The Effect of Time Delay on the Stability of a Diffusive Eco–epidemiological System

Kejun Zhuang

Abstract—The disease has a vital effect on the dynamical behaviors of predator-prey system in ecology. When disease spreads in the prey population, the infected preys are more likely to be captured by predators. On the other hand, spatial diffusion and gestation delay are ubiquitous in the nature and can generate rich spatiotemporal dynamical behaviors. In this study, a delayed and diffusive predator-prey system with disease in the prey is proposed. First, the existence, uniqueness, positivity and boundedness of solutions are established. Second, the stability condition of constant predator-free equilibrium solution is derived. Third, the stability of constant coexistence equilibrium solution and the existence of Hopf bifurcation are investigated by regarding time delay as the bifurcation parameter. Finally, some numerical simulations and conclusions are given to illustrate the theoretical results.

Index Terms—eco-epidemiological system, time delay, Hopf bifurcation, periodic solution.

I. INTRODUCTION

T HE predator-prey system is the basic model in population dynamics, so the interactions between predator and prey in natural ecosystem have drawn extensive attention from scholars in different kinds of fields. As most species are prone to various diseases, abundant improved predatorprey models with disease in the prey or predator have been proposed in the recent past, see [1], [2], [3], [4], [5], [6], [7], [8], [9]. These studies show that the disease in prey or predator species may greatly influence the permanence and stability of the ecosystem, such as the stabilization of predator-prey oscillations, the occurrences of periodic and chaotic oscillations, and so on.

Based on the assumption that the plankton species is only a portion of the food for the fish population in lake ecosystem, the overall fish density depends on the productivity of the lake and does not relate directly with plankton density, Bhattacharyya and Mukhopadhyay [10] established the following epidemiological model with SIS disease in the population:

$$\begin{cases} \frac{dS}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \beta I^p S^q - dS + \gamma I, \\ \frac{dI}{dt} = \beta I^p S^q - dI - \gamma I - \frac{PI^2}{I^2 + h^2}, \end{cases}$$
(1)

where S(t) and I(t) are the densities of susceptible prey and infected prey, respectively. It is assumed that only the susceptible prey is capable of reproducing and its birth rate is r. Infected prey is removed by death or by predation. The disease transmission follows simple mass action law with infected incidence rate β ; p and q are the fractions of infected

Manuscript received February 1, 2018; revised June 16, 2018.

K. Zhuang is with the School of Statistics and Applied Mathematics, Anhui University of Finance and Economics, Bengbu, Anhui, 233030 China, e-mail: zhkj123@163.com. This work is supported by the youth project of the humanities and Social Sciences in the Ministry of Education (17YJC630175), Anhui Province philosophy and social science planning project(AHSKQ2017D01) and the NSF of Education Bureau of Anhui Province (KJ2017A432). and susceptible prey population, respectively (0 < p, q < 1). The prey population has the same natural death rate d and the infected ones suffer additional loss due to recovery at a rate γ and subsequently join the susceptible class. The infected population also suffers loss of biomass due to predator pressure at a rate P. The nonnegative initial conditions are imposed and all the coefficients are positive constants. For system (1), the local and global stabilities of various steady states and existence of Hopf bifurcation behavior were investigated by theoretical analyses and numerical simulations.

However, it is more legitimate to consider the impact of predator if the abundance of large piscivorous fish is increasing in a lake [11]. Through taking account of the density of fish population as a dynamic variable which will significantly influence the dynamics of the system, Chakraborty et al. in [12] considered the extended model as follows:

$$\begin{cases} \frac{dS}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \beta I^p S^q - dS + \gamma I, \\ \frac{dI}{dt} = \beta I^p S^q - dI - \gamma I - \frac{mPI^2}{I^2 + h^2}, \\ \frac{dP}{dt} = \frac{\alpha PI^2}{I^2 + h^2} - \mu P - \sigma P^2, \end{cases}$$
(2)

where P(t) denotes the density of predator population, μ and σP^2 are the natural death rate and the density-dependent mortality rate of predator, respectively. It is a well known fact that the infected prey is more vulnerable and it is assumed that the predator only consumes the infected one. The coefficient m is the maximal per capita consumption rate of infected prey, h represents the amount of infected prey at which predation rate is maximal and α represents the conversion efficiency of consumed prey into new predator. By regarding the infected incidence rate β as the bifurcation parameter, Chakraborty et al. [12] examined the existence of Hopf bifurcation around the coexisting equilibrium and discussed the uniform strong persistence of the system.

In fact, spatial diffusion process is ubiquitous. The majority of populations do not stay in a fixed place and will move from one place to another driven by the outside influences. Spatial diffusion factor can also generate rich dynamics. For instance, stationary pattern, Hopf bifurcation, Turing instability and pattern formation have been recently studied in [13], [14], [15], [16]. On the other hand, time-delay factor cannot be ignored, because the density of predator is closely related to the state at some time before due to the food supply, competition, sexual maturity, and so on. By introducing time delay, ecologists are able to successfully explain regular population cycles.

