Dynamics of Disease Spread in a Predator-Prey System

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Abstract

This paper discusses the dynamics of a communicable disease in a predator-prey system. It is assumed that a parasite is infectious and it spreads among preys according to a SIS (*susceptible-infective-susceptible*) model but not among predators, the predators stay healthy. Two predation response functions of Holling type II are used for both healthy and infected preys. The feasibility and stability conditions of the equilibrium points of the system are analyzed. The model has one trivial equilibrium (E_1^*) and two free-disease positive equilibria; (i) in the absence of predator (E_2^*) and (ii) in the presence of predator (E_3^*). The threshold condition of E_1^* is derived. If it is below threshold, E_1^* becomes stable and if above threshold, it is unstable. The basic reproduction numbers for E_2^* and E_3^* are

also derived, called R_{02} dan R_{03} , respectively. These basic numbers serve as thresholds. If $R_{02} < 1$ (or $R_{03} < 1$), the equilibrium point E_2^* (or E_3^*) becomes asymptotically locally stable. If $R_{02} > 1$ (or $R_{03} > 1$), the equilibrium E_2^* (or E_3^*) is unstable.

Keywords: SIS, predator-prey model, next generation matrix, stability

1. Introduction

Many examples of a predator-prey interaction among species can be easily observed in ecological system throughout the world, such as a fox-rabbit relation. In a normal life, predator and prey species exhibit regular cycles of abundance or population increase and decrease. The explanation of the fluctuation which is in apparently stable patterns has long been of interest to animal conservationists and mathematicians. The dynamics of predator-prey interactions have been studied extensively in the recent years by researchers, see for example [2, 3, 16] and the references therein.

Within a population, it is often to see that a parasite spreads among individuals and the population becomes disease affected [9, 14], see for example rabbit fever and deer fly fever [20]. Mathematical epidemiology to study the dynamics of disease spread has also become an interesting topic of research work and received much attention from scientists after the pioneering work of Kermack-McKendrick [1, 3]. A number of mathematical models of disease spread have been introduced relevant to the type of diseases, for example SI, SIS, SIR, SEIR, SEIRS [6, 19] and references therein.

The presence of both predators and parasites has affected prey populations. A little attention has been addressed to the merge of these two important areas, ecology and epidemiology, see for some examples [2, 6, 12, 21]. Eco-epidemiology itself is still a relatively new subject in science. However, this particular subject is essential in particular to find some explanations relating to the persistence of biodiversity. This paper will explore the dynamics of such eco-epidemiological system. Although the number is still limited, some modified predator-prey models with disease have been introduced, see for example, the disease in prey [17], predators consume only infected preys [6], predators avoid infected prey [11], the disease in predators only [13], predators consume both healthy and infected preys but with Cosner type functional response [18]. In this study, we deal with the case where a SIS disease spreads only in a prey population and predators consume both healthy and infected preys with two different Holling type II functional responses. Collera [7] conducted a study of a system consists of two predators feeding on a single prey with Holling type II functional responses, but in the absence of disease.

2. Model Formulation

2.1. Assumptions

Individuals are classified into three groups; sound preys (x_S), infected preys (x_I), and predators (y). The growth of prey population follows a logistic function with intrinsic growth rate r , with carrying capacity K . Once a sound prey gets infected, due to the contact with an infected prey, it moves to the infected compartment at rate proportional to the number of infected prey, αx_I . It is assumed that an infected prey will get recovery due to, for example to its immune system, at constant rate θ . In addition to the rate of natural death for all preys μ , an infected prey also experiences a infection-related death at rate σ . In the presence of predators, the population of preys will decrease due to predation with Holling type II functions $\left(\frac{a_1 y}{1+b_1 x_S}\right)$ and $\left(\frac{a_2 y}{1+b_2 x_I}\right)$ for healthy and infected prey, respectively. Simultaneously, the population of predators will increase at the same rate as the predation response functions. In the absence of prey, predators will experience natural death at rate c .

