^2 - 2A_1A_3 + 2B_1B_3 - B_2^2)\hat{\nu}^2 + A_3^2 - B_3^2 = 0.$$
(3.12)

In order to establish Hopf bifurcation at $\tau = \hat{\tau}$, we need to show that $\frac{d\mu}{dt} \neq 0$ at $\tau = \hat{\tau}$. Differentiating (3.3) with respect to τ , and setting $\tau = \hat{\tau}$, $\mu = 0$ and

 $\nu = \hat{\nu}$, we obtain

$$L_1 \frac{d\mu(\hat{\tau})}{d\tau} + L_2 \frac{d\nu(\hat{\tau})}{d\tau} = Y_1, \quad -L_2 \frac{d\mu(\hat{\tau})}{d\tau} + L_1 \frac{d\nu(\hat{\tau})}{d\tau} = Y_2, \quad (3.13)$$

where

$$L_{1} = -3\hat{\nu}^{2} + A_{2} + B_{2}\cos(\hat{\nu}\hat{\tau}) + 2B_{1}\hat{\nu}\sin(\hat{\nu}\hat{\tau}), \qquad (3.14)$$

$$L_{2} = -2A_{1}\hat{\nu} - 2B_{1}\hat{\nu}\cos(\hat{\nu}\hat{\tau}) + B_{2}\sin(\hat{\nu}\hat{\tau}), \qquad (3.14)$$

$$Y_{1} = -B_{1}\hat{\nu}^{3}\sin(\hat{\nu}\hat{\tau}) + B_{3}\hat{\nu}\sin(\hat{\nu}\hat{\tau}) - B_{2}\hat{\nu}^{2}\cos(\hat{\nu}\hat{\tau}), \qquad (3.14)$$

$$Y_{2} = B_{2}\hat{\nu}^{2}\sin(\hat{\nu}\hat{\tau}) - B_{1}\hat{\nu}^{3}\cos(\hat{\nu}\hat{\tau}) - B_{3}\hat{\nu}\cos(\hat{\nu}\hat{\tau}).$$

Solving (3.13), we get

$$\frac{d\mu(\hat{\tau})}{d\tau} = \frac{L_1 Y_1 - L_2 Y_2}{L_1^2 + L_2^2}.$$

Clearly, $\frac{d\mu(\hat{\tau})}{d\tau}$ has the same sign as $L_1Y_1 - L_2Y_2$. From (3.14), after some simplification, we obtain

$$L_1Y_1 - L_2Y_2 = \hat{\nu}^2 \left[3\hat{\nu}^4 + 2(A_1^2 - 2A_2 - B_1^2)\hat{\nu}^2 + (A_2^2 - 2A_2A_3 + 2B_1B_3 - B_2^2) \right].$$
(3.15)

Let

$$F(z) = z^3 + b_1 z^2 + b_2 z + b_3, (3.16)$$

where b_1 , b_2 , b_3 are defined in (3.7). Then, $F(\hat{\nu}^2) = 0$ and from (3.15) and (3.16), we obtain

$$\frac{d\mu(\hat{\tau})}{d\tau} = \frac{\hat{\nu}^2}{L_1^2 + L_2^2} \frac{dF}{dz} (\hat{\nu}^2).$$

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

- (HH1) If the polynomial F(z) has no positive roots, there can be no change of stability.
- (HH2) If F(z) is increasing (decreasing) at all of its positive roots, instability (stability) is preserved. If $F(0) = b_3 > 0$ and $b_1 > 0$, then F(z) will either have two positive roots or no positive roots. We consider the following cases in order that F(z) may have no positive roots.
- (HH3) If $b_1 > 0$, $b_2 \ge 0$ and $b_3 > 0$, then F(z) > 0 for all z > 0. Obviously, the above (HH1) holds. Namely, stability or instability will be preserved in this case.
- (HH4) Consider $b_1 > 0$, $b_2 < 0$ and $b_3 > 0$. If $b_1 > 0$, $b_3 > 0$ and $b_2 < 0$, then the minimum of F(z) will exist at $z_{min} = \frac{-b_1 + \sqrt{b_1^2 - 3b_2}}{3}$ and (HH1) will be satisfied if $F(z_{min}) > 0$, i.e.,

$$2b_1^3 - 9b_1b_2 + 27b_3 > 2(b_1^2 - 3b_2)^{\frac{3}{2}}$$
(3.17)

or

$$2b_1(b_1^2 - 3b_2) + 27b_3 - 3b_1b_3 > 2(b_1^2 - 3b_2)^{\frac{3}{2}}$$

Since $27b_3 - 3b_1b_3 > 27b_3$ (since $b_1 > 0$, $b_3 > 0$ and $b_2 < 0$), and $b_1^2 - 3b_2 > b_1^2$, hence

$$2b_1(b_1^2 - 3b_2) + 27b_3 - 3b_1b_3 > 27b_3 + 2b_1^3$$

Thus, for inequality (3.17) to hold it is sufficient that

$$27b_3 + 2b_1^3 > 2(b_1^2 - 3b_2)^{\frac{3}{2}} \Rightarrow b_2 > \frac{1}{3} \left[b_1^2 - \left(\frac{27b_3 + 2b_1^3}{2}\right)^{\frac{2}{3}} \right]$$

