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ADVANTAGE OF PREY DUE TO HERD BEHAVIOR IN DISEASES-SELECTIVE SYSTEM

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ABSTRACT

In this paper the mathematical model of disease-selective predation as proposed by Roy and Chattopadhyay [10] is considered to identify the true risk of selective predation where the predator can recognize the infected prey and avoids those during predation. Furthermore, the model is modified and find out the conditions of getting advantage of prey due to herd in diseases-selective model both numerically and analytically to review the risk factors. Finally, we observe that the risk of prey extinction in disease selective can be arrest in certain level due to herd behavior of prey.

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INTRODUCTION

In general, diseases either in prey or in predator plays an important role in the dynamics of a prey-predator system. In recent years researchers have taken several approaches to study this interesting phenomenon. Previously a good number of studies have shown that predators take disproportionate number of prey that are infected by parasites (see, Vaughn and Coble (Vaughn, 1975). Temple (1987), Holmes and Bethel (1972), discussed many examples in which the parasite changes the external features or behavior of the prey, so that infected prey are more vulnerable to predator. Infected prey sometimes choose such locations that are more accessible to predators; for example, infected fish or aquatic snails may live close to the water surface or snails may live on top of vegetation rather than under protective plant cover. Similarly, infected prey sometimes became weaker or less active, so that they are caught more easily by predator (Dobson, 1988). In a prey-predator model with disease in prey Anderson and May (Anderson, 1978), found that the pathogen tends to destabilize the prey-predator interactions and exhibits limit cycles when predation on infected prey is much and no reproduction in infected prey. Hadeler and Freedman discuss the situation where predator could only survive on the prey if some of the prey were more vulnerable due to disease. Freedman (Freedman, 1990), studied a predator-prey system in which some members of the prey population all the predators are subjected to infection by parasites, and obtained conditions for persistence of all populations and global stability of the positive equilibrium. Mukherjee (Mukherjee, 2014), analysed a generalized prey-predator system with parasite infection and obtained conditions for persistence and impermanence. Chattopadhyay and Arino (Yang, 1996), studied predator-prey system when predator eat infected prey and derived the persistence and extinction conditions and also determined the condition for Hopf bifurcation. Xiao and Chen (Xiao, 2001), modified the model of Chattopadhyay and Arino by introducing the delay term and studied the dynamics of the modified system. 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 predator-prey system, but it certainly sites an instance for

disease selective consumption. Now if the predator can recognize and avoid infected prey then this selection of predator may accrue the enhanced nutritional benifits of eating phylogenetically close prey while limiting risk of disease (Pfennig, 2000). Now, naturally a question arise, 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? Roy and Chattopadhyay (Roy, 2005), introduced a mathematical model of disease-selective predation incorporating this concept. They considered a predator-prey system where the predator has specific choice regarding predation and it can recognize the infected prey and avoid those during predation. Recently, S.Alam (Freedman, 1983), introduced a mathematical model of risk of disease-selective predation in an infected prey-predator system and analyzed the effect of discrete time delay in the term involving the gestation of susceptible prey by the predator. In this paper, we have modified the disease-selective model as suggested by Roy and Chattopadhyay (2005), and Alam (2009), just incorporating square root term in the response function due to herd behavior of prey and analyze the model system in details. Here analytically we find out the conditions of disease free equilibrium point and stability conditions interior equilibrium point which is supported by numerical findings. Finally, we observe that the risk of prey extinction in disease selective can be arrest in certain level due to herd behavior of prey.

Model for Diseases-Selective Predation under Herd of Prey

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

- 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 and carrying capacity K_1 which is shared by the entire prey population (i.e. both susceptible and infected class).
- Susceptible prey exhibits herd behavior to protect themselves from the predation.
- Susceptible class becomes infected at a rate α and this transformation follows the law of mass action.
- The infected prey population suffers a constant death rate β.
- 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.
- Finally, it is assumed that the predators have some choice for their food and they are only consuming the susceptible group of prey population.