2.2. Mathematical Equations

Based on the assumptions, the mathematical model is formulated as follows.

$$\begin{aligned}\frac{dx_S}{dt} &= r(x_S + x_I) \left(1 - \frac{x_S + x_I}{K}\right) - \alpha x_S x_I + \theta x_I - \mu x_S - \frac{a_1 x_S y}{1+b_1 x_S} \\ \frac{dx_I}{dt} &= \alpha x_S x_I - (\theta + \mu + \sigma) x_I - \frac{a_2 x_I y}{1+b_2 x_I} \\ \frac{dy}{dt} &= \beta_1 \left(\frac{a_1 x_S y}{1+b_1 x_S}\right) + \beta_2 \left(\frac{a_2 x_I y}{1+b_2 x_I}\right) - cy\end{aligned}\quad (3.1)$$

where $x_S \equiv x_S(t)$ stands for the number of healthy preys at time t , $x_I \equiv x_I(t)$ stands for the number of infected preys at time t , and $y \equiv y(t)$ denotes for the number of predators.

3. Analysis

3.1. Boundedness of the system

Proposition 1. Total population of sound and infected prey, $n = x_S + x_I$, is bounded.

Proof. By adding the first and the second equations in equation (3.1), we have

$$\begin{aligned}\frac{dn}{dt} &= rn(1 - n/K) - \mu n - \frac{a_1 x_S y}{1+b_1 x_S} - \sigma x_I - \frac{a_2 x_I y}{1+b_2 x_I} \\ &< rn(1 - n/K)\end{aligned}\quad (3.2)$$

Thus, if the total population prey n is below $K > 0$, $\frac{dn}{dt} < 0$ and the number of prey grows. Once $n > K$, the population becomes decreasing.

3.2. Equilibria

Equation (3.1) results in three equilibria; (i) trivial equilibrium; $E_1^*(0,0,0)$, (ii) the absence of both infected prey and predator; $E_2^*(1 - \mu K/r, 0, 0)$, (iii) the absence of infected prey only; $E_3^*\left(\frac{c}{\Lambda}, 0, \frac{\Lambda(r-\mu)-cr}{\Lambda^2}\right)$, with $\Lambda = \beta_1 a_1 - cb_1$.

3.3. Stability

The stability of E_1^* was analyzed by applying the linearization approach and the stability condition for E_2^* and E_3^* are derived by using the next generation matrix approach introduced in [10].

- *The stability of equilibrium E_1^**

The Jacobian matrix of the system (3.1), $J_1 = (a_{ij})$, $i, j = 1, 2, 3$, has elements as follows;

$$\begin{aligned} a_{11} &= r - \frac{2r}{K}(x_S + x_I) - \alpha x_I - \mu + A_1 y(B_1 y - 1); \\ a_{12} &= r - \frac{2r}{K}(x_S + x_I) - \alpha x_S + \theta; \quad a_{13} := -A_1 x_S; \quad a_{21} := \alpha x_I; \\ a_{22} &:= \alpha x_S - (\theta + \mu + \sigma) + A_2 y(B_2 x_I - 1); \quad a_{23} := -A_2 x_I; \\ a_{31} &:= \beta_1 A_1 y(1 - B_1 x_S); \quad a_{32} := \beta_2 B_2 y(1 - B_2 x_I); \\ a_{33} &:= \beta_1 A_1 x_S + \beta_2 B_2 x_I - c \end{aligned}$$

$$\text{with } A_1 = \frac{a_1}{1+b_1 x_S}; \quad A_2 = \frac{a_2}{1+b_2 x_S}; \quad B_1 = \frac{b_1}{1+b_1 x_S}; \quad B_2 = \frac{b_2}{1+b_2 x_S};$$

Evaluated at E_1^* , the matrix J_1 has three eigenvalues

$$\lambda_1 = -\theta - \sigma - \mu, \quad \lambda_2 = r - \mu, \quad \lambda_3 = -c.$$

Since all model parameters are nonnegative, then $\lambda_1, \lambda_3 \leq 0$. Thus, if $r < \mu$ ($\lambda_2 < 0$), the equilibrium E_1^* becomes stable. Otherwise, if $\geq \mu$ ($\lambda_2 \geq 0$), E_1^* is unstable. Let write $R_{01} = \frac{r}{\mu}$, and summarize the result in the following Proposition.

Proposition 2.

If $R_{01} < 1$, E_1^* is asymptotically locally stable. Otherwise, it is unstable if $R_{01} > 1$.