Thus, the polynomial F(z) has also no positive roots in this case. This completes the proof of the theorem. \Box

Theorem 4. Assume that $b_1 > 0$ and $b_3 \leq 0$ hold. Then if E_2 is unstable at $\tau = \tau_0 \geq 0$, then E_2 remains unstable for all $\tau > \tau_0$.

Proof. If $F(0) = b_3 \leq 0$, then by Descartes's rule of signs, F(z) has at most one positive root. Note that if F(z) has only one positive root, then F(z) must be increasing at this positive root. This completes the proof of the theorem. \Box

Theorem 5. Assume that b > 0 and $b_3 < 0$ hold. If $\hat{\nu}_0$ is the first positive root of Eq. (3.12). Then a Hopf bifurcation occurs as τ passes through $\hat{\tau}_0$.

Proof. Note that if the polynomial F(z) has two or three distinct positive roots, the above criterion (HH2) cannot hold, since it is decreasing at one root and increasing at the other. Hence, stability cannot be preserved if $F(z_0) < 0$ for some $z_0 > 0$. In particular, a Hopf bifurcation may occur as τ passes through critical value $\hat{\tau}_0$. To do so, in the following we always assume that b > 0 and $b_3 < 0$. For b > 0, by Theorem 1, we have shown that the infected equilibrium E_2 is stable at $\tau = 0$. The cubic Eq. (3.12) in $\hat{\nu}^2$ has one or more positive real roots $\hat{\nu}_0^2$, since when $\hat{\nu} = 0$, the left side of Eq. (3.12) $b_3 = A_3^2 - B_3^2$ is negative, and for sufficiently large values of $\hat{\nu}$, it is positive. Namely, the characteristic Eq. (3.3) has a pair of purely imaginary roots, denoted by $\pm i\hat{\nu}$. Thus from (3.5) and (3.6), we can determine $\hat{\tau}$, which is of the form

$$\tau_n = \frac{1}{\nu_0} \arccos\left[\frac{(A_1\nu_0^2 - A_3)(B_3 - B_1\nu_0^2) + (\nu_0^3 - A_2\nu_0)B_2\nu_0}{(B_3 - B_1\nu_0^2)^2 + (B_2\nu_0)^2}\right] + \frac{2n\pi}{\nu_0},$$

$$(n = 0, 1, 2, 3, \ldots). \quad (3.18)$$

If $\hat{\nu}_0$ is the first positive root of Eqs. (3.5) and (3.6), it follows from (3.15) that

$$\frac{d\mu(\hat{\tau}_0)}{d\tau} = \frac{\hat{\nu}_0^2}{L_1^2 + L_2^2} \frac{dF}{dz}(\hat{\nu}_0^2) > 0.$$

After the Hopf bifurcation theorem (Hassard et al., [16]), we obtain the conclusions. $\ \Box$

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4 Estimation of the Length of Delay to Preserve Stability

In this paper, we introduce the type of discrete time delay in the prey infection equations based upon the hypothesis that the infection rate of prey depends on the susceptible preys and the infected preys present within the system at the previous time. Since we are interested in the time rate of change of the infectious preys at time t and since it takes τ number of days for the infected preys to become infectious, we should be interested in the number of susceptible preys who contact an infected prey at the time $t - \tau$ not at the time t. Hence, time delay τ is crucial for understanding this paper. In this section, we will estimate the length of delay to preserve stability.

We consider the system (1.2) and the space of all real valued continuous functions defined on $[-\tau, \infty)$ satisfying the initial conditions (1.3) on $[-\tau, 0]$. We linearize the system (1.2) about its interior equilibrium $E_2(S_2, I_2, P_2)$ and get

$$\begin{cases} \dot{S} = -\frac{rS_2}{K}S - \frac{\beta S_2}{(1+\alpha I_2)^2}I(t-\tau), \\ \dot{I} = \frac{\beta I_2}{1+\alpha I_2}S - [\frac{cmP_2^2}{(mP_2+I_2)^2} + d]I + \frac{\beta S_2}{(1+\alpha I_2)^2}I(t-\tau) - \frac{cI_2^2}{(mP_2+I_2)^2}P, \\ \dot{P} = \delta I/h - \delta P. \end{cases}$$
(4.1)

