Disease-selective predation may lead to prey extinction

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SUMMARY

In real world bio-communities, predational choice plays a key role to the persistence of the prey population. Predator's 'sense' of choice for predation towards the infected and noninfected prey is an important factor for those bio-communities. There are examples where the predator can distinguish the infected prey and avoids those at the time of predation. Based on the examples, we propose two mathematical models and observe the dynamics of the systems around biologically feasible equilibria. For disease-selective predation model there is a high risk of prey extinction. On the other hand, for non-disease selective predation both populations co-exist. Local stability analysis and global stability analysis of the positive interior equilibrium are performed. Moreover, conditions for the permanence of the system are obtained. Finally, we conclude that strictly disease-selective predation may not be acceptable for the persistence of the prey population. Copyright © 2005 John Wiley & Sons, Ltd.

KEY WORDS: disease; selective-predation; non-selective predation; local stability; global stability; permanence; extinction

1. INTRODUCTION

In recent years, disease in the prey–predator system is one of the most important fields of interest. Many researchers have attempted several approaches to study this interesting field. Holmes and Bethel [1] and Dobson [2] discussed situations where the behaviour of infected individuals of a prey population as a host is modified by the action of a parasite. Freedman [3] has studied a predator–prey system in which some members of the prey population and all the predators are subjected to infection by parasites, and obtained conditions for persistence of all populations and global stability of the positive equilibrium. Anderson and May [4] showed that invasion of a resident predator–prey or host–parasite system by a new strain of parasites could cause destabilization and exhibit limit cycles. Hadeler and Freedman [5] obtained a similar phenomenon. Mukherjee [6] analysed a generalized prey–predator system with parasite

infection and obtained conditions for persistence and impermanence. Chattopadhyay and Arino [7] have studied predator–prey system when predators mainly eat infected prey, and derived the persistence and extinction conditions and also determined the condition when the system enters a Hopf bifurcation. Xiao and Chen [8] modified the model of Chattopadhyay and Arino [7] by introducing the delay term and studied the dynamics of the modified system. However, the choice or rejection of infected species by the predator cannot be ignored. The above studies have not taken into account this factor while formulating or introducing the models.

In the real world there are situations where the predator distinguishes between the infected and noninfected (susceptible) prey and rejects the infected part. Bullfrog (*Rana catesbenia*) tadpoles avoid conspecifics carrying infectious yeast, *Candida humicola*, by detecting chemical cues emanating from infected individuals at a distance [9]. This is an alternative way of using phylogenetic similarity to assess disease risk, which may arise from infected prey. Although this is not a direct example of the predator–prey system, it certainly sites an instance for disease selective consumption. Now if the predator can recognize and avoid infected prey, then this selection of the predator may accrue the enhanced nutritional benefits of eating phylogenetically close prey while limiting risk of disease [10].

Also, if a predator's immune system 'learns' to recognize and destroy parasites in its food or its nervous system has 'sense' to identify the infected prey, which may cause disease of the predator, then it is quite natural that the predator will always try to avoid the infected prey. So in this case, for the persistence of the predator, it should have more than one source of food. A population like human beings (*Homo sapiens*) has such kind of 'sense' and thus it always exhibits disease-selective predation to prey populations like fish.

Now naturally a question arises, what will be the fate of the prey population if it becomes highly infected and its predator exhibits disease-selective predation? Will this prey population survive in this case? If not, then what restriction should be taken in order to overcome this situation?

In this paper we have considered such a predator–prey system where the predator has specific choice regarding predation and it can recognize the infected prey and avoids those during predation. As stated earlier this is realistic when the predator has more than one source of food, and thus we consider that the predator itself grows logistically.

Firstly, we consider the situation mathematically when the predator is not selecting the diseased prey and taking only the sound (susceptible) part. Our analysis predicts that there is a high risk for the prey to become extinct. Next, in order to overcome this risk, we make some modifications of the predator choice and consider another model with the necessary modifications and find whether we can have any means to save the sound prey population.