This implies that the presence of few species around the extinction point E_1^* will not be able to support the coexistence of all species unless the intrinsic growth rate of prey r is greater than its natural death rate μ . As $R_{01} > 1$, the coexistence of predator-prey population might occur.

- *The Stability of Equilibrium $E_2^*(K(1 - 1/R_{01}), 0, 0)$*

When $R_{01} > 1$ holds, the equilibrium E_2^* occurs and the following proposition holds.

Proposition 3.

Let $R_{02} = \frac{\alpha K}{\theta + \sigma + \mu} \left(1 - \frac{1}{R_{01}}\right)$ with $R_{01} > 1$. The equilibrium E_2^* is locally asymptotically stable if $R_{02} < 1$ and it becomes unstable if $R_{02} > 1$.

Proof. To give a proof, we follow the steps of the next generation matrix method. First, rewrite the nonlinear vector function $f(x_S, x_I, y)$ in (3.1) as $f = \mathcal{F} - \mathcal{V}$, with

$$\mathcal{F} = \begin{pmatrix} 0 \\ \alpha x_S x_I \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} \frac{r}{K}(x_S + x_I)^2 - (\theta + r)x_I + (\alpha x_I - r + \mu + A_1 y)x_S \\ (\theta + \mu + \sigma + A_2 y)x_I \\ -\beta_1 A_1 x_S y - \beta_2 A_2 x_I y + cy \end{pmatrix}$$

with A_1, A_2, B_1 , and B_2 defined as previous.

Then, calculate the Jacobian matrices of the corresponding functions $F \equiv D\mathcal{F}$ and $V \equiv D\mathcal{V}$. These matrices $F = (f_{ij})$ and $V = (v_{ij})$, for $i, j = 1, 2, 3$, have elements;

$$\begin{aligned} f_{12} &= \alpha x_I; \quad f_{22} = \alpha x_S; \quad v_{11} = -r + \frac{2r}{K}(x_S + x_I) + \alpha x_I + \mu + A_1 y(1 - B_1 x_S); \\ v_{12} &= -r + \frac{2r}{K}(x_S + x_I) + \alpha x_S - \theta; \quad v_{13} = A_1 x_S; \quad v_{22} = \theta + \mu + \sigma + \frac{a_2 y}{(1 + b_2 x_I)^2}; \\ v_{23} &= A_2 x_I; \quad v_{31} = -\frac{\beta_1 a_1 y}{(1 + b_1 x_S)^2}; \quad v_{32} = -\frac{\beta_2 a_2 y}{(1 + b_2 x_I)^2}; \\ v_{33} &= -\beta_1 A_1 x_S - \beta_2 A_2 x_I + c; \quad f_{11} = f_{13} = f_{21} = f_{23} = f_{33} = v_{21} = 0. \end{aligned}$$

Thus, the Jacobian matrices F and V for the equilibrium E_2^* have elements as follows

$$\begin{aligned} f_{22} &:= \alpha K \left(1 - \frac{\mu}{r}\right); \quad v_{11} := -r + \mu; \quad v_{12} := f_{22} + r - 2\mu - \theta; \quad v_{13} = \frac{\beta_1 a_1 K(r - \mu)}{r + b_1 K(r - \mu)}; \\ v_{22} &:= \theta + \sigma + \mu; \quad v_{33} := -v_{13} + c; \quad f_{11} = f_{13} = f_{21} = f_{23} = f_{33} = v_{21} = 0 \end{aligned}$$

The inverse of matrix V is obtained

$$V^{-1} = \frac{1}{v_{11}v_{22}v_{33}} \begin{pmatrix} v_{22}v_{33} & 0 & 0 \\ -v_{12}v_{33} & v_{11}v_{33} & 0 \\ -v_{13}v_{22} & 0 & v_{11}v_{22} \end{pmatrix},$$

with v_{ij} 's are defined as previous.

Thus, the product of FV^{-1} are as follows

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ -\frac{f_{22}v_{12}}{v_{11}v_{22}} & \frac{f_{22}}{v_{22}} & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

So,

$$R_{02} = \rho(FV^{-1}) = \frac{f_{22}}{v_{22}} = \frac{\alpha K(r-\mu)}{r(\theta+\sigma+\mu)} = \frac{\alpha K}{\theta+\sigma+\mu} \left(1 - \frac{1}{R_{01}}\right) \quad (3.5)$$

By applying the results in [10] (see Theorem 2), the proof is completed.