Nevertheless, there have been comparatively rare results on such eco-epidemic models taking account of both spatial diffusion and time delay. For instance, Mukhopadhyay and Bhattacharyya [17] considered a delay-diffusion predatorprey model with disease in the prey and Holling type-II functional response. They only discussed the linear stability of boundary equilibrium and the dissipativeness of the system. Crauste et al. [18] introduced a delay reactiondiffusion model of the interaction between susceptible fish and bacterium without predator, and analyzed the stability of uniform steady states and existence of Hopf bifurcation. Zhang et al. [19] established a reaction-diffusion model with disease in the prey and ratio-dependent Michaelis-Menten functional response. They considered the temporal-spatial delay and investigated the dynamic properties. Therefore, it is necessary to further study the joint effects of diffusion and delay on the eco-epidemiological systems.

Motivated by the work of [10], [12], [17], [19], in the present paper, we mainly consider the reaction-diffusion predator-prey with disease in the prey and gestation time delay as follows:

$$\begin{cases} \frac{\partial S(x,t)}{\partial t} = \Delta S(x,t) + rS(x,t) \left(1 - \frac{S(x,t) + I(x,t)}{K}\right) \\ -(\beta I(x,t) + d)S(x,t) + \gamma I(x,t), & x \in \Omega, t > 0, \\ \frac{\partial I(x,t)}{\partial t} = \Delta I(x,t) + I(x,t)(\beta S(x,t) - d - \gamma) \\ -\frac{mP(x,t)I^2(x,t)}{I^2(x,t) + h^2}, & x \in \Omega, t > 0, \\ \frac{\partial P(x,t)}{\partial t} = \Delta P(x,t) + \frac{\alpha P(x,t - \tau)I^2(x,t - \tau)}{I^2(x,t - \tau) + h^2} \\ -\mu P(x,t - \tau) - \sigma P^2(x,t - \tau), & x \in \Omega, t > 0, \\ \frac{\partial S(x,t)}{\partial n} = \frac{\partial I(x,t)}{\partial n} = \frac{\partial P(x,t)}{\partial n} = 0, & x \in \partial\Omega, t > 0, \\ S(x,t) = S_1(x,t) \ge 0, & x \in \overline{\Omega}, t \in [-\tau,0], \\ I(x,t) = I_1(x,t) \ge 0, & x \in \overline{\Omega}, t \in [-\tau,0], \\ P(x,t) = P_1(x,t) \ge 0, & x \in \overline{\Omega}, t \in [-\tau,0], \end{cases}$$
(3)

where S(x,t), I(x,t) and P(x,t), respectively, represent the densities of susceptible prey, infected prey and predator at the location $x \in \Omega$ and time t; $\tau > 0$ is the time required for the gestation of the predator. The region $\Omega \subset \mathbb{R}^N (N \leq 3)$ denotes a bounded domain with smooth boundary $\partial\Omega$; Δ is the Laplace operator; $\partial/\partial n$ indicates the outward normal derivative on $\partial\Omega$. The system is subject to homogeneous Neumann boundary conditions, which means that the ecoepidemiological system is self contained and the populations cannot cross the boundary. Unlike the infection incidence function in systems (1) and (2), here, we adopt the classic bilinear function βSI for simplicity.

The main purpose of this paper is to provide a new perspective for both ecologists and mathematicians. More concretely, we shall focus on the asymptotic stability of the predator-free and coexisting steady states, and the existence of Hopf bifurcation around the coexisting steady state induced by time delay. The rest of the paper is organized as follows. In Section 2, we investigate the fundamental properties of solutions for system (3). In Section 3, we carry out the linear stability of two uniform steady states and the existence of Hopf bifurcation by analyzing the corresponding characteristic equations. In Section 4, we conduct some numerical simulations in support of the analytical findings. Finally, we draw some conclusions in the Section 5.

II. FUNDAMENTAL PROPERTIES OF SOLUTIONS

In this section, we shall establish the well-posedness of solutions for system (3), including the existence, uniqueness, positivity and boundedness of the solutions.

First, we denote the Banach space of continuous functions from $[-\tau, 0]$ into X with the usual supremum norm by $C = C([\tau, 0], X)$. In our case, X is the Banach space $C(\overline{\Omega}, \mathbb{R}^3)$ and C(E, F) represents the space of continuous functions

from topological space E into space F. For convenience, we identify an element $\varphi \in C$ as a function from $\overline{\Omega} \times [-\tau, 0]$ into \mathbb{R}^3 defined by $\varphi(x, s) = \varphi(s)(x)$.

For any continuous function $w(.) : [-\tau, b) \to X$ for b > 0, we define $w_t \in C$ by $w_t(s) = w(t+s)$, $s \in [-\tau, 0]$. It is easy to find that $t \to w_t$ is a continuous function from [0, b)to C.

Proposition 1 For any nonnegative initial conditions of system (3), there exists a unique solution and this solution remains nonnegative and bounded for any $t \ge 0$.