Taking Laplace transform of the system given by (4.1), we get

$$\begin{aligned} \zeta &(\varsigma + \frac{rS_2}{K})L_S(\varsigma) = -\frac{\beta S_2}{(1+\alpha I_2)^2} e^{-\varsigma\tau} L_I(\varsigma) - \frac{\beta S_2}{(1+\alpha I_2)^2} e^{-\varsigma\tau} K_1(\varsigma) + L_S(0), \\ &(\varsigma + \frac{cmP_2^2}{(mP_2 + I_2)^2} + d - \frac{\beta S_2}{(1+\alpha I_2)^2})L_I(\varsigma) = \frac{\beta I_2}{1+\alpha I_2} L_S(\varsigma) \\ &+ \frac{\beta S_2}{(1+\alpha I_2)^2} e^{-\varsigma\tau} K_1(\varsigma) - \frac{cI_2^2}{(mP_2 + I_2)^2} L_P(\varsigma) + L_I(0), \\ &\zeta &(\varsigma + \delta)L_P(\varsigma) = \delta L_I(\varsigma)/h + L_P(0), \end{aligned}$$

where $K_1(\varsigma) = \int_{-\tau}^0 e^{-\varsigma t} I(t) dt$, and L_T , L_I and L_P are the Laplace transforms of S(t), I(t) and P(t), respectively.

The inverse Laplace transformation of $L_I(\varsigma)$ will have terms which exponentially increase with time if $L_I(\varsigma)$ has poles with positive real parts. For E_2 to be locally asymptotically stable it is necessary and sufficient condition that all poles of $L_I(\varsigma)$ have negative real parts. We will employ the Nyquist criterion (Freedman and Rao, [8]; see Appendix A) which states that if ς is the arc length of a curve encircling the right half plane, the curve $L_I(\varsigma)$ will encircle the origin a number of times equal to the difference between the numbers of poles and zeroes of $L_I(\varsigma)$ in the right half plane. Proceeding along the line of Freedman and Rao [8], we see that the conditions for local asymptotic stability of E_2 is given by the Nyquist criteria

$$ImH(i\eta_0) > 0, \quad ReH(i\eta_0) = 0,$$
 (4.2)

where $H(\varsigma) = \varsigma^3 + A_1\varsigma^2 + A_2\varsigma + A_3 + e^{-\varsigma t}(B_1\varsigma^2 + B_2\varsigma + B_3)$ and η_0 is the smallest positive root of the second equation (4.2).

In our case, (4.2) gives

$$A_2\eta_0 - \eta_0^3 > -B_1\eta_0^2\sin(\eta_0\tau) + B_3\sin(\eta_0\tau) - B_2\eta_0\cos(\eta_0\tau).$$
(4.3)

$$A_3 - A_1 \eta_0^2 = B_1 \eta_0^2 \cos(\eta_0 \tau) - B_3 \cos(\eta_0 \tau) - B_2 \eta_0 \sin(\eta_0 \tau).$$
(4.4)

If (4.3) and (4.4) are satisfied simultaneously, they are sufficient conditions to guarantee stability. We shall apply them to get an estimate on the length of delay. Our aim is to find an upper bound η_+ on η_0 , independent of τ and then to estimate τ so that (4.3) holds for all values of η , $0 \le \eta \le \eta_+$ and hence in particular at $\eta = \eta_0$. We rewrite (4.4) as

$$A_1\eta_0^2 = A_3 + B_3\cos(\eta_0\tau) + B_2\eta_0\sin(\eta_0\tau) - B_1\eta_0^2\cos(\eta_0\tau).$$
(4.5)

Maximizing $A_3 + B_3 \cos(\eta_0 \tau) + B_2 \eta_0 \sin(\eta_0 \tau) - B_1 \eta_0^2 \cos(\eta_0 \tau)$ subject to

$$|\sin(\eta_0 \tau)| \le 1, \quad |\cos(\eta_0 \tau)| \le 1$$

we obtain

$$A_1\eta_0^2 \le A_3 + |B_3| + |B_2|\eta_0 + |B_1|\eta_0^2.$$
(4.6)

Hence, if

$$\eta_{+} = \frac{|B_2| + \sqrt{B_2^2 + 4(A_1 - |B_1|)(A_3 + |B_3|)}}{2(A_1 - |B_1|)}, \tag{4.7}$$

then clearly from (4.6) we have $\eta_0 \leq \eta_+$. To get (4.7), we need $A_1 > |B_1|$. From (4.4) we obtain

$$\eta_0^2 < A_2 + B_1 \eta_0 \sin(\eta_0 \tau) + B_2 \cos(\eta_0 \tau) - B_3 \sin(\eta_0 \tau) / \eta_0.$$
(4.8)