Note that the presence of few infected preys and predators will straightforward deploy healthy prey population if $R_{02} < 1$. However, if $R_{02} < 1$ the population of healthy prey will still increase for a while before all species go extinct.

• *The Stability of Equilibrium E_3^**

The positive equilibrium, $E_3^* \left(\frac{c}{\Lambda}, 0, \frac{r}{\Lambda} \left(1 - \frac{1}{R_{01}}\right) - \frac{c}{K\Lambda^2} \right)$ with $\Lambda = \beta_1 a_1 - cb_1$, will occur when $\frac{r}{\Lambda} \left(1 - \frac{1}{R_{01}}\right) - \frac{c}{K\Lambda^2} > 0$, i.e., $r > \frac{cR_{01}}{K\Lambda^2(R_{01}-1)}$ and $R_{01} > 1$.

The Jacobian matrices of E_3^* , $F = (f_{ij})$ and $V = (v_{ij})$, for $i, j = 1, 2, 3$, have elements as follows

$$\begin{aligned} f_{22} &:= \alpha K \left(1 - \frac{1}{R_{01}}\right); \quad v_{11} := -(r - \mu); \quad v_{12} := r + \alpha K \left(1 - \frac{1}{r}\right) - 2\mu - \theta; \\ v_{13} &:= \frac{\beta_1 a_1 K(r-\mu)}{r+b_1 K(r-\mu)}; \quad v_{22} := \theta + \sigma + \mu + \beta_2 a_2 \frac{K\Lambda(r-\mu)-cr}{K\Lambda^2}; \quad v_{33} := -v_{13} + c \end{aligned}$$

and other elements of f_{ij} 's and v_{ij} 's are zero.

The matrix of the product of F and V^{-1} has zero elements $(FV^{-1})_{ij} = 0$, except $(FV^{-1})_{22} = \frac{f_{22}}{v_{22}}$ with $f_{22} := \alpha K \left(1 - \frac{1}{R_{01}}\right)$ and $v_{22} := \theta + \sigma + \mu + \beta_2 a_2 \frac{r}{\Lambda} \left(1 - \frac{1}{R_{01}} - \frac{c}{K\Lambda}\right)$.

Thus,

$$R_{03} = \rho(FV^{-1}) = \frac{f_{22}}{v_{22}} = \frac{\alpha c K \Lambda}{K\Lambda[(\theta+\mu+\sigma)\Lambda - \beta_2 a_2 \left(1 - \frac{1}{R_{01}}\right)] - r\beta_2 a_2 c} \quad (3.6)$$

where $\Lambda = \beta_1 a_1 - cb_1$. Combining with the result, see Theorem 2 in [8], the following Proposition 3 is established.

Proposition 4.

Let R_{03} be as defined in (3.6). Then, the equilibrium $E_3^* \left(\frac{c}{\Lambda}, 0, \frac{r}{\Lambda} \left(1 - \frac{1}{R_{01}}\right) - \frac{c}{K\Lambda^2} \right)$ with $\Lambda = \beta_1 a_1 - cb_1$ becomes stable if $R_{03} < 1$ and, otherwise, if $R_{03} > 1$, it is unstable.

It implies that the presence of disease in prey population around E_3^* will be able to invade the prey population when the condition $R_{03} > 1$, see Equation (3.6), holds. On the other hands, the disease will die out.

3. Numerical Experiments and Discussion

In this section we present a range of numerical simulations that illustrate the accuracy and the dynamical features described previously in the analysis.

Case I (E_1^* and R_{01})

Suppose the initial populations of the species are around the trivial equilibrium $E_1^*(0,0,0)$, take an example $x_S = 5$; $x_I = 1$; dan $y = 5$. All model parameter values are fixed, except the intrinsic value of r , which is varied as $R_{01} = \frac{r}{\mu} > 1$, $R_{01} = 1$, and $R_{01} < 1$. As predicted in the analysis, the second and third eigen values of equation (3.3), which correspond to the dynamics of infected prey and predator population, respectively, are always negative. On the other hand, the first eigen value representing the dynamics of healthy prey population will depend on the value of R_{01} . This imply that the population of infected prey and predator will always goes to extinct whereas the population of healthy prey depend on threshold R_{01} ; it goes straight away to extinct for $R_{01} < 1$, be steady for $R_{01} = 1$, or increases from the initial population fro $R_{01} > 1$. These results can be seen in Figure 1(a).