Based on the above mentioned assumption we consider the model as:

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I}{K_1}\right) - \delta\sqrt{S}Y - \alpha SI,$$

$$\frac{dI}{dt} = \alpha SI - \beta I,$$

$$\frac{dY}{dt} = RY\left(1 - \frac{Y}{K_2}\right) + \delta_1 Y\sqrt{S}. \qquad \ldots\ldots\ldots\ldots(2.1)$$

The basic model (2.1) takes the following dimensionless form:

$$\frac{ds}{d\tau} = s(1 - s - i) - \theta_1 si - \theta_2 y\sqrt{s},$$
$$\frac{di}{d\tau} = \theta_1 si - \theta_3 i,$$
$$\frac{dy}{d\tau} = \theta_4 y(1 - y) + \theta_5 y\sqrt{s}. \qquad \ldots\ldots\ldots\ldots(2.2)$$

With the re-scalling variables

$s = \frac{S}{K_1}$, $i = \frac{I}{K_1}$, $y = \frac{Y}{K_2}$ where $\tau = rt$, $\theta_1 = \frac{\alpha K_1}{r}$, $\theta_2 = \frac{\delta K_2}{r\sqrt{K_1}}$, $\theta_3 = \frac{\beta}{r}$, $\theta_4 = \frac{R}{r}$ and $\theta_5 = \frac{\sqrt{K_1}\delta_1}{r}$.

Positive invariance of the system

Let us put equation (2.1) in a vector form by setting $x = \mathrm{col}(s, i, y) \in R^3$

$$F(X) = \begin{bmatrix} F_1(X) \\ F_2(X) \\ F_3(X) \end{bmatrix} = \begin{bmatrix} s(1 - s - i) - \theta_1 si - \theta_2 y\sqrt{s} \\ \theta_1 si - \theta_3 i \\ \theta_4 y(1 - y) + \theta_5 y\sqrt{s} \end{bmatrix}.$$

Where $F: C_+ \to R^3$ and $F \in C^\infty$. The equation (2.2) becomes $X = F(X)$, with $X(\theta) = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C_+$ and $\phi_i(\theta) > 0$ $(i = 1, 2, 3)$. It is easy to cheek in the above quation that whenever choosing $X(\theta) \in C_+$ such that $X_i = 0$, then $F_i(x)$ $x_i(t) = 0$, $x(t) \in C_+ \geq 0, (i = 1, 2, 3)$. Due to lemma (Yang et al. [18]) any solution of the above equation with $X(\theta) \in C_+$, say $X(t) = X(t, X(\theta))$, such that $X(\theta) \in R^3$ for all $t > 0$.

1.2 Boundedness of solution

Lemma 2.2.1

If the initial condition of equation (2.2) satisfies

$\phi_1(\theta) + \phi_2(\theta) \geq 1, \theta \in [-\tau, 0]$, then either (i) $s(t) + i(t) \geq 1$ for all $t \geq 0$ and therefore $t \to \infty$, $(s(t), y(t), i(t) \to E_1(1, 0, 0))$

Or (ii) there exists a $t_0 > 0$ such that $s(t) + i(t) < 1$ for all $t > 0$. Also if $\phi_1(\theta) + \phi_2(\theta) < 1$, $\theta \in [-\tau, 0]$, then $s(t) + i(t) < 1$ for all $t \geq 0$.

Lemma 2.2.2

There is a $M > 0$ such that for any positive solution (s(t), y(t), i(t)) of the system (2.2) $y(t) < M$ for large t, where $M = \frac{(1+\theta_4)(1+\gamma_0)^2}{4}$ and $\gamma_0 \leq \theta_3$.

Proof of lemma 2.2.1 is routine work and hence omitted.

Theorem 2.2.1

The set $\Omega = \{(s, y, i) \in R_0^3, +: s + i \leq 1, y \leq M\}$ is a global attractor in $R_0^3, +$ and it is positively invariant.