Proof of Proposition 1: For any $\varphi = (\varphi_1, \varphi_2, \varphi_3)^T \in C$ and $x \in \overline{\Omega}$, we define $F = (F_1, F_2, F_3) : C \to X$ by

$$F_{1}(\varphi)(x) = rS(x,0) \left(1 - \frac{S(x,0) + I(x,0)}{K}\right) -(\beta I(x,0) + d)S(x,0) + \gamma I(x,0),$$

$$F_{2}(\varphi)(x) = I(x,0)(\beta S(x,0) - d - \gamma) - \frac{mP(x,0)I^{2}(x,0)}{I^{2}(x,0) + h^{2}},$$

$$F_{3}(\varphi)(x) = \frac{\alpha P(x,-\tau)I^{2}(x,-\tau)}{I^{2}(x,-\tau) + h^{2}} - \mu P(x,-\tau) -\sigma P^{2}(x,-\tau).$$

Then, system (3) can be rewritten as the abstract functional differential equation:

$$\begin{cases} w'(t) = Aw + F(w_t), \quad t > 0, \\ w(0) = \phi \in X, \end{cases}$$
(4)

where

$$w = (S, I, P)^{T},$$

$$\phi = (S_1, I_1, P_1)^{T},$$

$$Aw = (\Delta S, \Delta I, \Delta P)^{T}.$$

It is clear that F is locally Lipschitz in X. According to the results in [20], [21], [22], [23], [24], we can conclude that system (4) admits a unique local solution on $[0, T_{max})$, where T_{max} is the maximal existence time for solution of system (4).

Besides, we can also obtain that $S(x,t) \ge 0$, $I(x,t) \ge 0$ and $P(x,t) \ge 0$ because $\mathbf{O} = (0,0,0)$ is a lower solution of system (3).

Next, we verify the boundedness of solutions. In fact, we only need to prove that any solution is uniformly bounded. It is easy to see that $\mathbf{M} = (M_1, M_2, M_3)$ is a upper solution of system (3), where

$$\begin{split} M_1 &= \max\left\{K, \sup_{-\tau \le t \le 0} \parallel S_1(\cdot, s) \parallel_{C(\overline{\Omega}, \mathbb{R})}\right\}, \\ M_2 &= \max\left\{K, \sup_{-\tau \le t \le 0} \parallel I_1(\cdot, s) \parallel_{C(\overline{\Omega}, \mathbb{R})}\right\}, \\ M_3 &= \max\left\{\frac{\alpha}{\sigma h^2}, \sup_{-\tau \le t \le 0} \parallel P_1(\cdot, s) \parallel_{C(\overline{\Omega}, \mathbb{R})}\right\}. \\ \text{Based on the comparison principle, we have} \end{split}$$

$$0 \le S(x,t) \le M_1, 0 \le I(x,t) \le M_2, 0 \le P(x,t) \le M_3$$

for $x \in \overline{\Omega}$ and $t \in [0, T_{max})$. Then the solutions of (3) are bounded on $\overline{\Omega} \times [0, T_{max})$. From the stand theory for semilinear parabolic systems in [25], we deduce that $T_{max} = +\infty$. The proof is complete.

III. STABILITY OF UNIFORM STEADY STATES

As we know, spatial diffusion and time delay do not change the number and location of the uniform steady states. With the similar method in [12], we can obtain the predator-free equilibrium $E_0 = (S_0, I_0, 0)$ and the coexisting equilibrium $E^* = (S^*, I^*, P^*)$, where

$$S_{0} = \frac{d + \gamma}{\beta},$$

$$I_{0} = \frac{(d + \gamma)(K\beta r - K\beta d - rd - r\gamma)}{\beta(K\beta d + rd + r\gamma)},$$

$$S^{*} = \frac{\sqrt{b^{2} - 4ac} - b}{2a},$$

$$P^{*} = \frac{1}{\sigma} \left(\frac{\alpha I^{*2}}{I^{*2} + h^{2}} - \mu\right),$$

$$a = r,$$

$$b = rI^{*} + \beta KI^{*} + dK - Kr,$$

$$c = -\gamma KI^{*},$$

and I^* is the positive root of the following equation

$$r\left\{1 - \frac{I^*}{K} - \left[d + \gamma + \frac{m\alpha I^{*3}}{\sigma (I^{*2} + h^2)^2} - \frac{m\mu I}{\sigma (I^{*2} + h^2)}\right]\right\}$$
$$-d + \gamma I^* \left[d + \gamma + \frac{m\alpha I^{*3}}{\sigma (I^{*2} + h^2)^2} - \frac{m\mu I}{\sigma (I^{*2} + h^2)}\right]^{-1} = 0.$$

Next, we will investigate the asymptotic stability of the two uniform steady states.

We first introduce some useful concepts from [24]. Let $0 = \mu_0 < \mu_1 < \mu_2 < \cdots$ denote the eigenvalues of the operator $-\Delta$ in Ω under homogeneous Neumann boundary conditions and $s(\mu_k)$ be the eigenfunction space corresponding to μ_k with dimension number $n_k = \dim[s(\mu_k)]$ in $C^1(\overline{\Omega})$. (i) $\mathbf{X}_{\mathbf{k}} := \{\sum_{j=1}^{n_k} c_j \varphi_{kj} : c_j \in \mathbb{R}\}$, where $\{\varphi_{kj}\}_{j=1}^{n_k}$ are an orthogonal basis of $s(\mu_k)$. (ii) $\mathbf{X} := \{(S, I, P) \in C^1(\overline{\Omega}) \times C^1(\overline{\Omega}) \times C^1(\overline{\Omega}) : \frac{\partial S}{\partial \mathbf{n}} = \frac{\partial I}{\partial \mathbf{n}} = \frac{\partial P}{\partial \mathbf{n}} = 0$ on $\partial \Omega\}$, so that $\mathbf{X} = \bigoplus_{k=0}^{\infty} \mathbf{X}_{\mathbf{k}}$.

