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CHAOTIC EFFECTS ON DISEASE SPREAD IN A SIMPLE ECO-EPIDEMIOLOGICAL SYSTEM*

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Abstract In this paper, an eco-epidemiological model where prey disease is structured as a susceptible-infected model is investigated. Thresholds that control disease spread and population persistence are obtained. Existence, stability and instability of the system are studied. Hopf bifurcation is shown to occur where a periodic solution bifurcates from the coexistence equilibrium. Simulations show that the system exhibits chaotic phenomena when the transmission rate is varied.

Keywords Eco-epidemiological, Hopf bifurcation, chaos.

MSC(2010) 92D30, 92D25.

1. Introduction

Both general compartmental epidemic models and classic Lotka-Volterra (LV) models [3, 14, 22] have been investigated by many authors during the recent years. In most cases, these two types of models are separately considered. To model the spread of disease in ecologically interacting populations, we need to examine epidemic models where the local species is subject to an ecological interaction such as competition with another species or predation. The emerging models combining epidemics with ecological interactions, termed eco-epidemiological models, are not only biologically meaningful but also can show rich dynamics. Anderson and May [1] were the pioneers who connected the LV models with epidemic models and built a prey-predator model where the prey spices are infected by a diseases. Since then, a myriad of results on eco-epidemiological systems [2, 4–6, 8–12, 15, 17, 23–25] have appeared.

Earlier research was devoted to the study of stability and persistence of a system. Now ecologists and mathematicians are interested in studying models exhibiting much more complex dynamics, such as chaos, strange attractors, fractal and so on. Allen [2] investigated an eco-epidemiological model indicating that chaos can prevent global population extinction if there are several distinct subpopulations that are weakly coupled by migration and subject to a locally varying external noise. Stiefs *et al* showed that for certain classes of eco-epidemiological models

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quasiperiodic and chaotic dynamics was generic and likely to occur [17]. Chatterjee et al [11] proposed two eco-epidemiological models with mass incidence and standard incidence rate, respectively. Their results showed that both models can exhibit chaos and standard incidence can enlarge the stable region compared with the mass incidence.

As mentioned above, some eco-epidemiological systems can exhibit chaotic phenomena; however, results in this respect are rare. For a general eco-epidemiological model, Hopf bifurcation may occur at the coexistence equilibrium and sustained oscillations are possible [13]. Bate and Hilker built in [7] an SI-type disease in the predator population of a Rosenzweig-MacArthur model and then found a wealth of complex dynamics that did not exist in the absence of the disease. Upadhyay and Roy in [21] proposed an eco-epidemiological model with simple law of mass action and modified Holling type II functional response to understand how a disease may spread among natural populations. They showed that the death rate of a predator and the growth rate of a susceptible prey may produce rich complex dynamics. Sieber et al in [19] considered an eco-epidemiological model that allowed for differential competition amongst and between infected and uninfected prey individuals. They found that disease-induced modifications of competition can tremendously alter the stability and persistence of predator-prey systems.

The aim of this paper is to find a mechanism that results in chaos for a general simple eco-epidemiological model. We use logistic growth rate in the prey population and we assume that the predator predates at different rates on the susceptible and infective prey. The small variance between the predation rates can lead to the system exhibiting chaos.

The paper is organized as follows. In Section 2, we propose an eco-epidemiological model and give some results stemming from the biological application. In Section 3, we obtain the existence of equilibria of the system. In Section 4, we obtain some sufficient conditions for the asymptotical stability of the equilibria, and we find the existence of Hopf bifurcation around the coexistence equilibrium. In Section 5, numerical simulations are carried out to illustrate the validity of the main results. The paper ends with a discussion.

2. Model Development

In [9], Chattopadhyay and Arino introduced a disease into the prey population. They classify the population into three classes, susceptible prey $S(t)$, infected prey $I(t)$ and predator $P(t)$. They also assume that the disease is transmitted only in the prey and does not affect the predator. The infected prey population doesn't recover. The incidence rate is of mass action type given by βSI. We assume that the infected prey can reproduce and that all the newborns are susceptible. The infected prey population competes for the limit resources with the susceptible prey population. The predator population can predate both on the susceptible and on the infected prey populations. The predator functional response follows Holling II type, $\frac{\gamma_S SP}{a_1+S}$ and $\frac{\gamma_I IP}{a+I}$, respectively. The model is as follows:

$$\begin{cases} \frac{dS}{dt} = r(S+I)(1-\frac{S+I}{K}) - \frac{\gamma_S SP}{a_1+S} - \beta SI, \\ \frac{dI}{dt} = \beta SI - \frac{\gamma_I IP}{a+I} - \mu_0 I, \\ \frac{dP}{dt} = \varepsilon(\frac{\gamma_S S}{a_1+S} + \frac{\gamma_I I}{a+I})P - dP. \end{cases} \tag{2.1}$$

They show that Hopf bifurcation appears when they assume that r is large enough and γ_S is small enough. In order to show the importance of reproduction of infected prey as one of key mechanisms, we apply a linear predator response for Chattopadhyay and Arino proposed model. This model is changed as follows.

$$\begin{cases} \dfrac{dS}{dt} = r(S+I)(1-\dfrac{S+I}{K}) - \gamma_S SP - \beta SI, \\ \dfrac{dI}{dt} = \beta SI - \gamma_I IP - \mu_0 I, \\ \dfrac{dP}{dt} = \varepsilon(\gamma_S S + \gamma_I I)P - dP, \end{cases} \tag{2.2}$$

where γ_S and γ_I are the predation rates of susceptible and infected prey, respectively. β is the transmission rate of the disease in the prey. μ_0 is the natural death rate or the disease-induced death rate. The detailed explanation of the parameters is listed in the following table.