Proof: First assume that $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in \Omega$. Then lemma 2.2.1 implies that $s(t) + i(t) < 1$ for all $t > 0$ and also by the lemma 2.2.2 we know that $y(t) < M$ for large t. Let us remark that if $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in \delta\Omega, \theta \in [-\tau, 0]$, because $\phi_1(\theta) + \phi_2(\theta) = 1$ or $\phi_3(\theta) = M$ or both, then still the corresponding solutions $(s(t), y(t), i(t))$ immediately enter into interior Ω or coincide with E_1. Next assume that

($_1(\theta)$, $_2(\theta)$, $_3(\theta)$) does not belongs to Ω for $\theta \in [\ \tau,\ 0]$. Now due to lemma 2.2.1 and 2.2.2 either there exists a positive time $T = \max(t\ , t_0)$, such that the corresponding solution $(s(t), y(t), i(t) \in \Omega$ for $t > T$ or the corresponding solution is such that $(s(t), y(t), i(t) \to E_1(1,0,0))$ as $t \to +\infty$. But $E_1 \in \delta\Omega$. Hence the global attractively of Ω in $R_0^3, +$ has been proved.

3. Qualitative Analysis of the Model System

3.1 Equilibria and Existence

The system of equation (2.2) has six equilibrium points, namely $E_0(0,0,0)$, $E_1(0,0,0)$, $E_2(0,0,1)$, $E_3\left(\frac{\theta_3}{\theta_1}, \frac{\theta_1-\theta_3}{\theta_1(1+\theta_1)}, 0\right)$ $E_4(s\ , 0, y\ )$, where s can be obtained from the equation $\theta_4^2 s^{\ 3}\ \ 2\theta_4(\theta_4\ \ \theta_2\theta_5)s^{\ 2} + (\theta_4\ \ \theta_2\theta_5)s\ \ \theta_2^2\theta_4^2 = 0$ and $E_5(s, \iota, \hat{y})$ Where $s = \frac{\theta_3}{\theta_1}$, $\iota = \frac{\theta_3\theta_4(\theta_1-\theta_3)-\theta_2\theta_4\theta_1^{\frac{3}{4}}\theta_3^{\frac{1}{2}}-\theta_1\theta_2\theta_3\theta_5}{\theta_1\theta_4\theta_3(1+\theta_1)}$ and $\hat{y} = 1 + \frac{\theta_5}{\theta_4}\sqrt{\frac{\theta_3}{\theta_1}}$. It is easy to see that the equilibrium point $E_0(0,0,0)$, $E_1(k,0,0)$, $E_2(0,0,1)$ and $E_4(s\ , 0, y\ )$ exist for all parametric values. The equilibrium point $E_3\left(\frac{\theta_3}{\theta_1}, \frac{\theta_1-\theta_3}{\theta_1(1+\theta_1)}, 0\right)$ and $E_5(s, \iota, \hat{y})$ both exist if $\theta_1 > \theta_3$.

Lemma 3.1.1

The diseases free equilibrium point $E_4(s\ , 0, y\ )$ is always exists.

Proof: We obtained from the equation (2.2), $y\ = 1 + \frac{\theta_5}{\theta_4}\sqrt{s}$. Clearly y is positive. Now we consider the case for s, s can be obtained from the relation $\theta_4^2 s^{\ 3}\ \ 2\theta_4(\theta_4\ \ \theta_2\theta_5)s^{\ 2} + (\theta_4\ \ \theta_2\theta_5)s\ \ \theta_2^2\theta_4^2 = 0$. Now if $\theta_4 > \theta_2\theta_5$ then change of sign can be shown as follows: By descartes rule of sign there is at least one positive root. Again replace s by s we obtained $\theta_4^2 s^{\ 3}\ \ 2\theta_4(\theta_4\ \ \theta_2\theta_5)s^{\ 2}\ \ (\theta_4\ \ \theta_2\theta_5)s\ \ \theta_2^2\theta_4^2 = 0$.

So in this case we have no negative real root. Since it is cubic equation so one positive root is gauranted. One the other hand if $\theta_4 < \theta_2\theta_5$ similarly by Descartes rule of sign we must have a positive root. Thus any case we must have a positive root.