2. MODEL FOR DISEASE-SELECTIVE PREDATION

In the formulation of the mathematical model for disease-selective predation, the following assumptions are made:

(1) The prey population is divided into two classes, namely, susceptible class (S) and infected class (I). The susceptible class follows logistic growth with intrinsic growth rate 'r', carrying capacity 'K_1', which is shared by the entire prey population (i.e. both susceptible and infected class).

(2) A part of the susceptible class becomes infected at a rate 'α' and this transformation follows the law of mass action.
(3) The infected prey population suffers a constant death rate 'β'.
(4) The predator (Y) is not solely dependent on this prey population for its food, i.e. the predator has some other sources of food. Hence, the predator is supposed to follow logistic growth with intrinsic growth rate 'R' and carrying capacity 'K_2'.
(5) Finally, it is assumed that the predator has some choice regarding the predation of this particular prey. According to assumption (4) made earlier, the predator has some other alternative source for food, so it is reasonable to think that the predator may have choice among the different groups of this particular prey. Without considering the case of size-selective predation, let us consider that the predator is not at all consuming the infected part of the prey population, i.e. it is exhibiting disease-selective predation. So the predator is only consuming the susceptible group of prey population. Under this assumption it is reasonable to think that due to this choice the predator will consume the disease free population in some greater rate. Also, due to the presence of other sources, it can be assumed that the predator will not face much difficulty to obtain the appropriate quantity of food needed for its growth. So this particular selective predation will not lead the predator population to extinction.

Following the above mentioned assumptions, the system with disease-selective predation may be written as

$$\left.\begin{aligned} \frac{dS}{dt} &= Sr\left(1-\frac{S+I}{K_1}\right)-\alpha IS-\delta YS \\ \frac{dI}{dt} &= I\alpha S-\beta I \\ \frac{dY}{dt} &= YR\left(1-\frac{Y}{K_2}\right)+\delta_1 YS \end{aligned}\right\} \tag{1}$$

The basic model (1) takes the following form after non-dimensionalization

$$\left.\begin{aligned} \frac{ds}{d\tau} &= s(1-s-i)-\theta_1 si-\theta_2 ys \\ \frac{di}{d\tau} &= \theta_1 is-\theta_3 i \\ \frac{dy}{d\tau} &= \theta_4 y(1-y)+\theta_6 ys \end{aligned}\right\} \tag{2}$$

with the following rescaling variables:

$$s=\frac{S}{K_1},\quad i=\frac{I}{K_1},\quad y=\frac{Y}{K_2}$$

where, $\tau=rt$, $\theta_1=\alpha K_1/r$, $\theta_2=\delta K_2/r$, $\theta_3=\beta/r$, $\theta_4=R/r$, $\theta_6=\delta_1 K_1/r$.

Now a few simple results will be established for which both the susceptible prey population and infected prey population will eventually go extinct due to disease-selective predation.

2.1. Criterion for the extinction of susceptible prey population

Theorem 2.1
Let the inequality $Y_0 > 1/\delta\{r-(r/K_1+\alpha)I_0\}$ hold, where Y_0 is the initial predator population and I_0 is the initial infected prey. Then $\lim_{t\longrightarrow\infty} S_t = 0$.

Proof
From system (1), one has

$$\frac{dS}{dt} = Sr\left(1-\frac{S+I}{K_1}\right) - \alpha IS - \delta YS \leqslant S\left(r-\left(\frac{r}{K_1}+\alpha\right)I_0 - \delta Y_0\right) \leqslant 0$$
$$\text{if } Y_0 > \frac{1}{\delta}\left(r-\left(\frac{r}{K_1}+\alpha\right)I_0\right)$$

Hence the theorem follows. □

It is clear that if there is no infected prey present initially (i.e. $I_0 = 0$) then the susceptible prey population will be eliminated from the dynamical system if the initial predator population is just larger than the ratio of intrinsic growth rate of the prey to the predation rate of susceptibility.

2.2. Criterion for the extinction of infected prey population

Theorem 2.2
Let the inequality $S_0 < \beta/\alpha$ hold, where S_0 is the initial susceptible prey population. Then $\lim_{t\longrightarrow\infty} I_t = 0$.