As E_2 is locally asymptotically stable for $\tau = 0$, therefore for sufficiently small $\tau > 0$, (4.7) is satisfied. Substituting (4.5) in (4.8) and rearranging we get,

$$(B_3 - A_1 B_2 - B_1 \eta_0^2) \left[\cos(\eta_0 \tau) - 1 \right] + \left[(B_2 - A_1 B_1) \eta_0 + \frac{A_1 B_3}{\eta_0} \right] \sin(\eta_0 \tau) < A_1 A_2 - A_3 - B_3 + A_1 B_2 + B_1^2 \eta_0.$$
(4.9)

Using the bounds

$$\left(B_3 - A_1 B_2 - B_1 \eta_0^2 \right) \left[\cos(\eta_0 \tau) - 1 \right] = 2 \left(B_1 \eta_0^2 + A_1 B_2 - B_3 \right) \sin^2 \left(\frac{\eta_0 \tau}{2} \right)$$

$$\leq \frac{1}{2} \left| B_1 \eta_+^2 + A_1 B_2 - B_3 \right| \eta_+^2 \tau^2$$

$$\left[\left| B_2 - A_1 B_1 \right| \eta_0 + \frac{A_1 B_3}{\eta_0} \right] \sin(\eta_0 \tau) \leq \left[\left(B_2 - A_1 B_1 \right) \eta_+^2 + A_1 |B_3| \right] \tau,$$

we obtain from (4.9) $K_1 \tau^2 + K_2 \tau < K_3$, where

$$K_{1} = \frac{1}{2} \Big| B_{1} \eta_{+}^{2} + A_{1} B_{2} - B_{3} \Big| \eta_{+}^{2}, \quad K_{2} = \Big| B_{2} - A_{1} B_{1} \Big| \eta_{+}^{2} + A_{1} \Big| B_{3} \Big|,$$

$$K_{3} = A_{1} A_{2} - A_{3} - B_{3} + A_{1} B_{2} + B_{1}^{2} \eta_{+}.$$

Thus if $K_1\tau^2 + K_2\tau < K_3$ holds, then the inequality (4.3) is satisfied automatically. A positive root of $K_1\tau^2 + K_2\tau = K_3$ is given by

$$\tau_{+} = \frac{1}{2K_{1}} \Big(-K_{2} + \sqrt{K_{2}^{2} + 4K_{1}K_{3}} \Big).$$
(4.10)

For $0 \leq \tau \leq \tau_+$, the Nyquist criteria hold. τ_+ gives estimate for the length of delay for which stability is preserved. Here τ_+ is dependent on the expression of K_1 , K_2 and K_3 in (4.10) which is again function of the system parameters. Hence we can conclude that the estimate for the delay is totally dependent on the system parameters for which the interior equilibrium E_2 is locally asymptotically stable. Thus we are now in a position to state the following theorem.

Theorem 6. Suppose

$$\delta + d + \frac{cm}{(m+h)^2} + \frac{rS_2}{K} > \frac{\beta S_2}{(1+\alpha I_2)^2}$$

If there exists a parameter $0 \le \tau < \tau_+$ such that $K_1\tau^2 + K_2\tau < K_3$, then τ_+ is the maximum value (length of delay) of τ for which E_2 is asymptotically stable.

5 Permanence

From biological point of view, permanence of a system means the survival of all populations of the system in future. Mathematically, permanence of a system means that strictly positive solutions do not have omega limit points on the boundary of the non-negative cone. Butler et al. [5], Freedman and Waltman ([9], [7]) developed the following definition of persistence:

DEFINITION 1. System (1.2) is said to be permanent if there are positive constants \overline{M}_1 , \overline{M}_2 such that each positive solution (S(t), I(t), P(t)) of system (1.2) with initial conditions (1.3) satisfies

$$\bar{M}_1 \leq \lim_{t \to +\infty} \inf S(t) \leq \lim_{t \to +\infty} \sup S(t) \leq \bar{M}_2,$$

$$\bar{M}_1 \leq \lim_{t \to +\infty} \inf I(t) \leq \lim_{t \to +\infty} \sup I(t) \leq \bar{M}_2,$$

$$\bar{M}_1 \leq \lim_{t \to +\infty} \inf P(t) \leq \lim_{t \to +\infty} \sup P(t) \leq \bar{M}_2.$$

DEFINITION 2. (Hale et al., [12]) The semigroup T(t) is said to be point dissipative in X if there is a bounded nonempty set B in X such that, for any $x \in X$, there is a $t_0 = t_0(x, B)$ such that $T(t)x \in B$ for $t \ge t_0$.

Let X be a complete metric space. Suppose that X^0 is open and dense in X and $X^0 \cup X_0 = X$, $X^0 \cap X_0 = \emptyset$. Assume that T(t) is a C_0 semigroup on X satisfying

$$T(t): X^0 \to X^0, \quad T(t): X_0 \to X_0.$$
 (5.1)

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

Let M_1 , M_2 be isolated invariant sets (not necessarily distinct). M_1 is said to be chained to M_2 , written $M_1 \mapsto M_2$.