Lemma 3.1.2:

The planner equilibrium point $E_5(s, \iota, \hat{y})$ exists if $\theta_1 > \theta_3$.

Proof

We obtained from the equation (2.2), $s = \frac{\theta_3}{\theta_1}$ and $\hat{y} = \frac{\theta_3}{\theta_1}\sqrt{s}$.

Clearly they are both positive. Now we find out ι as, $\iota = \frac{s(1-s)-\sqrt{s}\theta_2\hat{y}}{s(1+\theta_1)}$. Now we substitute the values of s and $\hat{y}$ then we obtained $\iota = \frac{\theta_3\theta_4(\theta_1-\theta_3)-\theta_2\theta_4\theta_1^{\frac{3}{4}}\theta_3^{\frac{1}{2}}-\theta_1\theta_2\theta_3\theta_5}{\theta_1\theta_4\theta_3(1+\theta_1)}$.

Since $\theta_1, \theta_2, \theta_3,\ \theta_4, \theta_5$ are all positive, so it is positive if $\theta_1 > \theta_3$. Hence the equilibrium point is exists if $\theta_1 > \theta_3$.

Stability Analysis

In this section we perform stability analysis of our model system (2.2). Here we first calculate the variational matrix of this system (2.2)

$$\begin{matrix} 1 & 2s & i & i\theta_1 & \frac{y\theta_2}{2\sqrt{s}} & s(1+\theta_1) & \theta_2\sqrt{s} \\ & i\theta_1 & & s\theta_1 & \theta_3 & 0 & \\ & \frac{y\theta_5}{2\sqrt{s}} & & 0 & \theta_4 & 2y\theta_4 + \theta_5\sqrt{s} & \end{matrix}$$

Lemma 3.2.1

The equilibrium points $E_0(0,0,0)$, $E_1(0,0,0)$ and $E_3\left(\frac{\theta_3}{\theta_1}, \frac{\theta_1-\theta_3}{\theta_1(1+\theta_1)}, 0\right)$ is unstable and $E_2(0,0,1)$ is stable.

The proof of the above lemma is obvious and hence omitted.

Lemma 3.2.2

The equilibrium point $E_4(s\ , 0, y\ )$ is always locally asymptotically stable. The variational matrix of the system around the this equilibrium point is as follows:

$$\begin{matrix} 1 & 2s & i & i\theta_1 & \frac{y\ \theta_2}{2\sqrt{s}} & s\ (1+\theta_1) & \theta_2\sqrt{s} \\ & 0 & & s\ \theta_1 & \theta_3 & 0 & \\ & \frac{y\ \theta_5}{2\sqrt{s}} & & 0 & \theta_4 & 2y\ \theta_4 + \theta_5\sqrt{s} & \end{matrix}$$

This can be written as

$$V = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ 0 & b_{22} & 0 \\ b_{31} & 0 & b_{33} \end{pmatrix}$$

Where, $b_{11} = 1\ \ 2s\ \ i\ \ i\theta_1\ \ \frac{y\ \theta_2}{2\sqrt{s}}$, $b_{12} =\ \ s\ (1+\theta_1)$, $b_{13} =\ \ \theta_2\sqrt{s}$, $b_{22} = s\ \theta_1\ \ \theta_3$, $b_{31} = \frac{y\ \theta_5}{2\sqrt{s}}$, $b_{33} = \theta_4\ \ 2y\ \theta_4 + \theta_5\sqrt{s}$. The corresponding characteristic equation is $\lambda^3 + A\lambda^2 + B\lambda + C = 0$, where $A =\ \ (b_{11}+b_{33} + b_{22})$, $B = (b_{22}b_{33} + b_{11}b_{33}\ \ b_{31}b_{13} + b_{11}b_{22})$, $C = b_{13}b_{31}b_{22}\ \ b_{11}b_{22}b_{33}$. Clearly, $b_{11} < 0$, $b_{22} < 0$ then $A > 0$, $C > 0$ and $AB\ \ C > 0$. Hence, by Routh-Hurth criteria all the eigen values are of negative real part. Hence it is stable.