Proof
From system (1), one has $dI/dt = (I\alpha S - \beta I) \leqslant I(\alpha S_0 - \beta) < 0$, if $S_0 < \beta/\alpha$. Hence the theorem follows. □

The above theorem demonstrates that in case of disease-selective predation the infected prey population will go extinct if the initial susceptible prey population is less than the ratio of death rate of infected prey to the rate of infection.

3. STABILITY ANALYSIS OF THE EQUILIBRIA

System (4) possesses five equilibria, namely,

$$E_0(0,0,0),\ E_1(1,0,0),\ E_2\left(\frac{\theta_3}{\theta_1}, \frac{(\theta_1-\theta_3)}{\theta_1(1+\theta_1)}, 0\right),\ E_3\left(\frac{\theta_4-\theta_2\theta_4}{\theta_4+\theta_2\theta_6}, 0, \frac{\theta_4+\theta_6}{\theta_4+\theta_2\theta_6}\right)$$

and the interior equilibrium of the above system is $E^*(s^*, i^*, p^*)$, where

$$s^* = \frac{\theta_3}{\theta_1},\ i^* = \frac{1 - \dfrac{\theta_3}{\theta_1} - \theta_2 y^*}{1 + \theta_1},\ y^* = 1 + \frac{\theta_3 \theta_6}{\theta_1 \theta_4}$$

The behaviour of the interior equilibrium of the above system is of interest to us, as in this case both prey and predator population will be present and the predator will exhibit selective predation. The above equilibrium point exists if $\theta_3/\theta_1 + \theta_2 y^* < 1$.

The community matrix of system (3) at the interior equilibrium point is

$$J^* = \begin{bmatrix} -s^* & -(1+\theta_1)s^* & \theta_2 s^* \\ \theta_1 i^* & 0 & 0 \\ \theta_6 y^* & 0 & -\theta_4 y^* \end{bmatrix}$$

Hence the characteristic equation is given by

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0, \text{ where}$$

$$A_1 = s^* + \theta_4 y^* > 0$$

$$A_2 = \theta_4 s^* y^* - \theta_1(1+\theta_1)s^* i^* - \theta_2\theta_5 s^* y^* > 0 \text{ if } \frac{y^*}{i^*} > \frac{\theta_1(1+\theta_1)}{\theta_4} \text{ and}$$

$$A_3 = -\theta_1\theta_4(1+\theta_1)s^* i^* y^* < 0$$

since $A_3 < 0$, so by Routh–Hurwitz criterion the interior equilibrium is always unstable. So in the long run the prey population (S) will decrease and may lead to extinction.

Thus, it is seen that due to disease-selective predation, there is a high risk of prey extinction. Clearly this risk will be increased if we consider any 'close' system.

4. MODEL OF NON-DISEASE-SELECTIVE PREDATION

Now in order to overcome the situation, the following new assumptions are made:

(6) The predator consumes not only the susceptible part but also the infected part at a rate γ and a part of the consumed infected-prey exhibits a positive effect to the growth of the predator, this rate is assumed to be γ_1. Clearly $\gamma > \gamma_1$. Further, it is assumed that the rate of predation for susceptibles (δ) and infected class (γ) are not equal although in both the cases the functional responses are taken to be density dependent.

(7) Due to the consumption of infected prey, the predator is supposed to acquire some infection or disease, which causes the death of the predator at a rate λ_2.

So, under the above mentioned assumptions (1)–(4), (6) and (7) the model (1) takes the form

$$\left.\begin{aligned}\frac{dS}{dt} &= Sr\left(1-\frac{S+I}{K_1}\right)-\alpha IS-\delta YS\\ \frac{dI}{dt} &= I\alpha S-\beta I-\gamma YI\\ \frac{dY}{dt} &= YR\left(1-\frac{Y}{K_2}\right)-\lambda_2 Y+\delta_1 YS+\gamma_1 YI\end{aligned}\right\} \tag{3}$$