Table 1. The meanings of the parameters in (2.2)

Symbol	Definition	Value
r	prey growth rate	2
K	prey carrying capacity	1000
β	Transmission rate	varied
μ_0	disease-induced death rate	0.001
ε	predator conversion efficiency	0.2
γ_S	attack rate on susceptible prey	9.1
γ_I	attack rate on infectious prey	1.0
d	predator death rate	0.1

For biological reasons, all parameters are positive and constant. (2.2) has nonnegative initial conditions $S(0) = S_0 > 0, I(0) \geq 0, P(0) \geq 0$.

Define

$$\Gamma = \{(S,I,P) | S \geq 0, I \geq 0, P \geq 0, S+I \leq K, P \leq \frac{\varepsilon(\gamma_S+\gamma_I)K}{d}\}.$$

Lemma 2.1. Γ *is a positive invariant set for system* (2.2) *with nonnegative initial conditions.*

Proof. From the second and third equation of (2.2), we have

$$I(t) = I_0 e^{\int_0^t (\beta S(s) - \gamma_I P(s) - \mu_0)ds}, \quad P(t) = P_0 e^{\int_0^t (\varepsilon(\gamma_S S(s) + \gamma_I I(s)) - d)ds}.$$

If $I_0 \geq 0$ and $P_0 \geq 0$, then $I(t) \geq 0$ and $P(t) \geq 0$. Denote $x = S + I$, $\underline{\gamma} = \min\{\gamma_S, \gamma_I\}$, and $\bar{\gamma} = \max\{\gamma_S, \gamma_I\}$. Adding the first two equations of (2.2) yields

$$\frac{dx}{dt} \leq rx(1 - \frac{\underline{\gamma}}{r}P - \frac{x}{K}).$$

Hence, it follows from the non-negativity of P and $x_0 = S_0 + I_0 \leq K$ that $x \leq K$. We show the non-negativity of $S(t)$ by contradiction. It follows the continuity of $S(t)$ and $S_0 > 0$ that there exist a

$$t^* = \liminf\{t | S(t) < 0\}.$$

So, $S(t^*) = 0$, $S(t) > 0$ for $0 \le t < t^*$ and $S'(t^*) < 0$. By the first equation of (2.2) and $I(t) \le K$, we have

$$\frac{dS(t^*)}{dt} = I(t^*)(1 - \frac{I^*}{K}) \ge 0.$$

This leads to a contradiction. Therefore, $S(t) \ge 0$. Substituting $S \le K$ and $I \le K$ into the third equation of (2.2) yields

$$P(t) \le \frac{\varepsilon(\gamma_S + \gamma_I)K}{d}.$$

□

In the remaining part of this paper, we focus on the limit behaviors of (2.2) with initial conditions starting from Γ.

3. Equilibria

In this section, we derive some thresholds and we show that system (2.2) has five equilibria whose existence depends on the thresholds. The thresholds also determine the persistence of the disease or the persistence of the system. For the convenience, define

$$\begin{aligned} &R_0 = \tfrac{\beta K}{\mu_0}, R_1 = \tfrac{\varepsilon(\gamma_S S_3 + \gamma_I I_3)}{d}, R_P = \tfrac{\beta d}{\varepsilon\gamma_S(\mu_0 + \gamma_I P_2)}, \\ &R_0^P = \tfrac{K\varepsilon\gamma_S}{d}, R_2 = \tfrac{K\varepsilon\gamma_I}{d}, \end{aligned} \tag{3.1}$$

where S_2, I_3 and P_2 are the elements of equilibria defined below. Note that equilibria of (2.2) satisfy the following equations

$$\begin{cases} 0 = r(S+I)(1 - \frac{S+I}{K}) - \gamma_S SP - \beta SI, \\ 0 = \beta SI - \gamma_I IP - \mu_0 I, \\ 0 = \varepsilon(\gamma_S S + \gamma_I I)P - dP. \end{cases} \tag{3.2}$$

Solving (3.2) yields five equilibria. The first four equilibria are defined as $E_0 = (0,0,0), E_1 = (K,0,0), E_2 = (S_2,0,P_2) = (\frac{d}{\varepsilon\gamma_S}, 0, \frac{rd(R_0^P-1)}{K\varepsilon\gamma_S^2})$, $E_3 = (S_3, I_3, 0) = (\frac{\mu_0}{\beta}, \frac{x^*}{\beta}, 0)$ where x^* is the solution of

$$rx^2 + (K\beta\mu_0 - K\beta r + 2\mu_0 r)x + \mu_0^2 r(1 - R_0) = 0.$$

Summarizing the above discussion, we have the following theorem.

Theorem 3.1. *If $R_0 < 1, R_0^P < 1$, (2.2) have two equilibria E_0 and E_1. If $R_0 > 1$, and $R_0^P < 1$, (2.2) has three equilibria E_0 E_1 and E_3. If $R_0 < 1$, and $R_0^P > 1$, (2.2) has three equilibria E_0 E_1 and E_2. If $R_0 > 1$, and $R_0^P > 1$, (2.2) has four equilibria E_0 E_1 E_2 and E_3.*

The coexistence equilibrium is given as $E_4 = (S_4, I_4, P_4)$, where $S_4 = \frac{d}{\varepsilon\gamma_S} - \frac{\gamma_I}{\gamma_S} I_4 \triangleq f(I_4)$, $P_4 = \frac{1}{\gamma_I}(\beta f(I_4) - \mu_0)$. I_4 satisfies the following equation

$$F(I) \triangleq r(f(I) + I)(1 - \frac{f(I) + I}{K}) = \frac{\gamma_S}{\gamma_I} f(I)(\beta f(I) - \mu_0) + \beta f(I) I \triangleq G(I). \tag{3.3}$$

If $I = 0$, then

$$F(0) = r\frac{d}{\varepsilon\gamma_S}(1 - \frac{d}{\varepsilon\gamma_S K}) = rS_2(1 - \frac{S_2}{K}) = \gamma_S S_2 P_2$$

and

$$G(0) = \frac{\gamma_S}{\gamma_I}\frac{d}{\varepsilon\gamma_S}(\beta\frac{d}{\varepsilon\gamma_S} - \mu_0) = \frac{\gamma_S}{\gamma_I}S_2(\beta S_2 - \mu_0).$$

Hence if $R_P > 1$, then $F(0) < G(0)$.