Cases II (E_2^* and R_{02})

The initial populations of all three species, healthy and infected prey and predator, are chosen near the equilibrium E_2^* . All model parameters are fixed except which are varied to obtain three different values of R_{02} . The numerical simulations for three different R_{02} can be seen in Figure 1(b). Comparing to the result of numerical simulations of Case I where infected prey always goes to extinct, it is possible the number of infected prey to increase when $R_{02} > 1$ (above threshold).

Cases III (R_{03} and E_3^*)

Suppose the number of all species is set initially to the equilibrium E_3^* , i.e., only healthy preys and predators are present and there is no disease in the system. The values of all model parameters are fixed except α and let its value be varied such that we get three different values of R_{03} , i.e., $R_{03} < 1$; $R_{03} = 1$; and $R_{03} > 1$, see Figure 1 (c). Figure 1(c) shows that the presence of small number of infected preys near E_3^* will spread the disease in the prey population when $R_{03} > 1$ (above threshold). However, if $R_{03} < 1$ the disease vanishes. This dynamics is in agreement with the stability analysis in the previous section, see Proposition 4.

Figure 1.

(a). Dynamics of infected preys for three different values of R_{01} ; (a) $R_{01} < 1$; (b) $R_{01} = 1$; $R_{01} > 1$. (b). Dynamics of infected preys for three different values of R_{01} ; (a) $R_{01} < 1$; (b) $R_{01} = 1$; $R_{01} > 1$. (c). The dynamic trajectories of infected prey around the equilibrium E_2^* for three different values of infection rate α such that R_{02} , see (3.4), has three different values; i.e., $R_{02} > 1$; $R_{02} = 1$, and $R_{02} < 1$.

Cases IV (a stable limit cycle)

It has been shown that a stable limit cycle will be present in a predator-prey system and this fact has been observed in real life. However, it is still not many studies whether the presence of infectious disease in a predator-prey system will still generate a stable cycle.

To see this, we choose $\alpha = 0.8, r = 1.5$; $K = 100$; $\theta = 0.8$; $\beta_1 = 0.5$; $\beta_2 = 0.8$; $\sigma = 0.8$; $a_1 = a_2 = 1$; $b_1 = 0.3$; $b_2 = 0.5$; $\mu = 0.01$; $c = 0.8$. Two initial population of species are chosen as $X_1(70; 30; 5)$ and $X_2(40; 10; 5)$ for healthy prey, infected prey, and predator, respectively. From a numerical simulation as shown in Figure 2, a stable limit cycle can be present in a predator-prey system with a disease in prey.

Figure 2.

(a) Existence of a stable limit cycle for model (3.1) with the above parameter values and two initial values $X_1(70; 30; 5)$ and $X_2(40; 10; 5)$, (b): Time courses of the healthy prey density (light solid-curve), the infected prey density (dashed-curve), and predator density (solid-curve) when the model stabilizes into its oscillatory behavior.

4. Conclusion and further research

A mathematical model describing the spread of a disease in a predator-prey system has been discussed. An infectious disease is assumed to spread only among preys with SIS model and predators consume both healthy and infected preys with two different predation response. The model has three equilibrium points; a trivial equilibrium and two free-disease equilibriums. Threshold conditions for all three equilibriums are derived by applying linearization method and the next generation method. The threshold conditions serve as the criteria for the local stability of the equilibriums. As the parameters are below the threshold; $R_{01}, R_{02}, R_{03} < 1$, the equilibriums are locally asymptotically stable, and if the conditions are $R_{01}, R_{02}, R_{03} > 1$ (above threshold), the equilibrium points become locally asymptotically unstable. In this study, we numerically show that a stable limit cycle can exist in the presence of disease in a predator-prey system. In future, it is interesting to see the dynamics of the infectious disease spread when it can spread also in a predator population. Future research will be considered the effect of diffusion properties where, the spatial spread of the predator and prey are taken into account. On itself, such model yields travelling wave solution [15], and also an interaction of two groups of delta like function which act as an initial condition [4, 5].

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