A. Stability of predator-free equilibrium

Theorem 1 If $K\beta r > K\beta d + rd + r\gamma$, then for any $\tau \ge 0$, the predator-free equilibrium E_0 is asymptotically stable when $\alpha I_0^2 < \mu(I_0^2 + h^2)$ or unstable when $\alpha I_0^2 > \mu(I_0^2 + h^2)$.

Proof of Theorem 1: The linearization of system (3) at the predator-free equilibrium $E_0 = (S_0, I_0, 0)$ can be expressed by

$$Y_t = (E\Delta + F_Y(E_0))Y,$$

where E is the unit matrix, $Y = (S(x,t), I(x,t), P(x,t))^T$, and

$$= \begin{pmatrix} -\frac{\gamma I_0}{S_0} - \frac{rS_0}{K} & -\frac{r(d+\gamma)}{K\beta} - d & 0\\ \beta I_0 & 0 & -\frac{mI_0}{I_0^2 + h^2}\\ 0 & 0 & \frac{\alpha I_0^2}{I_0^2 + h^2} e^{-\lambda\tau} - \mu \end{pmatrix}.$$

For $k \ge 0$, it is observed that $\mathbf{X}_{\mathbf{k}}$ is invariant under the operator $E\Delta + F_Y(E_0)$ and λ is an eigenvalue of $E\Delta + F_Y(E_0)$ on $\mathbf{X}_{\mathbf{k}}$ if and only if λ is an eigenvalue of the matrix $-\mu_k E + F_Y(E_0)$. That is

$$\left(\lambda + \mu_k - \frac{\alpha I_0^2}{I_0^2 + h^2} e^{-\lambda \tau} + \mu\right)$$

$$\left[\lambda^2 + \left(2\mu_k + \frac{\gamma I_0}{S_0} + \frac{rS_0}{K}\right)\lambda + \frac{r(d+\gamma)I_0}{K} + d\beta I_0\right] = 0.$$
It is apparent that all roots of the quadratic equation
$$\lambda^2 = \left(\lambda - \frac{\gamma I_0}{S_0} - \frac{rS_0}{K}\right)\lambda = 2$$

$$\lambda^2 + \left(2\mu_k + \frac{\gamma I_0}{S_0} + \frac{\gamma S_0}{K}\right)\lambda + \mu_k^2$$
$$+\mu_k \left(\frac{\gamma I_0}{S_0} + \frac{rS_0}{K}\right) + \frac{r(d+\gamma)I_0}{K} + d\beta I_0 = 0$$

must have strictly negative real parts. Then we only need to consider the following transcendental equation:

$$\lambda + \mu_k - \frac{\alpha I_0^2}{I_0^2 + h^2} e^{-\lambda \tau} + \mu = 0.$$
 (5)

When $\tau = 0$, the root of (5) is $\lambda = -\mu_k + \frac{\alpha I_0^2}{I_0^2 + h^2} - \mu$. We can find that $\lambda < 0$ for any $k \ge 0$ when $\alpha I_0^2 < \mu (I_0^2 + h^2)$, and $\lambda > 0$ for some $k \ge 0$ when $\alpha I_0^2 > \mu (I_0^2 + h^2)$.

When $\tau > 0$, assume that $\lambda = i\omega \ (\omega > 0)$ is a root of (5), and we have

$$\frac{\alpha I_0^2}{I_0^2 + h^2} \left(\cos \omega \tau - i \sin \omega \tau \right) = \mu_k + \mu + i\omega.$$

Separating the real and imaginary parts leads to

$$\begin{cases} \frac{\alpha I_0^2}{I_0^2 + h^2} \cos \omega \tau = \mu_k + \mu, \\ \frac{\alpha I_0^2}{I_0^2 + h^2} \sin \omega \tau = -\omega, \end{cases}$$

and

$$\omega^{2} = \left(\frac{\alpha I_{0}^{2}}{I_{0}^{2} + h^{2}} + \mu_{k} + \mu\right) \left(\frac{\alpha I_{0}^{2}}{I_{0}^{2} + h^{2}} - \mu_{k} - \mu\right).$$
 (6)

If $\alpha I_0^2 < \mu (I_0^2 + h^2)$, then equation (6) has no positive root and equation (5) has no purely imaginary root. According to the Corollary 2.4 in [26], as parameter τ varies, the sum of the orders of the roots of (5) in the open right half plane can change only if a root appears on or crosses the imaginary axis. As a consequence, equation (5) has roots only with negative real part if the condition $\alpha I_0^2 < \mu (I_0^2 + h^2)$ holds. It is known that the constant equilibrium solution is asymptotically stable only if all the characteristic values have strictly negative parts. Thus, the proof is complete.

The asymptotic stability of predator-free equilibrium implies the extinction of predator. Therefore, from Theorem 1, it can be observed that the predator population may extinct when its death rate μ is large or the conversion efficiency coefficient α is sufficiently small.