Let $\hat{I}$ be the solution of $\beta f(I) - \mu_0 = 0$. Then we have $f(\hat{I}) = \frac{\mu_0}{\beta}$. Substituting this expression into F and G yields

$$\begin{aligned} F(\hat{I}) &= r(\tfrac{\mu_0}{\beta} + \hat{I})(1 - \tfrac{\frac{\mu_0}{\beta}+\hat{I}}{K}), \\ G(\hat{I}) &= \mu_0\hat{I}. \end{aligned} \tag{3.4}$$

If $\hat{I} < I_3$, then $F(\hat{I}) > G(\hat{I})$ and there exists only one positive solution I_4 of (3.3). This solution satisfies $I_3 > \hat{I} > I_4$. If $\hat{I} > I_3$, then $F(\hat{I}) < G(\hat{I})$. This implies that (3.3) has no solution or two positive solutions. If they exist, then $I_4^- < I_4^+ < I_3 < \hat{I}$ or $I_4^- < I_3 < I_4^+$ and $I_3 < \hat{I}$, where I_4^- and I_4^+ are the smaller and the larger solutions of (3.3), respectively.

$\hat{I} = \frac{d/(\varepsilon\gamma_S) - S_3}{\gamma_I/\gamma_S} > (<) I_3$ implies that $R_1 = \frac{\varepsilon(\gamma_I I_3 + \gamma_S S_3)}{d} < (>) 1$. Summarizing the above discussion, we have the following theorem.

Theorem 3.2. *Let $R_P > 1$ always hold, then if $R_1 < 1$* (3.3) *has only one unique positive equilibrium $I_4 < \hat{I} < I_3$; if $R_1 > 1$,* (3.3) *has no equilibria or has two positive equilibria which satisfy $I_4^- < I_4^+ < I_3 < \hat{I}$ or $I_4^- < I_3 < I_4^+$ and $I_3 < \hat{I}$.*

For the direction of the bifurcation, we define

$$\mathcal{F}(\beta, I) = F(\beta, I) - G(\beta, I) = a_1 I^2 + a_2(\beta)I + a_3(\beta),$$

here

$$\begin{aligned} a_1 &= -\tfrac{r}{K}(1 - \tfrac{\gamma_I}{\gamma_S})^2, \\ a_2(\beta) &= r(1 - \tfrac{\gamma_I}{\gamma_S})(1 - \tfrac{2d}{\varepsilon\gamma_S K}) + \tfrac{\beta d}{\varepsilon\gamma_S} - \mu_0, \\ a_3(\beta) &= \tfrac{d}{\varepsilon}[\tfrac{r}{\gamma_S}(1 - \tfrac{d}{\varepsilon\gamma_S K}) - \tfrac{1}{\gamma_I}(\tfrac{\beta d}{\varepsilon\gamma_S} - \mu_0)]. \end{aligned}$$

Therefore,

$$\frac{d\beta}{dI} = (\frac{dI}{d\beta}|_{I=0})^{-1} = -\frac{\frac{da_3}{d\beta}}{a_2} = \frac{d^2}{\varepsilon^2\gamma_I\gamma_S a_2}. \tag{3.5}$$

Remark 3.1. If $R_P > 1$, $R_1 > 1$, even if $a_2 > 0$, then (2.2) may have two positive equilibria. These results are very different from backward bifurcation.

In order to illustrate Remark 3.1, we set $r = 0.5, K = 2, \gamma_I = 0.1, \gamma_S = 1.3, \epsilon = 1, d = 0.5, \mu = 0.1$. Using matcont package we note that I_4 bifurcates from I_3 at a bifurcation point(BP) $(0.277778, 1.388889, 0.000000)$ when we set $\beta = 0.36$. Figure 1 shows that there exist two endemic equilibria when β varies from 0.36 to 0.415 (see Figure 1). When we set $\beta = 0.4$ which is between 0.36 and 0.415, system (2.2) exhibits two positive equilibria $(0.6126, 0.486, 1.4505)$ and $(0.5654, 1.1, 1.2615)$ and $I_4^- = 0.486 < I_4^+ = 1.1 < I_3$. This verifies Theorem 3.2. When $\beta = 0.35$

which is between 0.34 and 0.36, system (2.2) still has two positive equilibria given by $(0.6455, 0.058, 1.2594)$ and $(0.5394, 1.438, 0.8878)$. Figure 1 shows that $I_4^- = 0.058 < I_3 < I_4^+ = 1.438$. When $\beta = 0.2$, system (2.2) only has one positive equilibrium $(0.5246, 1.63, 0.5738)$ and $I_3 < I_4 = 1.63$.

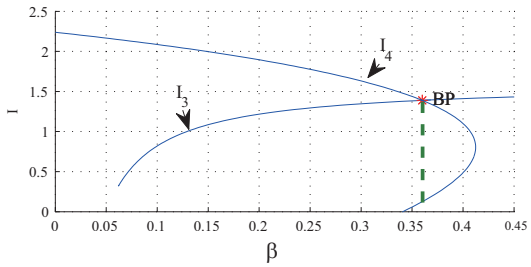


Figure 1. The number of equilibria of system (2.2) with respect to β. System (2.2) has one equilibrium when β varies from 0 to 0.34 while system (2.2) has two equilibria when β varies from 0.34 to 0.415.