For the sake of simplicity of analysis, system (3) is now non-dimensionalized to the following system:

$$\left.\begin{aligned}\frac{ds}{d\tau} &= s(1-s-i)-\theta_1 si-\theta_2 ys\\ \frac{di}{d\tau} &= \theta_1 is-\theta_3 i-\eta_1 yi\\ \frac{dy}{d\tau} &= \theta_4 y(1-y)-\theta_5 y+\theta_6 ys+\eta_2 yi\end{aligned}\right\} \tag{4}$$

where $s=S/K_1$, $i=I/K_1$, $y=Y/K_2$, $\tau=rt$, $\theta_1=\alpha K_1/r$, $\theta_2=\delta K_2/r$, $\theta_3=\beta/r$, $\theta_4=R/r$, $\theta_5=\lambda_2/r$, $\theta_6=\delta_1 K_1/r$, $\eta_1=\gamma K_2/r$, $\eta_2=\gamma_1 K_2/r$.

5. BOUNDEDNESS

Lemma 5.1
All the solutions of system (4) [and hence of (3)], which initiate in R^3_+, are uniformly bounded if $\gamma_0 \leqslant \theta_3$, where γ_0 is a positive constant, which should be appropriately chosen.

Proof
Let the following function be considered, $w=s+i+y$. The time derivative of the above function along the solution of (4) is

$$\frac{dw}{dt}=[s(1-s-i)-\theta_1 is-\theta_2 ys]+[\theta_1 is-\theta_3 i-\eta_1 yi]+[\theta_4 y(1-y)-\theta_5 y+\theta_6 ys-\eta_2 yi]$$

Assuming $\theta_2>\theta_6$ and since, $\eta_1>\eta_2$, it is clear that

$$\frac{dw}{dt}\leqslant s(1-s)+\theta_4 y(1-y)-\theta_3 i$$

Hence,

$$\frac{dw}{dt}+\gamma_0 w\leqslant \frac{(1+\theta_4)(1+\gamma_0)^2}{4}+(\gamma_0-\theta_3)i-\left(s-\frac{1+\gamma_0}{2}\right)^2-\theta_4\left(y-\frac{\theta_4+\gamma_0}{2\theta_4}\right)^2$$

If we take $\gamma_0 \leqslant \theta_3$, then the above expression reduces to, $dw/dt + \gamma_0 w \leqslant (1+\theta_4)(1+\gamma_0)^2/4$. Clearly the right hand side of the above expression is bounded. So we can find a $\mu_0 > 0$ such that, $dw/dt + \gamma_0 w < \mu_0$.

Now applying the theory of differential inequality see Reference [11], we obtain, $0 < w(s,i,y) < \mu_0/\gamma_0(1-\exp(-\gamma_0 t)) + w(s(0),i(0),y(0))\exp(-\gamma_0 t)$ and for $t \longrightarrow \infty$, we have $0 < w < \mu_0/\gamma_0$. Hence, all the solutions of (4) that initiate in R^3_+ are confined in the region $B = \{(s,i,y) \in R^3_+ : w = \mu_0/\gamma_0 + \varepsilon \text{ for any, } \varepsilon > 0\}$ □

6. EQUILIBRIA

System (4) possesses five equilibria, namely, $E_0(0,0,0)$, $E_1(1,0,0)$, $E_2(s_2,i_2,0)$, $E_3(s_3,0,y_3)$ and $E_4(s^*,i^*,y*)$, where,

$$s_2 = \frac{\theta_3}{\theta_1}$$

$$i_2 = \frac{(\theta_1 - \theta_3)}{\theta_1(1+\theta_1)}$$

$$s_3 = \frac{\theta_4 - \theta_2\theta_4 + \theta_2\theta_5}{\theta_4 + \theta_2\theta_6}, \quad y_3 = \frac{\theta_4 - \theta_5 + \theta_6}{\theta_4 + \theta_2\theta_6}$$

$$s^* = \frac{\theta_3\theta_2\eta_2 + \theta_4(1+\theta_1) + \eta_1(\eta_2 - (1+\theta_1)(\theta_5 - \theta_4))}{\Delta}$$