Lemma 3.2.3

The interior equilibrium point $E_5(s, \iota, \hat{y})$ is locally asymptotically stable if $\theta_3\theta_4 > \theta_1\theta_2[\theta_5 + \theta_4\sqrt{\frac{\theta_1}{\theta_3}}]$ and unstable if $\theta_3\theta_4 < \theta_1\theta_2[\theta_5 + \theta_4\sqrt{\frac{\theta_1}{\theta_3}}]$.

Proof

The variational matrix V of system (2.2) around the positive equilibrium point $E_5(s,\ \iota,\ \hat{y})$ is as follows:

$$\begin{matrix} 1 & 2s & \iota & \iota\theta_1 & \frac{\hat{y}\theta_2}{2\sqrt{s}} & s(1+\theta_1) & \theta_2\sqrt{s} \\ & \iota\theta_1 & & s\theta_1 & \theta_3 & 0 & \\ & \frac{\hat{y}\theta_5}{2\sqrt{s}} & & 0 & \theta_4 & 2\hat{y}\theta_4 + \theta_5\sqrt{s} & \end{matrix}.$$

This can be written as

$$V = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & 0 & 0 \\ a_{31} & 0 & a_{33} \end{pmatrix}.$$

Where $a_{11} > 0$ or $a_{11} < 0$ according as $2s + \iota + \iota\theta_1 + \frac{\hat{y}\theta_2}{2\sqrt{s}} < 1$ or $2s + \iota + \iota\theta_1 + \frac{\hat{y}\theta_2}{2\sqrt{s}} > 1$, $a_{12} < 0$, $a_{13} < 0$, $a_{21} > 0$, $a_{31} > 0$, $a_{33} < 0$. The corresponding characteristic equation is $\lambda^3 + A_1\lambda^2 + B_1\lambda + C_1 = 0$, where $A_1 = (a_{11}+a_{33})$, $B_1 = (a_{11}a_{33} \quad a_{31}a_{13} \quad a_{21}a_{12})$, $C_1 = a_{12}a_{21}a_{33}$.

Case I: When $\theta_3\theta_4 > \theta_1\theta_2[\theta_5 + \theta_4\sqrt{\frac{\theta_1}{\theta_3}}]$, then $A_1 > 0$, $C_1 > 0$ and $A_1B_1 \quad C_1 > 0$. In this case, by Routh-Hurwitz criterion the interior equilibrium point is locally asymptotically stable.

Case II: When $\theta_3\theta_4 < \theta_1\theta_2[\theta_5 + \theta_4\sqrt{\frac{\theta_1}{\theta_3}}]$, then we have $A_1 < 0$. Hence, it is unstable.

Interpretation of system dynamics and Numerical calculation

In previous section we find out the existence and stability condition of the different equilibrium points. We see that the diseases free equilibrium point $E_4(s\ ,0,y\ )$ is locally asymptotically stable when $s < \frac{\theta_3}{\theta_1}$ and the interior equilibrium point $E_5(s,\iota,\hat{y})$ exists if $\theta_1 > \theta_3$ and locally asymptotically stable if $\theta_3\theta_4 > \theta_1\theta_2[\theta_5 + \theta_4\sqrt{\frac{\theta_1}{\theta_3}}]$. It is to be noted that in disease-selective predation model suggested by Roy, Chattopadhyay (2005) and Alam (2009), the predation rate plays an important role either to get disease free system or to keep the three population in stable. But in this model system we observe that the existence and stability of disease free equilibrium point $E_4(s\ ,0,y\ )$ does not depend on predation rate (θ_2).; rather it depends jointly on rate of infection (θ_1) and death rate of infected prey (θ_3) . Thus, here the control of the system shifts from the predation rate to the rate of infection and the death rate of infected prey, just because of the herd behavior of prey. Here we observe if the number of susceptible prey can be maintained below a threshold value which is jointly determined by the rate of (θ_1) and the death rate of infected prey (θ_3) , then the system can easily move to stable disease free equilibrium point. This phenomenon has been shown numerically in Figure 1. The Figure 1 depicts the stability of disease free equilibrium point. Here the simulation is perform over a wide range of predation rate (θ_2) and we obtained robust stability of disease free equilibrium point. This can be interpreted in the way that due to herd behavior of prey, the predation rate (θ_2) does not play much crucial role in the stability of disease free equilibrium point; whereas it was crucially effected in the dynamics of the model which was suggested by Roy and Chattopadhyay (2005) and Alam (2009).