4. Local stability

The stability of these equilibria is determined by the Jacobian matrix evaluated at the corresponding equilibrium. The general Jacobian matrix is given by

$$\begin{pmatrix} r(1-\frac{2(S+I)}{K}) - \gamma_S P - \beta I & r(1-\frac{2(S+I)}{K}) - \beta S & -\gamma_S S \\ \beta I & -\gamma_I P + \beta S - \mu_0 & -\gamma_I I \\ \varepsilon\gamma_S P & \varepsilon\gamma_I P & \varepsilon(\gamma_S S + \gamma_I I) - d \end{pmatrix}, \tag{4.1}$$

which determines the local stability of equilibria of system (2.2).

Theorem 4.1. *(1) The trivial equilibrium E_0 is always unstable.*

(2) If $R_0 < 1$ and $R_0^P < 1$, the disease free and non-predator equilibrium E_1 is locally asymptotically stable.

(3) If $R_P < 1$, the disease free equilibrium E_2 is locally asymptotically stable.

(4) If $R_1 < 1$, the non-predator equilibrium E_3 is locally asymptotically stable.

(5) The stability of the coexistence equilibrium E_4 is determined by (4.4).

Proof.

1. For the equilibrium E_0 of (2.2), the characteristic roots of (4.1) are $r, -\mu_0, -d$, respectively. It is always unstable since there is a positive characteristic root of (4.1) $r > 0$.

2. For the equilibrium E_1 of (2.2), the characteristic roots of (4.1) are $-r, \mu_0(R_0 - 1), d(R_0^P - 1)$, respectively. Hence if $R_0 < 1$ and $R_0^P < 1$, it is locally asymptotically stable.

3. For the equilibrium E_2 of (2.2), one of the characteristic roots of (4.1) is $(\mu_0 + \gamma_I P_2)(R_P - 1)$. The other characteristic roots are determined by

$$\lambda^2 + \frac{rS_2}{K}\lambda + \varepsilon\gamma_S^2 S_2 P_2 = 0. \tag{4.2}$$

It is easy to show that (4.2) just has roots with negative real parts. The stability of (4.1) is determined by the value of R_P. If $R_P < 1$, E_2 is locally asymptotically stable, otherwise, E_2 is unstable.

4. For the equilibrium E_3 of (2.2), one of the characteristic roots of (4.1) is $d(R_1 - 1)$. The other characteristic roots are determined by

$$\lambda^2 + (\beta I_3 - r(1 - \frac{S_3 + I_3}{K}) + r\frac{S_3 + I_3}{K})\lambda + \beta I_3[\beta S_3 - r[1 - \frac{S_3 + I_3}{K}] + r\frac{S_3 + I_3}{K}] = 0. \tag{4.3}$$

It follows from $\beta I_3 - r(1 - \frac{S_3+I_3}{K}) = r\frac{I_3}{S_3}(1 - \frac{S_3+I_3}{K}) > 0$ and $\beta S_3 - r(1 - \frac{S_3+I_3}{K}) = r\frac{S_3}{I_3}(1 - \frac{S_3+I_3}{K}) > 0$ that (4.3) just has roots with negative real parts. The stability of (4.1) is determined by the value of R_1. If $R_1 < 1$, E_3 is locally asymptotically stable, otherwise, E_3 is unstable.

5. For the stability of E_4, the characteristic equation of (2.2) is

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{4.4}$$

where

$$\begin{aligned}
a_1 &= \gamma_S P_4 + \beta I_4 - r(1 - \tfrac{2(S_4+I_4)}{K}) = r(1 - \tfrac{S_4+I_4}{K})\tfrac{I_4}{S_4} + r\tfrac{S_4+I_4}{K} > 0,\\
a_2 &= \varepsilon P_4(\gamma_S^2 S_4 + \gamma_I^2 I_4) + \beta I_4(\beta S_4 - r(1 - \tfrac{2(S_4+I_4)}{K})),\\
a_3 &= \varepsilon\gamma_I P_4 I_4[\gamma_I(\gamma_S P_4 + \beta I_4) + (\gamma_S - \gamma_I)r(1 - \tfrac{2(S_4+I_4)}{K})].
\end{aligned}$$

□

By the Routh Hurwitz Criterion, we have the following theorem.

Theorem 4.2. *If $a_2 > 0$, $a_3 > 0$, and $a_1a_2 - a_3 > 0$, then the coexistence equilibrium E_4 is locally asymptotically stable.*

We set the parameters as defined in Figure 1 and plot the real parts of the characteristic roots of (2.2) with respect to the transmission rate β. Figure 2 shows that two positive equilibria of (2.2) are locally asymptotically stable when β varies from 0.34 to 0.415. System (2.2) exhibits one local asymptotic equilibrium when β varies from 0.325 to 0.34 and from 0 to 0.225. However, the equilibrium of (2.2) is unstable if β varies from 0.225 to 0.325.

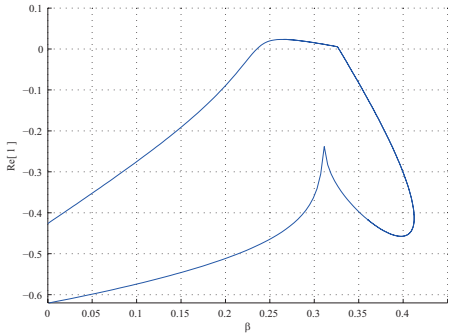


Figure 2. The real parts of the characteristic roots of system (2.2) with respect to the transmission rate β varied from 0 to 0.415.