$$i^* = \frac{-\theta_3\theta_4 + \eta_1(\theta_4 - \theta_5) - \eta_1\theta_6 + \theta_1\theta_4 + \theta_2(\theta_1\theta_5 - \theta_1\theta_4 - \theta_3\theta_6)}{\Delta}$$

$$p^* = \frac{\theta_1(-(1+\theta_1)(\theta_5 - \theta_4) + \eta_2) + \theta_3((1+\theta_1)\theta_6 - \eta_2)}{\Delta}$$

with $\Delta = \theta_1(\theta_2\eta_2 + \theta_4(1+\theta_1)) + \eta_1(\eta_2 - \theta_6(1+\theta_1))$.

Lemma 6.1
The equilibrium (i): E_2 exists for $\theta_1 > \theta_3$, (ii): E_3 exists for $\theta_4 + \theta_6 > \theta_5$ and $\theta_4 + \theta_2\theta_5 > \theta_2\theta_4$, (iii): E_4 exists for $(1+\theta_1)(\theta_5 - \theta_4) < \eta_2 < \theta_6$ and

$$\eta_1 > \frac{\theta_2\theta_3\theta_6 + \theta_1\theta_2\theta_4 + \theta_3\theta_4 - \theta_1\theta_2\theta_5 - \theta_1\theta_4}{\theta_5 - \theta_5 - \theta_6}$$

7. CONDITIONS FOR PERSISTENCE OF THE SPECIES

Theorem (local stability) 7.1
The equilibrium point (i) E_0 is locally unstable, (ii) E_1 is locally unstable if $\theta_1 > \theta_3$ or $\theta_4 + \theta_6 > \theta_5$, (iii) E_2 is locally unstable if $\theta_5 > \theta_4 + \theta_6\theta_3/\theta_1 + \eta_2 i_1$ or $\theta_4 + \theta_6\theta_3/\theta_1 + \eta_2 i_1 > \theta_3/\theta_1 + \theta_5$,

(iv) E_3 is locally unstable if $\eta_1 < (\theta_1 s_3 - \theta_3)/y_3$ or $s_3 + \theta_4 y_3 < 1$, and (v) a sufficient condition for the interior equilibrium to be locally asymptotically stable is,

$$\eta_1 > \max(\theta_1\theta_2, \theta_1\theta_4)$$

$$\eta_2 > \frac{(1+\theta_1)(\theta_2+\theta_4)}{2\theta_2} \quad \text{and} \quad \theta_4 > \theta_2\theta_6 \text{ i.e.}$$

$$R > \frac{\delta_1\delta_2 K_1 K_2}{r}$$

Proof

The community matrix of system (4) is

$$J = \begin{bmatrix} 1 - 2s - (1+\theta_1)i - \theta_2 y & -(1+\theta_1)s & -\theta_2 s \\ \theta_1 i & \theta_1 s - \theta_3 - \eta_1 y & -\eta_1 i \\ \theta_6 y & \eta_2 y & \theta_4 - \theta_5 - 2\theta_4 y + \theta_6 s + \eta_2 i \end{bmatrix}$$

Proof of (i) is obvious.

Again at $E_1(1,0,0)$, the eigenvalues of the variational matrix are -1, $\theta_1 - \theta_3$, $\theta_4 - \theta_5 + \theta_6$, so at least one eigenvalue is positive if either $\theta_1 > \theta_3$ or $\theta_4 + \theta_6 > \theta_5$; hence (ii) follows. The proof of (iii), (iv) and (v) are similar and so proof of (iv) is shown here in detail.