B. Existence of Hopf bifurcation around the positive equilibrium

The asymptotic stability of positive equilibrium implies the coexistence of both predator and prey species, which

would be helpful for the population conservation and the sustainable development of ecosystem. Consequently, we are more interested in the effect of time delay on the stability of the coexisting equilibrium E^* . Here, we concentrate on the stability of positive equilibrium and the existence of Hopf bifurcation by regarding time delay τ as the bifurcation parameter.

Linearizing system (3) at the positive equilibrium E^* , we can obtain the characteristic equation

$$\begin{vmatrix} \lambda + \mu_k + a_{11} & a_{12} & 0 \\ a_{21} & \lambda + \mu_k + a_{22} & a_{23} \\ 0 & a_{32}e^{-\lambda\tau} & \lambda + \mu_k + a_{33}e^{-\lambda\tau} + b_{33} \\ = 0, \qquad (7)$$

where

$$a_{11} = \frac{\gamma I^*}{S^*} + \frac{rS^*}{K},$$

$$a_{12} = \frac{r}{K}S^* + \beta S^* - \gamma,$$

$$a_{21} = -\beta I^*,$$

$$a_{22} = \frac{mP^*I^*}{I^{*2} + h^2} - \frac{2mp^*I^{*3}}{(I^{*2} + h^2)^2},$$

$$a_{23} = \frac{m(\mu + \sigma)}{\alpha},$$

$$a_{32} = \frac{2\alpha h^2 P^* I^{*2}}{(I^{*2} + h^2)^2},$$

$$a_{33} = -\frac{\alpha I^{*2}}{I^{*2} + h^2},$$

 $b_{33} = \mu - 2\sigma P^*.$

Then the characteristic equation (7) can be reduced to

$$\lambda^3 + A_k \lambda^2 + B_k \lambda + C_k + e^{-\lambda \tau} (D_k \lambda^2 + F_k \lambda + G_k) = 0,$$
(8)

where

$$\begin{split} A_k &= 3\mu_k + a_{11} + a_{22} + b_{33}, \\ B_k &= 3\mu_k^2 + 2(a_{11} + a_{22} + b_{33})\mu_k + a_{11}a_{22} \\ &\quad + a_{22}b_{33} + a_{11}b_{33} - a_{12}a_{21}, \\ C_k &= \mu_k^3 + (a_{11} + a_{22} + b_{33})\mu_k^2 + (a_{11}a_{22} + a_{11}b_{33} \\ &\quad + a_{22}b_{33} - a_{12}a_{21})\mu_k + a_{11}a_{22}b_{33} - a_{12}a_{21}b_{33}, \\ D_k &= a_{33}, \\ F_k &= 2a_{33}\mu_k + a_{11}a_{33} + a_{22}a_{33} - a_{23}a_{32}, \\ G_k &= a_{33}\mu_k^2 + (a_{11}a_{33} + a_{22}a_{33} - a_{23}a_{32})\mu_k \end{split}$$

$$+a_{11}a_{22}a_{33}-a_{11}a_{23}a_{32}-a_{12}a_{21}.$$

The expressions of the coefficients in equation (8) are too complex, so we can only derive the general conditions for stability of the positive equilibrium E^* . The detailed numerical calculations will be left in the next section. Then, we discuss the distribution of characteristic values in equation (8).

For $\tau = 0$, the characteristic equation (8) can be rewritten as

$$\lambda^{3} + (A_{k} + D_{k})\lambda^{2} + (B_{k} + F_{k})\lambda + (C_{k} + G_{k}) = 0.$$
(9)

By Routh-Hurwitz criterion, all roots of the cubic equation (9) have strictly negative real parts if and only if the following conditions hold:

$$(H_1)$$
 $A_k + D_k > 0;$
 (H_2) $B_k + F_k > 0;$

 $(H_3) \quad C_k + G_k > 0;$

 $(H_4) \quad (A_k + D_k)(B_k + F_k) - (C_k + G_k) > 0.$

Given this, the positive coexisting equilibrium E^* is locally asymptotically stable without time delay.

On the other hand, we discuss the effect of time delay τ on the stability of the positive equilibrium. Assume that $\lambda = i\omega$ $(\omega > 0)$ is a root of (8). Substituting it into the equation can yield

$$-i\omega^3 - A_k\omega^2 + iB_k\omega + C_k + (-D_k\omega^2 + iF_k\omega + G_k)(\cos\omega\tau - i\sin\omega\tau) = 0.$$
⁽¹⁰⁾

Segregating the real and imaginary parts of equation (10), we have

$$\begin{cases} (D_k\omega^2 - G_k)\sin\omega\tau + F_k\omega\cos\omega\tau = \omega^3 - B_k\omega, \\ F_k\omega\sin\omega\tau - (D_k\omega^2 - G_k)\cos\omega\tau = A_k\omega^2 - C_k. \end{cases}$$
(11)

Taking square on both sides of the equations of (11) and summing them up, we can obtain

$$\omega^{6} + (A_{k}^{2} - 2B_{k} - D_{k}^{2})\omega^{4} + Q_{k}\omega^{2} + C_{k}^{2} - G_{k}^{2} = 0,$$
(12)

where $Q_k = B_k^2 - 2A_kC_k + 2D_kG_k - F_k^2$.