Remark 4.1. If $K/2 \leq S_4 + I_4 \leq K$, and $\gamma_S > \gamma_I$, then a_3 may be less than 0 and $a_1a_2 - a_3 > 0$. in addition $S_4 + I_4 < K/2$ and $\gamma_S > \gamma_I$, then $a_3 > 0$ and $a_1a_2 - a_3$ may be less or equal to 0. There exists a Hopf bifurcation bifurcating from the coexistence equilibrium E_4.

Theorem 4.3. *If $R_0 < 1$ and $R_0^P < 1$, then the disease-free and predator-free equilibrium E_1 is globally asymptotically stable.*

Proof. Note that $S \leq K$. From the second equation of (2.2),

$$\frac{dI}{dt} \leq (\beta K - \mu_0)I = \mu_0(R_0 - 1)I,$$

If $R_0 < 1$, then $\lim_{t\to\infty} I(t) = 0$. Thus, there exist a t_0 such that for all $t > t_0$ and any small ϵ_1, we have $0 \leq I(t) < \epsilon_1$. It follows from the third equation of (2.2) and for all $t > t_0$ that

$$\frac{dP}{dt} \leq [\varepsilon(\gamma_S K + \gamma_I \epsilon_1) - d]P.$$

If $R_0^P < 1$, it follows from the arbitrary choice of ϵ_1 that $\lim_{t\to\infty} P(t) = 0$. Hence, there exist a t_1 and a small ϵ_2 such that $P \leq \epsilon_2$ for all $t > t_1$. Then, for all $t > \max\{t_0, t_1\}$ we have

$$\begin{aligned} &\tfrac{dS}{dt} \leq r(S + \epsilon_1)(1 - \tfrac{S}{K}), \\ &\tfrac{dS}{dt} \geq rS(1 - \tfrac{S+\epsilon_1}{K}) - \beta S\epsilon_1 - \gamma_S S\epsilon_2. \end{aligned}$$

Thus,

$$S \leq \frac{Ke^{A(\epsilon_1)}}{e^{A(\epsilon_1)} + 1} + \frac{\epsilon_1 e^{A(\epsilon_1)}}{e^{A(\epsilon_1)} + 1} - \epsilon_1$$

and

$$S \geq \frac{B(\epsilon_1, \epsilon_2)e^{B(\epsilon_1,\epsilon_2)t/K}}{CA - re^{B(\epsilon_1,\epsilon_2)t/K}},$$

where $A(\epsilon_1) = r(C + t)(1 + \epsilon/K)$ and $B(\epsilon_1, \epsilon_2) = K\beta\epsilon_1 + K\gamma_S\epsilon_2 - Kr + \epsilon_1 r$. For t large enough and the fact that ϵ_1 and ϵ_2 are arbitrary, we have

$$\lim_{t\to\infty} S(t) = K.$$

Therefore, E_1 is a global attractor with respect to Γ if $R_0 < 1$ and $R_0^P < 1$. Combining this result with Theorem 4.2 indicates that E_1 is globally asymptotically stable.

□

Define $\bar{\gamma} = \max\{\gamma_S, \gamma_I\}, \underline{\gamma} = \min\{\gamma_S, \gamma_I\}$, $\bar{P} = \frac{r(1-\frac{d}{\varepsilon K\bar{\gamma}})}{\underline{\gamma}}$, and $R'_P = \frac{\beta d}{\varepsilon\gamma_S\mu_0}$.

Theorem 4.4. *If $R'_P < 1$, then the disease is eliminated from system* (2.2)*. System* (2.2) *evolves as the general LV prey-predator model.*

Proof. Adding the first and the second equation of (2.2) yields

$$\frac{d(S + I)}{dt} = r(S + I)(1 - \frac{S + I}{K}) - (\gamma_S S + \gamma_I I)P. \tag{4.5}$$

Combining (4.5) with the third equation of (2.2) yields the following inequality

$$\begin{aligned} &\tfrac{d(S+I)}{dt} \le r(S+I)(1-\tfrac{S+I}{K}) - \underline{\gamma}(S+I)P, \\ &\tfrac{dP}{dt} \le \varepsilon\bar{\gamma}(S+I)P - dP. \end{aligned} \tag{4.6}$$

We can define an auxiliary systems as follows

$$\begin{aligned} &\tfrac{dx}{dt} = rx(1-\tfrac{S+I}{K}) - \underline{\gamma}xP, \\ &\tfrac{dP}{dt} = \varepsilon\bar{\gamma}(S+I)P - dP. \end{aligned} \tag{4.7}$$

$\bar{E}_2 = (\bar{x}, \bar{P}) = (\frac{d}{\varepsilon\bar{\gamma}}, \frac{r(1-\frac{d}{\varepsilon K\bar{\gamma}})}{\underline{\gamma}})$ denotes the equilibrium of (4.7) where $x = S + I$. Define a Lyapunov function

$$V = x - \bar{x} - \bar{x}\ln x + \frac{\underline{\gamma}}{\varepsilon\bar{\gamma}}(P - \bar{P} - \bar{P}\ln P).$$

Differentiating V along the solution of (4.7) yields

$$\begin{aligned} V' &= \tfrac{x-\bar{x}}{x}x' + \tfrac{\underline{\gamma}}{\varepsilon\bar{\gamma}}\tfrac{P-\bar{P}}{P}P' \\ &\le -\tfrac{r}{K}(x-\bar{x})^2 \\ &\le 0. \end{aligned}$$