The variational matrix at $E_4(s^*, i^*, p^*)$ is

$$J^* = \begin{bmatrix} -s^* & -(1+\theta_1)s^* & \theta_2 s^* \\ \theta_1 i^* & 0 & -\eta_1 i^* \\ \theta_6 y^* & \eta_2 y^* & -\theta_4 y^* \end{bmatrix}$$

Hence, the characteristic equation at $E_4(s^*, i^*, p^*)$ is

$$\lambda_3 + A\lambda_2 + B\lambda + C = 0$$

where $A = s^* + \theta_4 y^*$,

$$B = \theta_4 s^* y^* - \theta_1(1+\theta_1)s^* i^* + \eta_1\eta_2 y^* i^* - \theta_2\theta_6 s^* y^*$$

$$C = s^* y^* i^* [\eta_2(\eta_1 - \theta_1\theta_4) + (1+\theta_1)(\eta_1 - \theta_1\theta_4)]$$

Now $A > 0$. Again $C > 0$ if $\eta_1 > \max(\theta_1\theta_2, \theta_1\theta_4)$.

It can be shown that a sufficient condition for $AB - C > 0$ is $\eta_2 > (1+\theta_1)(\theta_2+\theta_4)/2\theta_2$ and $\theta_4 > \theta_2\theta_6$ i.e. $R > \delta_1\delta_2 K_1 K_2/r$.

Hence by Routh–Hurwitz criterion, the interior equilibrium $E_4(s^*, i^*, p^*)$ is locally asymptotically stable under the above parametric conditions. Hence (iv) follows.

This completes the proof. □

Theorem (global stability) 7.2

The interior equilibrium E_4 of system (4) is globally asymptotically stable if $4\eta_2\theta_1\theta_4 / \eta_1(1+\theta_1) > (\theta_6 - \eta_2\theta_1\theta_2/\eta_1(1+\theta_2))^2$.

Proof
Let the following Lyapunov function be considered:

$$V=\frac{\eta_2\theta_1}{\eta_1(1+\theta_1)}\left(s-s^*-s^*\ln\left(\frac{s}{s^*}\right)\right)+\frac{\eta_2}{\eta_1}\left(i-i^*-i^*\ln\left(\frac{i}{i^*}\right)\right)+\left(p-p^*-p^*\ln\left(\frac{p}{p^*}\right)\right)$$

Taking the time derivative along the solution of (4) and after some simplification one gets,

$$\frac{\mathrm{d}V}{\mathrm{d}t}=-\frac{\eta_2\theta_1}{\eta_1(1+\theta_1)}(s-s^*)^2-\theta_4(y-y^*)^2-\left(\theta_6-\frac{\eta_2\theta_1\theta_2}{\eta_1(1+\theta_1)}\right)(s-s^*)(y-y^*)$$

The right hand side of the above expression can be written as $-Z^{\mathrm{T}}QZ$, where $Z=(s-s^*,y-y^*)$ and the symmetric matrix Q is given by

$$Q=\begin{bmatrix}\dfrac{\eta_2\theta_1}{\eta_1(1+\theta_1)} & \dfrac{1}{2}\left(\theta_6-\dfrac{\eta_2\theta_1\theta_2}{\eta_1(1+\theta_1)}\right)\\ \dfrac{1}{2}\left(\theta_6-\dfrac{\eta_2\theta_1\theta_2}{\eta_1(1+\theta_1)}\right) & \theta_4\end{bmatrix}$$

If the symmetric matrix Q is positive definite, we can conclude that $\mathrm{d}V/\mathrm{d}t<0$. Q is positive definite if

$$\frac{4\eta_2\theta_1\theta_4}{\eta_1(1+\theta_1)}>\left(\theta_6-\frac{\eta_2\theta_1\theta_2}{\eta_1(1+\theta_2)}\right)^2$$

We can therefore apply Lasalle's theorem [8] and decide that any trajectory goes towards the maximal invariant set X (say) included in the set:

$$A=\{(s,i,p)\in B;\, s=s^*,\, y=y^*\}$$

On this straight line A, in the positive space, the only invariant set is the equilibrium E^*. Thus the trajectory converges towards E^* and hence the system considered above around the interior equilibrium point is globally asymptotically stable. □

8. PERMANENCE OF THE SYSTEM

To examine the permanence of system (4), we shall use the method of 'average Lyapunov function' [12,13].