DEFINITION 3. Finite cover $M = \bigcup_{k=1}^{m} M_k$ is called a cycle covering if $M_1 \mapsto M_2 \mapsto \cdots \mapsto M_k \mapsto M_1$, for some $k \in \{1, 2, \cdots, m\}$. Otherwise, M is called acyclic covering.

Remark 1. Readers can find some relative definitions in references (Butler et al., [5]; Hale et al., [12]).

In order to prove permanence of system (1.2), we present the permanence theory for infinite dimensional system from Theorem 4.1 in Hale et al., [12].

Lemma 2. (Hale et al., [12]) Suppose that T(t) satisfies (5.1) and we have the following:

- (i) There is a $t_0 \ge 0$ such that T(t) is compact for $t > t_0$;
- (ii) T(t) is point dissipative in X;
- (iii) $\overline{A}_b = \bigcup_{x \in A_b} \omega(x)$ is isolated and has an acyclic covering \overline{M} , where $\overline{M} = \{M_1, M_2, \cdots, M_n\};$
- (iv) $W^s(M_i) \cap X^0 = \emptyset$, for $i = 1, 2, \dots, n$, where $W^s(M_i)$ denotes the stable manifolds of M_i .

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

Theorem 7. If $\beta K > d$, then system (1.2) is permanent.

Proof. We begin by showing that the boundary planes of R^3_+ repel the positive solutions to system (1.2) uniformly. Let us define

$$C_0 = \Big\{ (\varphi_1, \varphi_2, \varphi_3) \in C([-\tau, 0], R^3_+) : \varphi_3(\theta) = 0, \varphi_1(\theta)\varphi_2(\theta) \neq 0, \theta \in [-\tau, 0] \Big\}.$$

If $C^0 = intC([-\tau, 0], R_+^3)$, it is suffices to show that there exists an ε_0 such that for all solutions u_t of system (1.2) initiating from C^0 , $\liminf_{t\to\infty} d(u_t, C_0) \ge \varepsilon_0$. To this end, we verify below that the conditions of Lemma 2 are satisfied. It is easy to see that C_0 and C^0 are positive invariant. Moreover, conditions (i) and (ii) of Lemma 2 are clearly satisfied.

Thus, we only need to verify conditions (iii) and (iv). There is a constant solution E_1 in C_0 corresponding to $S(t) = S_1$, $I(t) = I_1$, P = 0, where S_1 and I_1 are the values of the boundary equilibrium E_1 . If (S(t), I(t), P(t)) is a solution of system (1.2) initiating from C_0 with $\varphi_1(\theta)\varphi_2(\theta) \neq 0$, then $S(t) \rightarrow S_1$, $I(t) \rightarrow I_1$, $P \rightarrow 0$ as $t \rightarrow +\infty$. It is obvious that E_1 is isolated invariant. In fact, we only show that $W^s(E_1) \cap C^0 = \emptyset$. Assuming the contrary, then there exists a positive $(\tilde{S}(t), \tilde{I}(t), \tilde{P}(t))$ of system (1.2) such that $(\tilde{S}(t), \tilde{I}(t), \tilde{P}(t)) \rightarrow$ $(S_1, I_1, 0)$ as $t \rightarrow +\infty$. Choosing $\xi > 0$ small enough such that $I_1 - \xi > 0$ when $\beta K > d$. Let $t_0 > 0$ be sufficiently large such that $I_1 - \xi < \tilde{I}(t) < I_1 + \xi$ for $t \ge t_0 - \tau$. Then we have, for $t \ge t_0$, $\frac{d\tilde{P}}{dt} \ge \delta \tilde{P}(1 - \frac{h\tilde{P}}{I_1 - \xi})$. It is easy to prove that $\tilde{P}(t) \ge \frac{I_1 - \xi}{h}$ when $I_1 - \xi > 0$. This is a contradiction. Hence, $W^s(E_1) \cap C^0 = \emptyset$.

Therefore, we are able to conclude from Lemma 2 that C_0 repels the positive solutions of system (1.2) uniformly, then the conclusion of Theorem 7 follows. \Box

6 Numerical Simulation

We have incorporated delay due to infection. Stability switching is observed as the time lag increases. We have gained analytical understanding of possible dynamics of this nonlinear delay differential equation model to make it some extent. We now perform some simulation results with hypothetical set of parameters given in Table 1 and initial values S(0) = 50, I(0) = 80, y(0) = 100for better understanding of our analytical treatment. In fact we have considered different values of the delay factor (τ) to observe biologically plausible different dynamical scenarios of the model, enough to merit the mathematical study. First we observe that without delay there exits a unique interior equi-