By the LaSalle Invariance Principle, $\bar{E}_2$ is globally asymptotically stable. Therefore, $\limsup_{t\to\infty} x \le \bar{x}$ and $\limsup_{t\to\infty} P \le \bar{P}$. For any small ϵ_1, there exist a t_1 such that for all $t > t_1$, we have

$$x \le \bar{x} + \epsilon_1 = \frac{d}{\varepsilon\bar{\gamma}_S} + \epsilon_1, \quad P \le \bar{P} = \frac{r(1-\frac{d}{\varepsilon K\bar{\gamma}})}{\underline{\gamma}} + \epsilon_1.$$

By the second equation of (2.2), we have

$$I' \le [\beta(\frac{d}{\varepsilon\bar{\gamma}} + \epsilon_1) - \gamma_I P - \mu_0]I \le [\beta(\frac{d}{\varepsilon\gamma_S} + \epsilon_1) - \mu_0]I.$$

If $R'_P < 1$ then $I(t) \to 0$ as $t \to \infty$. □

In order to investigate the persistence of the disease, define a Poincare map $\pi : \mathbb{R}^3 \to \mathbb{R}$ as

$$\pi(S, I, P) = I.$$

Denote by $M_0 = \{(S, I, P) | \pi(S, I, P) \neq 0\}$, and $\partial M = \Gamma/M_0$. We have the following theorem.

Theorem 4.5. *If $R'_P > 1$, the disease is weekly persistent; that is, $\limsup_{t\to\infty} I(t) > \epsilon_1$.*

Proof. We claim $\limsup_{t\to\infty} I(t) > \epsilon$. By contradiction, assume that for all $(S_0, I_0, P_0) \in M_0$ we have

$$\limsup_{t\to\infty} I(t) \le \epsilon_1.$$

Then there exists a t_0 such that for all $t > t_0$, we have $0 \le I(t) \le \epsilon_1$. It follows from the first and third equation that

$$\begin{aligned} \frac{dS}{dt} &\ge rS(1 - \tfrac{S+\epsilon}{K}) - \gamma_S SP - \beta S\epsilon, \\ \frac{dP}{dt} &\ge \varepsilon\gamma_S SP - dP. \end{aligned} \tag{4.8}$$

According to the proof of Theorem 4.4 and the arbitrary choice of ϵ_1, we have $\liminf_{t\to\infty} S(t) \ge S_2$ and $\liminf_{t\to\infty} P(t) \ge P_2$. Hence, there exists a t_1 and a small ϵ_2 such that for all $t > t_1$, we have

$$S(t) > S_2 - \epsilon_2 = \frac{d}{\varepsilon\gamma_S} - \epsilon_2.$$

Substituting these expressions into the second equation of (2.2) yields

$$\frac{dI}{dt} \ge (\beta(S_2 - \epsilon_2) - \mu_0)I.$$

If $R'_P > 1$, then $I(t) \to \infty$ as $t \to \infty$. This leads to a contradiction with the boundedness of I.

□

Lemma 4.1 ([16, Theorem 3.4.6]). *If $T(t) : X \to X$, $t \in \mathbb{R}_+$ is asymptotically smooth, point dissipative and orbits of bounded sets are bounded, then there exists a global attractor.*

It is easy to check that system (2.2) is asymptotically smooth, point dissipative and orbits of bounded sets are bounded. By the Theorem 2.3 of [20] we have the following theorem.

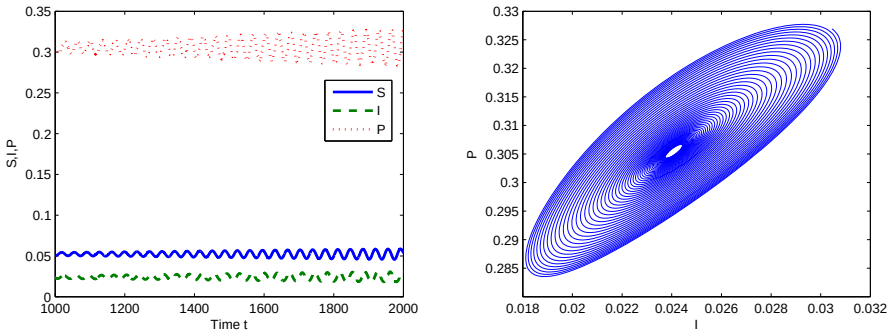
Theorem 4.6. *If $R'_P > 1$, then the disease is strongly persistent; that is, $\liminf_{t\to\infty} I(t) > \epsilon_1$.*

5. Simulations

Due to the complexity of system (2.2), numerical integration is a good choice to study the dynamics of (2.2). The simulations are performed with the help of Malab 2013b. The main purpose of this paper is to show how the disease influences the dynamics of the prey-predator system. Combining Theorem 3.2 and Remark 4.1 we obtain that, if $\gamma_S > \gamma_I$, (2.2) may exhibit complex dynamics. We will investigate the complex behavior of system (2.2) with respect to the transmission rate β and the other parameters fixed. The parameter values are listed in Table 1.

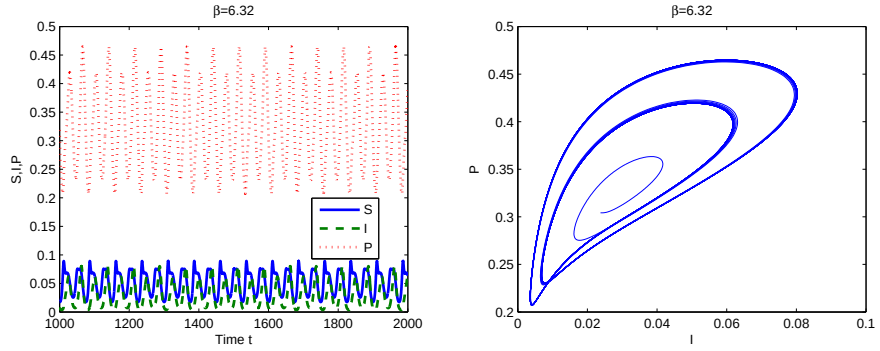
For $\beta = 5.86$ system (2.2) exhibits a stable limit cycle near the coexistence equilibrium E_4. The period of the limit cycle is only equal to 1. This implies that the periodic orbit makes one loop around the central point before starting to repeat itself. Figure 3 illustrates this phenomenon.