Let $z = \omega^2$, then equation (12) can be transformed into a cubic equation of z in the form of

$$\Lambda(z) = z^3 + (A_k^2 - 2B_k - D_k^2)z^2 + Q_k z + C_k^2 - G_k^2 = 0.$$
(13)

We make the following hypothesis:

 $(H_5) \quad C_k - G_k < 0.$

Assume that (H_3) and (H_5) hold. It is easy to show that $C_k^2 - G_k^2 < 0$. Hence, equation (13) has at least one positive root due to the Descartes' rule of singes [27]. Without loss of generality, we assume that equation (13) has three positive roots and denote any one by z^* . Then equation (12) has positive root $\omega^* = z^{*2}$, from which we can deduce that the characteristic equation (8) may have a pair of purely imaginary roots $\lambda = \pm i\omega^*$ under certain condition.

Solving the equations of (11), we get

$$\begin{cases} \cos \omega \tau = \frac{(F_k - A_k D_k)\omega^4 + (A_k G_k + C_k D_k - B_k F_k)\omega^2 - C_k G_k}{F_k^2 \omega^2 + (D_k \omega^2 - G_k)^2}, \\ \sin \omega \tau = \frac{D_k \omega^5 + (A_k F_k - B_k D_k - G_k)\omega^3 + (B_k G_k - C_k F_k)\omega}{F_k^2 \omega^2 + (D_k \omega^2 - G_k)^2}. \end{cases}$$

Therefore, if we denote

$$\tau_j^* = \frac{1}{\omega^*} \left\{ \arccos \frac{(F_k - A_k D_k) \omega^4 + S_k \omega^2 - C_k G_k}{F_k^2 \omega^2 + (D_k \omega^2 - G_k)^2} \right\},$$

where $S_k = A_k G_k + C_k D_k - B_k F_k$ and $j = 0, 1, 2, \cdots$, then $\pm i\omega^*$ is a pair of purely imaginary roots of (8) at $\tau = \tau_j^*$.

To guarantee the occurrence of Hopf bifurcation, we still need to verify the transversal condition. Taking the derivatives of (8) with respect to τ results in

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{3\lambda^2 + 2A_k\lambda + B_k + e^{-\lambda\tau} \left(2D_k\lambda + F_k\right)}{\lambda e^{-\lambda\tau} \left(D_k\lambda^2 + F_k\lambda + G_k\right)} - \frac{\tau}{\lambda}.$$
(14)

Substituting $\lambda = i\omega^*$ and $\tau = \tau_j^*$ into (14), we have

$$\left[\frac{d(\operatorname{Re}\lambda(\tau))}{d\tau}\right]_{\lambda=i\omega^*,\tau=\tau_j^*}^{-1} = \frac{HK - LT}{{\omega^*}^2(J^2 + K^2)},$$

where

 $H = -3\omega^{*2} + B_k,$ $J = 2A_k\omega^*,$ $K = G_k - D_k\omega^{*2},$ $L = F_k\omega^*,$ $M = H\sin\omega^*\tau_j^* + J\cos\omega^*\tau_j^* + 2D_k\omega^*,$ $T = H\cos\omega^*\tau_j^* - J\sin\omega^*\tau_j^* + F_k.$

Then we can obtain the transversal condition

$$\left[\frac{d(\operatorname{Re}\lambda(\tau))}{d\tau}\right]_{\lambda=i\omega^*,\tau=\tau_i^*}^{-1} > 0$$

when the following inequality

$$(H_6) \quad HK - LT > 0$$

is satisfied.

Again based on the significant results in [26], we can find that if the assumptions $(H_1) - (H_6)$ are satisfied, then all roots of characteristic equation (8) have negative real parts for $\tau \in [0, \tau_0^*)$. Moreover, a pair of purely imaginary roots exist for $\tau = \tau_0^*$ and a pair of roots with positive real parts will appear for $\tau > \tau_0^*$. By applying the Hopf bifurcation theorem in [24], the conclusions about the stability of positive equilibrium and existence of the Hopf bifurcation can be drawn as follows.

Theorem 2 If the assumptions $(H_1) - (H_6)$ are all satisfied, then the following statements are true.

(i) The positive equilibrium E^* is locally asymptotically stable when $\tau \in [0, \tau_0^*)$.

(ii) The positive equilibrium E^* is unstable when $\tau > \tau_0^*$. The Hopf bifurcation occurs at $\tau = \tau_0^*$. That is, system (3) has a series of periodic solutions around E^* when τ is slightly larger than τ_0^* .

IV. NUMERICAL SIMULATIONS

In this section, we shall give some numerical examples to describe the previous theoretical results with the help of Mathematica and MATLAB. Here, we consider the system in one dimensional space $\Omega = (0, \pi)$ for simplicity.