Parameter	Values	Unit
r (intrinsic birth rate of the sound prey) K (carrying capacity of the sound prey) β (infection rate) α (half saturation constant of infection) d (death rate of the infected prey) c (the search rate) m (half capturing saturation constant) δ (intrinsic growth rate of the predator) h (the maximum value of the per capita reduction rate of prey due to predator)	$\begin{array}{c} 0.3 \\ 1000 \\ 0.0015 \\ 0.0001 \\ 0.03 \\ 8 \\ 150 \\ 0.2 \\ 0.5 \end{array}$	1/day kg/km ² 1/day kg/day 1/day kg/day

Table 1. Parameter values used for simulation.

librium point E_2 (56.50365052, 192.3284945, 384.6569891) with the set of parameter values from Table 1. Positive steady state E_2 is locally asymptotically stable, since the eigenvalues associated with the variational matrix of the system (1.2) at E_2 , given by (-0.1998912343, -0.009226206361 - 0.1518129302i), -0.009226206361 + 0.1518129302i) have negative real parts. Simulation of the model in this situation with $\tau = 0$, shows that system (1.2) has stable positive equilibrium E_2 and is presented in Fig. 1. With the same set of parameters, we see that $b_3(-0.000187736964) < 0$ and $b_1(0.04044586767) > 0$, which indicates the existence of a positive root of F(z) = 0. Solving (3.5) and (3.6) numerically, we see that there exist one simple positive root of F(z) = 0, namely, $0.1463234602(=\hat{\nu}_0)$. Hence, by Theorem 5, we can say that as τ increases, stability switch may occur. The value of τ where stability switch occurs is $\hat{\tau}_0 = 0.8836114514$, which can be easily calculated using (3.5) and (3.6). Hence, by Butler's lemma (Freedman and Rao, [8]; see Appendix B),



Figure 1. Time evolution of all the population for the model (1.2) with $\tau = 0$ and initial value (50,80,100). (a), (b) and (c) are S, I and P versus t, respectively. And (d) is the projected S - I - P phase plane.

 E_2 remains stable for $\tau < \hat{\tau}_0$ (= 0.8836114514), which can be seen in Figs.1 and 2 and which are the solutions of the system (1.2) for $\tau = 0$ and $\tau = 0.5$, respectively. As τ increases through $\tau = \hat{\tau}_0 = 0.8836114514$, a periodic solution occurs which is the case of Hopf bifurcation. The importance of Hopf bifurcation in this context is that at the bifurcation point a limit cycle (see Fig.3) is formed around the fixed point, thus resulting in stable periodic solutions.

7 Discussion

In this paper we have considered a prey-predator system where the prey population is divided into two groups, infected and non-infected. The disease modifies the external features or the behaviour of the prey so as to make infected individuals more vulnerable to predation. The system is analysed for its equilibria and their stability. The conditions for persistence are given. Persistence conditions indicate that predator death rate has some upper threshold value. Local stability condition of E_2 is established which is very difficult to interpret biologically.

It is worth mentioning that, although we have presented a number of hypothetical examples of disease selective predation, our study is restricted only to the extensive theoretical analysis of such scenarios. Our theoretical study could only capture the major mechanisms associated with an eco-epidemiological aspect where a predator selectively consumes infected prey. And further exper-



Figure 2. Time evolution of all the population for the model (1.2) with $\tau = 0.5$ and initial value (50,80,100). (a), (b) and (c) are S, I and P versus t, respectively. And (d) is the projected S - I - P phase plane.



Figure 3. Time evolution of all the population for the model (1.2) with $\tau = 1$ and initial value (50,80,100). (a), (b) and (c) are *S*, *I* and *P* versus *t*, respectively. And (d) is the projected S - I - P phase plane.

imental or field investigations in this directions could be helpful to verify the processes prescribed by the model actually functioning in a similar manner in the real world (Haque M. and Venturino E., [15]; Greenhalgh D. and Haque M., [11]; Haque M. and Venturino E., [14]; Venturino E., [33]). Lastly, we can consider a modified system (1.2) as the following model

$$\begin{cases} \frac{dS(t)}{dt} = rS(t)(1 - \frac{S(t)}{K}) - \frac{\beta e^{-\mu_1 \tau} S(t)I(t - \tau)}{1 + \alpha I(t - \tau)}, \\ \frac{dI(t)}{dt} = \frac{\beta e^{-\mu_1 \tau} S(t)I(t - \tau)}{1 + \alpha I(t - \tau)} - \frac{cP(t)I(t)}{mP(t) + I(t)} - dI(t), \\ \frac{dP(t)}{dt} = \delta P(t) \Big(1 - \frac{hP(t)}{I(t)}\Big), \end{cases}$$
(7.1)

where μ_1 is the constant death rate for infected. From simple calculation, one can get that the characteristic equation of system (7.1) has coefficients depending on the steady state coordinates and hence on this exponential factor $\beta e^{-\mu_1 \tau}$. Beretta and Kuang [3] have developed a systematic approach to studying the difficult characteristic equations arising from such system. We will summarize their technique as it applies to our particular problem in the future work.