Increasing the transmission rate β to 6.32. A periodic solution of period two bifurcates from the periodic solution of period one of system (2.2). This indicates that there exists a solution that loops twice around the coexistence equilibrium E_4. Figure 4 illustrates the results. As we know, there is a general route that the chaotic behavior is produced by a series of period doubling solutions. Enlarging the



(a) Time series of S, I and P (b) Plane of IP

Figure 3. Figure shows the solution with initial conditions $S_0 = 0.05, I_0 = 0.024, P_0 = 0.304$ and parameters as listed in Table 1. The value of the parameter $\beta = 5.86$.

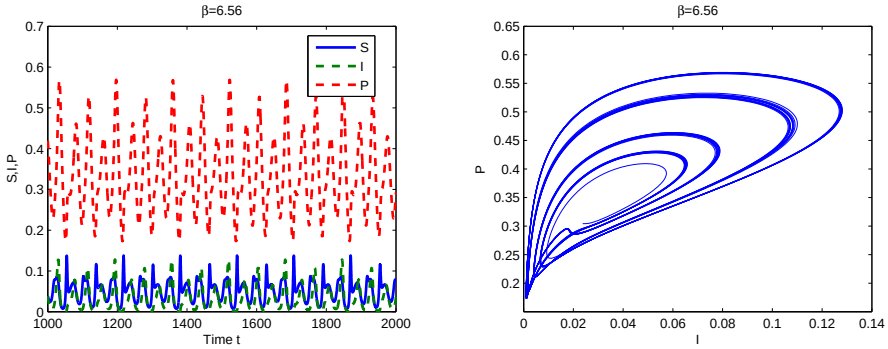


(a) Time series of S, I and P from 1000 to 2000 (b) Plane of IP

Figure 4. Figure shows the solution with initial conditions $S_0 = 0.05, I_0 = 0.024, P_0 = 0.304$ and the parameters as listed in Table 1. The value of the parameter $\beta = 6.32$.

transmission rate to 6.57, Figure 5 shows that the periodic solution with period two bifurcates into a periodic solution of period four. From mathematical perspective, continuing to enlarge the transmission rate β, system (2.2) must exhibit complex dynamics. To understand the route to chaos, a systematic investigation of the dynamics was done by constructing a bifurcation diagram. Figure 6(a) implies that system (2.2) produces chaos which is characterized with aperiodic behavior and sensitive dependence on the initial conditions. Figure 6(b) shows that the attractor of system (2.2) has two wings, which largely reside on the SP and the IP planes; projection of the attractor on the SI plane is minimal. The random spikes modeling outbreak of the disease are instead of the endemicity. This phenomenon is also happening for the predator population which coincides with the spikes of the infected prey.

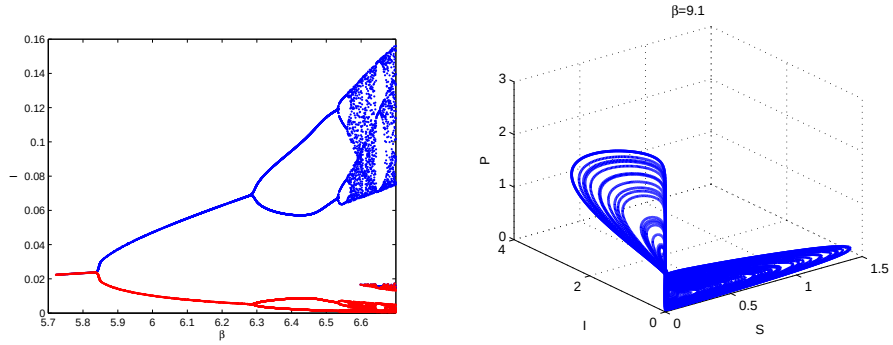
As we know, an important property of chaotic dynamics for a system is its sensitivity to initial conditions. Lyapunov exponents are commonly used to measure the mean rate of exponential divergence of neighboring trajectories. If the largest Lyapunov exponent of a trajectory is negative, then the trajectory is stable, while a



(a) Time series of S, I and P from 1000 to 2000

(b) Plane of IP

Figure 5. Figure shows solutions with initial conditions $S_0 = 0.05, I_0 = 0.024, P_0 = 0.304$ and the parameters are listed in Table 1. The value of parameter $\beta = 6.56$.



(a) Bifurcation diagram for system (2.2) with β varied from 5.5 to 6.7

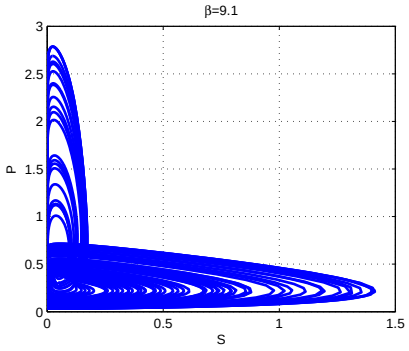
(b) The attractor of system (2.2) with $\beta = 9.1$

Figure 6. Figure shows solutions with initial conditions $S_0 = 0.05, I_0 = 0.024, P_0 = 0.304$ and the parameters are listed in Table 1. The value of the parameter β varies.