First, we choose

$$r = 1.5, K = 100, d = 0.003, \gamma = 0.05, \alpha = 0.09,$$

$$\mu = 0.5, \sigma = 0.1, h = 15, \beta = 0.45, m = 1.5, \tau = 2.5.$$

Then the predator-free equilibrium is $E_0 = (0.1178, 36.9452, 0)$, and the conditions in Theorem 1

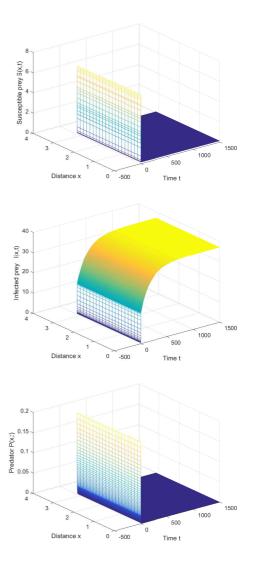


Fig. 1. The predator-free equilibrium is stable when $\tau = 2.5$.

are satisfied. It is to be noted that the boundary equilibrium E_0 is asymptotically stable (see Figure 1).

Second, we rechoose

$$r = 1.5, K = 100, d = 0.03, \gamma = 0.05, \alpha = 0.9,$$

$$\mu = 0.001, \sigma = 0.1, h = 15, \beta = 0.45, m = 1.5.$$

Then the coexisting equilibrium is $E^* = (9.8745, 24.7625, 6.2011)$. We can also derive the first Hopf bifurcation critical value $\tau_0^* = 1.61$ for k = 0. When time delay is smaller than the critical value, the positive equilibrium is asymptotically stable (see Figure 2). Otherwise, when time delay is slightly larger than the critical value, the positive equilibrium becomes unstable and periodic solution will bifurcate from E^* (see Figure 3).

V. CONCLUSIONS

In this paper, we have considered a delay-diffusion predator-prey system with disease in the prey. The model, which incorporates the spatial diffusion and time delay effects, is much more generalized than those in [10], [12]. As pointed in [12], the consideration of a disease in the prey or predator population makes the system extremely complex.

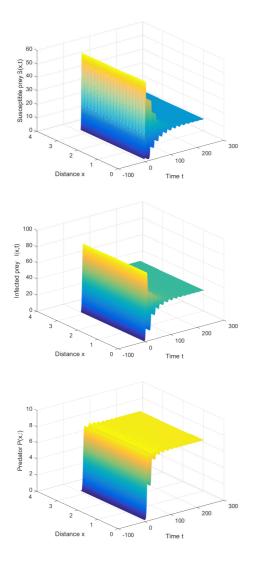


Fig. 2. The positive equilibrium is stable when $\tau = 0.3 < \tau_0^*$.

Despite all this, the dynamical behaviors in such systems still have a guidance function to the ecological diversity.

It is observed from our results that the uniform predatorfree steady state is asymptotically stable under certain condition, but the stability has nothing to do with the gestation delay. However, this stability is harmful, as the predator population will be ultimately extinct over time. To avoid this phenomenon, some measures may be taken to reduce the mortality of predator population or increase the food sources.

It can also be observed that the gestation delay has crucial impact on the asymptotic stability of the uniform coexisting steady state. The stability of positive equilibrium is not affected by time delay when it is sufficiently small. However, the stability changes when the gestation delay is larger than some critical value and spatially periodic solution will arise. According to these facts, we can know that both time delay and spatial diffusion can generate periodic pattern and play an important role in spatiotemporal dynamics. Therefore, in order to maintain the stability of the ecosystem, it is better to shorten the gestation delay of predator population.

Of course, the methods and results in this paper can be applied to other reaction-diffusion systems. We hope that our work could be instructive to both theoretical and applied

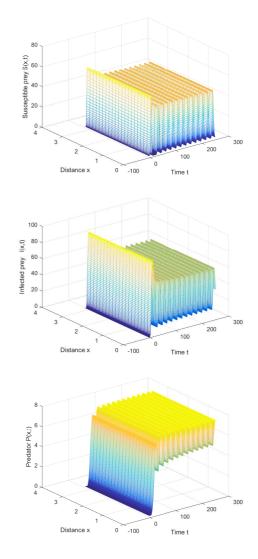


Fig. 3. The positive equilibrium is unstable and periodic solution appears when $\tau = 2.5 > \tau_0^*$.

ecologists.

References

- J. Chattopadhyay and O. Arino, "A predator-prey model with disease in the prey," *Nonlinear Analysis*, vol. 36, no. 6, pp. 747-766, 1999.
- [2] Y. N. Xiao and L. S. Chen, "Modeling and analysis of a predator-prey model with disease in the prey," *Mathematical Biosciences*, vol. 171, no. 1, pp. 59-82, 2001.
- [3] M. Haque and E. Venturino, "The role of transmissible diseases in the Holling-Tanner predator-prey model," *Theoretical Population Biology*, vol. 70, no. 3, pp. 273-288, 2006.
- [4] F. M. Hilker and K. Schmitz, "Disease-induced stabilization of predator-prey oscillations," *Journal of Theoretical Biology*, vol. 255, no. 3, pp. 299-306, 2008.
- [5] G. P. Hu and X. L. Li, "Stability and Hopf bifurcation for a delayed predator-prey model with disease in the prey," *Chaos, Solitons and Fractals*, vol. 45, no. 3, pp. 229-237, 2012.
- [6] W. J. Du, S. Qin, J. G. Zhang and J. N. Yu, "Dynamical behavior and bifurcation analysis of SEIR epidemic model and its discretization," *IAENG International Journal of Applied Mathematics*, vol. 47, no. 1, pp. 1-8, 2017.
- [7] K. Das, "Disease-induced chaotic oscillations and its possible control in a predator-prey system with disease in predator," *Differential Equations & Dynamical Systems*, vol. 24, no. 2, pp. 215-230, 2016.
- [8] H. Singh, J. Dhar and H. S. Bhatti, "Dynamics of a prey-generalized predator system with disease in prey and gestation delay for predator," *Modeling Earth Systems & Environment*, vol. 2, no. 2, pp. 1-9, 2016.