Theorem 8.1
System (4) is permanent if

$$\text{(i)}\ \theta_1>\theta_3$$

$$\text{(ii)}\ \theta_5<\min\left\{\theta_4+\theta_6,\theta_4+\frac{\theta_3\theta_6}{\theta_1}+\frac{\eta_2(\theta_1-\theta_3)}{\theta_1(1+\theta_1)}\right\}$$

$$\text{(iii)}\ \theta_3(\theta_4+\theta_2\theta_6)+\eta_1(\theta_4+\theta_6-\theta_5)<\theta_1(\theta_4-\theta_2\theta_4+\theta_2\theta_5)$$

Proof
We consider the average Lyapunov function of the form

$$V(s,i,y)=s^{\alpha_1}i^{\alpha_2}y^{\alpha_3}$$

where each α_i $(i=1,2,3)$ is assumed positive. In the interior of $\Re^3_{0,+}$, we have

$$\frac{\dot{V}}{V}=\psi(s,i,y)$$
$$=\alpha_1[(1-s-i)-\theta_1 i-\theta_2 y]+\alpha_2[\theta_1 s-\theta_3-\eta_1 y]+\alpha_3[\theta_4(1-y)-\theta_5+\theta_6 s+\eta_2 i] \quad (5)$$

We can show that $\psi(s,i,y)>0$ for all equilibria $s,i,y\in\Re^3_{0,+}$, for a suitable choice of $\alpha_i>0$ $(i=1,2,3)$ to prove permanent coexistence. The following conditions should be satisfied for equilibrium (s), planar equilibria, (si) and (sy):

$$s:\quad \alpha_2(\theta_1-\theta_3)+\alpha_3(\theta_4+\theta_6-\theta_5)>0 \quad (6)$$

$$si:\quad \alpha_3\left(\theta_4-\theta_5+\frac{\theta_3\theta_6}{\theta_1}+\frac{\eta_2(\theta_1-\theta_3)}{\theta_1(1+\theta_1)}\right)>0 \quad (7)$$

$$sy:\quad \alpha_2\left[\frac{\theta_1(\theta_4-\theta_2\theta_4+\theta_2\theta_5)}{\theta_4+\theta_2\theta_6}-\theta_3+\frac{\eta_1(\theta_4+\theta_6-\theta_5)}{\theta_4+\theta_2\theta_6}\right]>0 \quad (8)$$

After some algebraic calculations, it can be easily shown that conditions (6)–(8) are satisfied if the hypothesis of the above theorem holds. □

Remark
It is to be noted that the first condition will be satisfied only when conditions for the existence of '*si*' are satisfied. Further, it is clear from the above observation that the persistence of the three populations depends on the predation rate of both infected and susceptible prey population.

Remark
Clearly the condition for both local and global stability is dependent on the rate of predation (i.e. on γ_1) and the rate of assimilation (i.e. on γ_2) of the infected population.

9. CONCLUSION

In this paper, we have discussed selective predation of a biological community consisting of a predator population and a prey population, which is partly infected. Firstly, we have formulated and analysed the mathematical model considering the predator's 'sense' of disease-selective predation, which we have assumed to be valid on the hypothesis of the existence of some alternative source of food. We have found that in this case all the species cannot coexist. However, since the infected population is not assumed to recover there remains high risk of the prey not able to persist in the long run. Next, we have proposed some hypothesis

regarding the predation of the predator apart from its 'sense' of selection. We have found out the restriction on the rate of predation of the infected prey and its rate of assimilation under which all the population will persist. Also, the system has been proved to be locally as well as globally stable (Theorems 7.1 and 7.2) under certain parametric conditions depending on the predation and assimilation rate of the infected prey. Finally we have also shown the permanence of this modified model. From our analysis we like to conclude that it is not possible for any biological system with predators and infected prey to persist if the predator always selects 'only' the sound prey for predation. So for the real biological communities with this type of selective predation, such as human population and fish population in a certain pond or lake, disease-selective predation may lead to a situation of extinction of the prey population which may lead to ecological imbalance. However, the idea of disease-selective predation may be effectively applied to multi-prey systems where infection in some prey is harmful for their biodiversity. In those situations, strong disease-selective predation may help the predator to persist safely as well as remove the infection from the system rapidly.

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