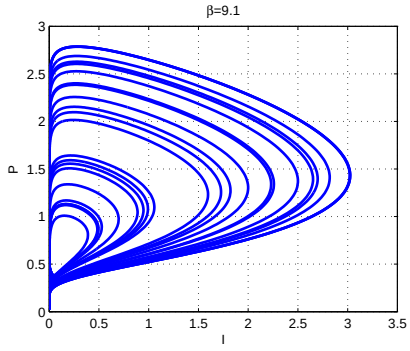
trajectory with the largest Lyapunov exponent as zero is periodic, but if the largest Lyapunov exponent is positive then that trajectory is chaotic.

Set $\beta = 6.7$. Figure 8(a) clearly shows that the largest Lyapunov exponent is larger than 0, other two Lyapunov exponents are 0 and negative, respectively. Therefore, the strange attractor is chaotic. In order to investigate the sensitivity of the strange attractor with respect to initial conditions, we take two different initial conditions $S_0 = 0.052335, I_0 = 0.024, P_0 = 0.304$, and $S_0 = 0.052335001, I_0 = 0.024, P_0 = 0.304$. Figure 8(b) implies that system (2.2) under two different initial conditions (very close to each other) leads to different dynamics. As the time evolves, system (2.2) becomes undetermined. That indicates that slight perturbation in species numbers may result in unpredictable dynamics as the time evolves.

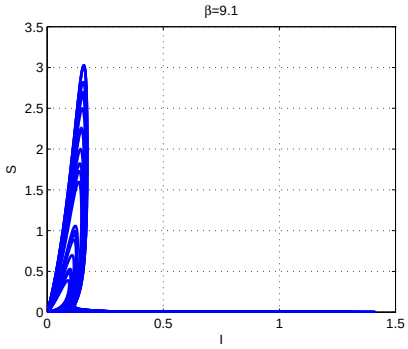
When β ranges from 5.85 to 6.29, system (2.2) exhibits bifurcation of a limit cycle from the coexistence equilibrium E_4. A limit cycle bifurcates into a period-doubling solution when the transmission rate changes from 6.3 to 6.57. A period-double solution evolves to a period-four solution when the transmission rate varies from 6.57 to 6.58. System (2.2) exhibits chaotic phenomena when $\beta > 6.58$.



(a) SP plane of system (2.2) with $\beta = 9.1$

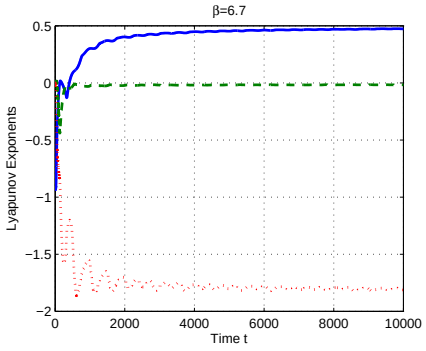


(b) IP plane of system (2.2) with $\beta = 9.1$

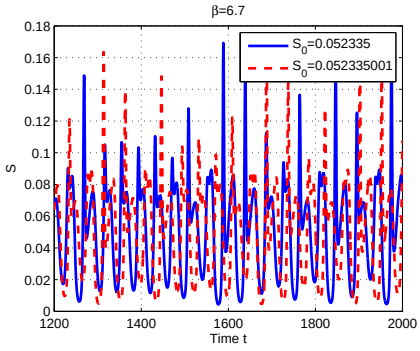


(c) SI plane of system (2.2) with $\beta = 9.1$

Figure 7. Figure shows solution with initial conditions $S_0 = 0.05, I_0 = 0.024, P_0 = 0.304$ and the parameters are listed in Table 1.



(a)



(b)

Figure 8. The parameters are listed in Table 1 and parameter $\beta = 6.7$, (a) The spectrum of Lyapunov exponents for system (2.2) around the strange attractor with initial conditions $S_0 = 0.052335, I_0 = 0.024, P_0 = 0.304$; (b) The divergence of trajectories for the susceptible prey population with different values $S_0 = 0.052335$ (solid blue line) and $S_0 = 0.052335001$(dash red line) keeping initial values of infectious prey population and predator population fixed and the same.

6. Conclusion

In this paper, we simplify an eco-epidemiological model with prey infected by a disease proposed by Chattopadhyay and Arino [9]. We use the mass law instead of the Holling II type predator response. Compared with the existent literature on eco-epidemiological models, Hopf bifurcation is a common phenomena for eco-epidemiological models [8–11, 15, 17, 23, 24]. However, Chaotic phenomena is scarce compared with the Hopf bifurcation phenomena occurring in ecp-epidemiological models [7, 18, 19, 21, 26]. In these models, they use various nonlinear incidence rates or nonlinear predator functional response. The results presented here appear to challenge previous studies [7, 9, 19, 21]. We show that reproduction of infected prey is one of key mechanisms leading to chaotic phenomena in eco-epidemiological systems if the intrinsic rate is not large enough. This implies that diseases spreading in a prey population may result in a complex evolution of the prey-predator population. It follows from numerical results that the transmission rate has been identified as a key parameter to control dynamics of system (2.2). For a fixed environment, this implies that all the parameters except the transmission rate are fixed. Controlling the disease spread in prey guarantees the balance of the environment.

The existence and local stability of the equilibria are obtained though analyzing the algebraic equations (3.1) and the characteristic equations with respect to the equilibria, respectively. Because the system is still rather complex to be analyzed analytically, for the stability of the coexistence equilibrium E_4 we mainly rely on numerical results. The long time dynamical behaviors of system (2.2) are revealed by the numerical integration. It follows from Section 5 that stable equilibrium, Hopf bifurcation of a limit cycle, period-doubling of the solution and chaotic behavior occur as the transmission rate β varies. In order to confirm whether or not the simulation leads to chaotic trajectories, we plot the Lyapunov exponents which indicate that system (2.2) exhibits chaotic behavior under parameters taken in Table 1 and $\beta = 6.7$.

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