Appendix A

Nyquist criterion: If ς is the arc length of a curve encircling the right half-plane, the curve $L(\varsigma)$ will encircle the origin a number of times equal to the difference between the number of poles and the number of zeroes of $L(\varsigma)$ in the right half-plane.

Appendix B

G. J. Butlers 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.

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References

- M.A. Aziz-Alaoui. Study of a Leslie-Gower-type tritrophic population. Chaos Solitons & Fractals, 14:1275–1293, 2002. Doi:10.1016/S0960-0779(02)00079-6.
- [2] M.A. Aziz-Alaoui and M. Daher Okiye. Boundedness and global stability for a predator-prey model with modified Leslie-Gower and Holling-type II schemes. *Appl. Math. Lett.*, 16:1069–1075, 2003.
- [3] E. Beretta and Y. Kuang. Geometric stability switch criteria in delay differential systems with delay dependent parameters. SIAM J. Math. Anal., 33:1144–1165, 2002. Doi:10.1137/S0036141000376086.

- [4] C.P. Bhunu and W. Garira. A two strain tuberculosis transmission model with therepy and quarantine. *Math. Model. Anal.*, 14(3):291–312, 2009. Doi:10.3846/1392-6292.2009.14.291-312.
- [5] G. Butler, H.I. Freedman and P. Waltman. Uniformly persistence system. Proc. Am. Math. Soc., 96:425–430, 1986. Doi:10.1090/S0002-9939-1986-0822433-4.
- [6] V. Capasso and G. Serio. A generalization of the Kermack-Mckendrick deterministic epidemic model. *Math Biosci.*, 42:41–61, 1978. Doi:10.1016/0025-5564(78)90006-8.
- [7] H.I. Freedman and P. Moson. Persistence definitions and their connections. Proc. Am. Math. Soc., 109:1025–1033, 1990.
 Doi:10.1090/S0002-9939-1990-1012928-6.
- [8] H.I. Freedman and V.S.H. Rao. The trade-off between mutual interference and time lags in predator-prey systems. *Bulletin of Mathematical Biology*, 45:991– 1004, 1983. Doi:10.1007/BF02458826.
- H.I. Freedman and P. Waltman. Persistence in a model of three competitive populations. *Mathematical Biosciences*, 73(11):89–101, 1985. Doi:10.1016/0025-5564(85)90078-1.
- [10] K. Gopalsamy. Stability and oscillation in delay different equations of population dynamics. Boston: Kluwer Academic Publishers, 1992.
- [11] D. Greenhalgh and M. Haque. A predator-prey model with disease in the prey species only. *Mathematical Methods in the Applied Sciences*, **30**(8):911–929, 2007. Doi:10.1002/mma.815.
- [12] J.K. Hale and P. Waltman. Persistence in infinite-dimensional systems. SIAM J. Math. Anal., 20:388–396, 1989. Doi:10.1137/0520025.
- [13] M. Haque and D. Greenhalgh. When a predator avoids infected prey: a modelbased theoretical study. *Math. Med. Biol.*, **27**(1):75–94, 2010. Doi:10.1093/imammb/dqp007.
- [14] M. Haque and E. Venturino. The role of transmissible disease in Holling-Tanner predator-prey model. *Theoretical Population Biology*, **70**:273–288, 2006. Doi:10.1016/j.tpb.2006.06.007.
- [15] M. Haque and E. Venturino. Modelling disease spreading in symbiotic communities? In John D. Harris and Paul L. Brown (Eds.)(Eds.), Wildlife: Destruction, Conservation and Biodiversity, pp. 135–180. Nova Science Publishers, New York, 2009.
- [16] B.D. Hassard, N.D. Kazarinoff and Y.H. Wan. Theory and application of Hopf bifucation. Cambridge: Cambridge University, 1981.
- [17] H.W. Hethcote. The mathematics of infectious disease. SIAM Review, 42:599– 653, 2000. Doi:10.1137/S0036144500371907.
- [18] H.W. Hethcote, W. Wang, L. Han and Ma Zhien. A predator-prey model with infected prey. *Theor. Pop. Biol.*, 66:259–268, 2004. Doi:10.1016/j.tpb.2004.06.010.
- [19] S.B. Hsu and T.W. Hwang. Hopf bifurcation analysis for a predator-prey system of Holling and Leslie type. *Taiwan J. Math.*, 3:35–53, 1999.
- [20] S.B. Hsu, T.W. Hwang and Y. Kuang. Global analysis of the Michaelis-Menten type ratio-dependent predator-prey system. J. Math. Biol., 42:489–506, 2001. Doi:10.1007/s002850100079.