Furthermore, the existence and stability of three population mainly depend on rate of infection (θ_1) and the death rate of infected prey (θ_3) along with other parameter values in a very complex fashion. To understand this complexity, we study the phenomenon numerically which has been shown in Figure 1. The Figure 2 depicts the stability of interior equilibrium point. Like our previous numerical study, here the simulation is also performed over a wide range of predation rate $(\theta_2)$and we obtained a very robust stability of disease free equilibrium point.

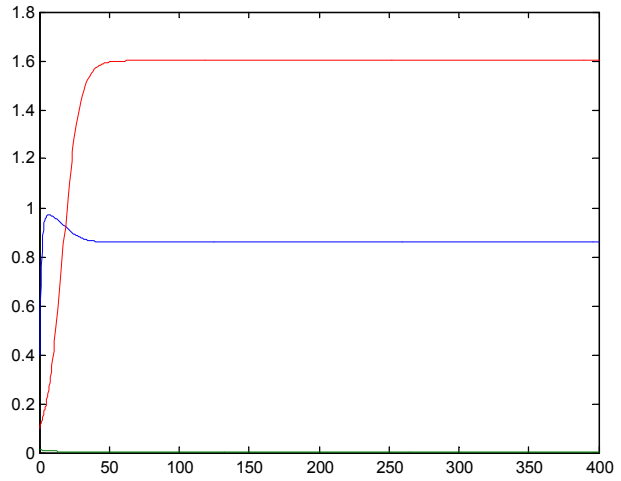

Figure 1. Depict the stability of planner equilibrium point. The model system (2.2) has been solved using following set of values of parameters : θ_1=0.11, θ_2=0.081, θ_3=0.17, θ_4=0.10, θ_5=0.8 $\theta_2$and the initial values as [s(0), i(0), y(0)]=[0.4,0.013,0.1]

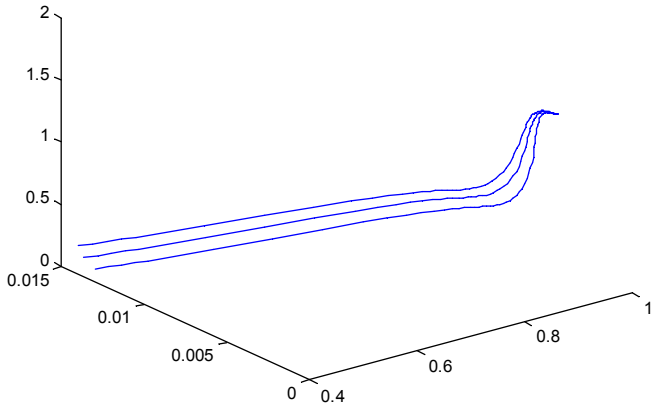

Figure 1.1. Depict the stability of planner equilibrium point in 3-dimentional space using same set of values of parameters and the three neighbouring initial values of (s, i, y)