- [9] S. Olaniyi, M. A. Lawal and O. S. Obabiyi, "Stability and sensitivity analysis of a deterministic epidemiological model with pseudorecovery," *IAENG International Journal of Applied Mathematics*, vol. 46, no. 2, pp. 160-167, 2016.
- [10] R. Bhattacharyya and B. Mukhopadhyay, "On an epidemiological model with nonlinear infection incidence: Local and global perspective," *Applied Mathematical Modelling*, vol. 35, no. 7, pp. 3166-3174, 2011.
- [11] S. R. Carpenter, J. F. Kitchell and J. R. Hodgson, "Cascading trophic interactions and lake productivity," *Bioscience*, vol. 35, no. 10, pp. 634-639, 1985.
- [12] K. Chakraborty, K. Das, S. Haldar and T. K. Kar, "A mathematical study of an eco–epidemiological system on disease persistence and extinction perspective," *Applied Mathematics and Computation*, vol. 254, pp. 99-112, 2015.
- [13] J. J. Li and W. J. Gao, "Analysis of a prey-predator model with disease in prey," *Applied Mathematics and Computation*, vol. 217, no. 8, pp. 4024-4035, 2010.
- [14] X. S. Tang and Y. L. Song, "Stability, Hopf bifurcations and spatial patterns in a delayed diffusive predator-prey model with herd behaviour," *Applied Mathematics and Computation*, vol. 254, pp. 375-391, 2015.
- [15] H. Y. Zhao, X. X. Huang and X. B. Zhang, "Turing instability and pattern formation of neural networks with reaction-diffusion terms," *Nonlinear Dynamics*, vol. 76, no. 1, pp. 115-124, 2014.
- [16] T. Wang, "Pattern dynamics of an epidemic model with nonlienar incidence rate," *Nonlinear Dynamics*, vol. 77, no. 1-2, pp. 31-40, 2014.
- [17] B. Mukhopadhyay and R. Bhattacharyya, "Dynamics of a delaydiffusion prey-predator model with disease in the prey," *Journal of Applied Mathematics and Computing*, vol. 17, no. 1-2, pp. 361-377, 2005.
- [18] F. Crauste, M. L. Hbid and A. Kacha, "A delay reaction-diffusion model of the dynamics of botulinum in fish," *Mathematical Biosciences*, vol. 216, no. 1, pp. 17-29, 2008.
- [19] X. L. Zhang, Y. H. Huang and P. X. Weng, "Stability and bifurcation of a predator-prey model with disease in the prey and temporal-spatial nonlocal effect," *Applied Mathematics and Computation*, vol. 290, pp. 467-486, 2016.
- [20] C. C. Travis and G. F. Webb, "Existence and stability for partial functional differential equations," *Transactions of the American Mathematical Society*, vol. 200, no. 7, pp. 395-418, 1974.
- [21] W. E. Fitzgibbon, "Semilinear functional differential equations in Banach space," *Journal of Differential Equations*, vol. 29, no. 1, pp. 1-14, 1978.
- [22] R. H. Martin and H. L. Smith, "Abstract functional differential equations and reaction-diffusion systems," *Transactions of the American Mathematical Society*, vol. 321, no. 1, pp. 1-44, 1990.
- [23] R. H. Martin and H. L. Smith, "Reaction-diffusion systems with time delays: monotonicity, invariance, comparison and convergence," J. *Reine Anges. Math.*, vol. 413, pp. 1-35, 1991.
- [24] J. H. Wu, Theory and Applications of Partial Functional Differential Equations. New-York: Springer-Verlag, 1996.
- [25] D. Henry, Geometric theory of semilinear parabolic equations. New-York: Springer-Verlag, 1993.
- [26] S. G. Ruan J. J. Wei, "On the zeros of transcendental functions with applications to stability of delay differential equations with two delays," *Dynamics of Continuous, Discrete and Impulsive Systems Series A: Mathematical Analysis*, vol. 10, no. 6, pp. 863-874, 2003.
- [27] R. Descartes, *The Philosophical Writings of Descartes*. Cambridge: Cambridge University Press, 1985.
- [28] S. N. Wu, J. P. Shi and B. Y. Wu, "Global existence of solutions and uniform persistence of a diffusive predator-prey model with preytaxis," *Journal of Differential Equations*, vol. 260, no. 7, pp. 5847-5874, 2016.
- [29] J. M. Lee, T. Hillen and M. A. Lewis, "Pattern formation in prey-taxis system," *Journal of Biological Dynamics*, vol. 3, no. 6, pp. 551-573, 2009.