- [21] P.J. Hudson, A.P. Dobson and D. Newborn. Do parasites make prey vulnerable to predation? Red grouse and parasites. J. Anim. Ecol., 61:681–692, 1992. Doi:10.2307/5623.
- [22] A. Hurwitz. On the conditions under which an equation has only roots with negative real parts. *Math. Ann.*, 46:273–284, 1895.
- [23] T.W. Hwang. Uniqueness of limit cycle for Gause-type predator-prey systems. J. Math. Anal. Appl., 238:179–195, 1999. Doi:10.1006/jmaa.1999.6520.
- [24] Y. Kuang and E. Beretta. Global qualitative analysis of a ratio-dependent predator-prey system. J. Math. Biol., 36:389–406, 1998. Doi:10.1007/s002850050105.
- [25] K.D. Lafferty and A.K. Morris. Altered behaviour of parasitized killfish increases susceptibility to predation by bird final hosts. *Ecology*, 77:1390–1397, 1996. Doi:10.2307/2265536.
- [26] M.X. Liu, Z. Jin and M. Haque. The periodic predator-prey Lotka-Volterra model with disease in the prey. *Nonlinear Analysis: Real World Applications*, 10:3098–3111, 2009. Doi:10.1016/j.nonrwa.2008.10.010.
- [27] W.M. Liu, H.W. Hethcote and S.A. Levin. Dynamical behavior of epidemiological models with nonlinear incidence rates. J. Math. Biol., 25:359–380, 1987. Doi:10.1007/BF00277162.
- [28] G. Magombedze, Z. Mukandavire, C. Chiyaka and G. Musuka. Optimal control of a sex-structured HIV/AIDS model with condom use. *Math. Model. Anal.*, 14(4):483–494, 2009. Doi:10.3846/1392-6292.2009.14.483-494.
- [29] D.L. Murray, J.R. Carry and L.B. Keith. Interactive effects of sublethal nematodes and nutritional status on snowshoe hare vulnerability to predation? J. Anim. Ecol., 66:250–264, 1997. Doi:10.2307/6026.
- [30] A.F. Nindjin, M.A. Aziz-Alaoui and M. Cadivel. Analysis of a predatorprey model with modified Leslie-Gower and Holling-type II schemes with time delay. *Nonlinear Analysis: Real World Applications*, 7(5):1104–1118, 2006. Doi:10.1016/j.nonrwa.2005.10.003.
- [31] G.P. Samanta. Analysis of a nonautonomous HIV/AIDS epidemic model with distributed time delay. *Math. Model. Anal.*, 15(3):327–347, 2010. Doi:10.3846/1392-6292.2010.15.327-347.
- [32] X.Y. Song and Y.F. Li. Dynamic behaviors of the periodic predatorprey model with modified Leslie-Gower Holling-type II schemes and impulsive effect. Nonlinear Analysis: Real World Applications, 9(1):64–79, 2008. Doi:10.1016/j.nonrwa.2006.09.004.
- [33] E. Venturino. Epidemics in predator prey models: disease in the prey. In O. Arino et al. (Eds.)(Ed.), Mathematical Population Dynamics, Analysis of Heterogeneity, volume 1, pp. 381–393, 1995. Theory of Epidemics. Wuertz Publishing Ltd.: Winnipeg, Canada
- [34] W. Wang and Z. Ma. Global dynamics of an epidemic model with time delay. Nonlinear Anal. RWA, 3:365–373, 2002. Doi:10.1016/S1468-1218(01)00035-9.
- [35] Y.N. Xiao and L.S. Chen. Analysis of a three species eco-epidemiological model. Journal of Mathematical Analysis and Applications, 258(2):733-754, 2001a. Doi:10.1006/jmaa.2001.7514.
- [36] Y.N. Xiao and L.S. Chen. Modeling and analysis of a predator-prey model with disease in the prey. *Math. Biosci.*, **171**:59–82, 2001b. Doi:10.1016/S0025-5564(01)00049-9.

- [37] R. Xu and Z.E. Ma. Global stability of a SIR epidemic model with nonlinear incidence rate and time delay. *Nonlinear Analysis: Real World Applications*, 10(5):3175–3189, 2009a. Doi:10.1016/j.nonrwa.2008.10.013.
- [38] R. Xu and Z.E. Ma. Stability of a delayed SIRS epidemic model with a nonlinear incidence rate. *Chaos, Solitons & Fractals*, 41(5):2319–2325, 2009b. Doi:10.1016/j.chaos.2008.09.007.
- [39] X. Yang, L.S. Chen and J.F. Chen. Permanence and positive periodic solution for the single-species nonautonomous delay diffusive models. *Computers and Mathematics with Applications*, **32**:109–116, 1996. Doi:10.1016/0898-1221(96)00129-0.