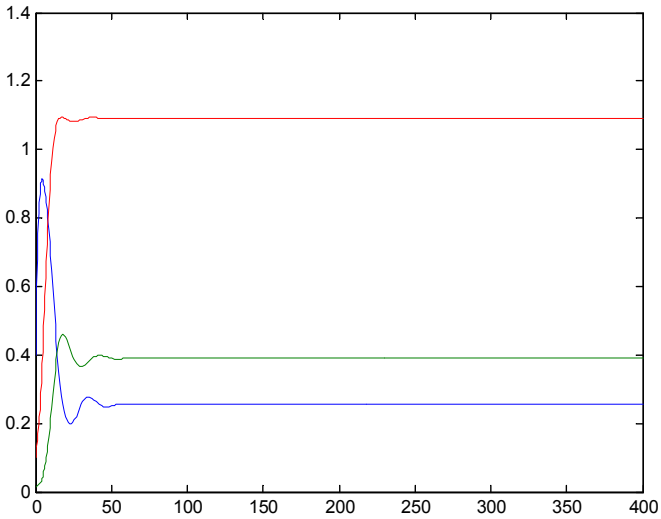

Figure 2. Depict the stability of interior equilibrium point. The model system (2.2) has been solved using following set of values of parameters: θ_1=0.51, θ_2=0.072, θ_3=0.13, θ_4=0.32, θ_5=0.8 θ_2 and the initial values as [s(0), i(0) ,y(0)] =[0.4,0.013,0.1]

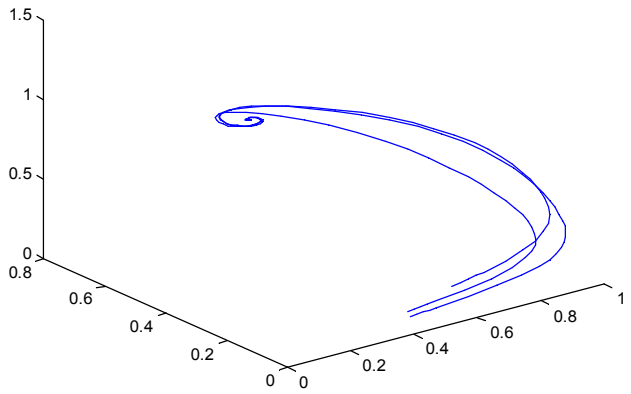

Figure 2.1. Phase space of 3-D plot of interior equilibrium point

Depict the stability of interior equilibrium point in 3-dimentional space using same set of values of parameters and the three neighbouring initial values of (s, i, y).

Conclusion

In the previous study of disease selective predation made by Alam [17], it was pointed out that the predation rate that is the parameter (θ_2) takes a crucial role in the diseases-selective predation system. If (θ_2) exceeds certain threshold value (regulated by other system parameters) then it has a possibility for extinction of the prey population. Further, the possibility of risk of extinction of prey population has been increased due to time lag for gestation. In real world biological communities with this type of selective predation, such as human population and fish population in a certain pond or lake, may tend to a situation of extinction of the prey population which sometimes may lead to ecological imbalance. But here analyzing our model system we observe that the existence and stability of diseases free equilibrium point $E_4(s\ ,0,y\ )$ does not depend on predation rate (θ_2) ; rather it depends jointly on rate of infection (θ_1) and death rate of infected prey (θ_3). Thus, here the control of the system shifts from the predation rate to the rate of infection and the death rate of infected prey, just because of the herd behavior of prey. Here we observe if the number of susceptible prey can be maintained below a threshold value which is jointly determined by the rate of (θ_1) and the death rate of infected prey (θ_3), then the system can easily move to stable disease free equilibrium point. Here we also observe that in case of fatal disease the disease free equilibrium can be achievable if the number of suspectable can be maintained below a certain threshold value which is determined by the ratio of death rate of infected prey and rate of infection of disease. This observation directly supports what the real practice we do in case of fatal diseases. Actually in our real life, in case of serious virus effects like bird flu, swine flu etc., we kill the suspectable prey to maintained the suspectable prey population below a certain threshold level as we observe in our analysis.

Furthermore, it has been pointed out by Flake et al. ([13]) that co-existence of three population (suspectable prey, infected prey and predator) is not possible if the infected prey has negative effect on the growth rate of predator population; where as in our problem we observe that for the disease selective with herd behavior of prey, the system may show the co-existence of three populations under certain restriction on the system parameter (mainly on the predation rate and on the death rate of infected prey). Thus, the idea of disease selective predation may be effectively applied to multi-prey systems where infection in some prey is harmful for their bio-diversity. 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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