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A predator-prey mathematical model with both the populations affected by disease

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Abstract. We propose and analyze a predator-prey model, in which both population is infected by disease. The total population has been classified into susceptible prey, infected prey, susceptible predator, and infected predator. The disease cannot be transmitted between prey and predator by predation. The predation ability of susceptible predator is stronger than infected ones. Local stability analysis of biologically feasible equilibria is worked out with the help of disease basic reproduction numbers. The system is persistent under some feasible parametric conditions. The numerical analysis is carried out to discuss some interesting results that our model exhibits.

1 Introduction

In ecology populations are infected by various diseases which plays a significant role in regulating the population sizes. Researchers give more attentions from several years past to study the dynamical behaviour of such populations. As a result several mathematical models have been developed. Also essential mathematical tools are derived in analyzing the interaction of different populations like predator-prey interaction. The study of predator-prey dynamics with an infected predator has a great importance in controlling the predator population. But this area has been neglected for a long time in theoretical ecology. Numerous prey-predator models with infectious diseases had investigated [1, 2, 3, 4, 5, 6, 7, 8, 9, 10] in which most of the authors considered the predator-prey model with only prey affected by some diseases. In particular, the authors in the reference [8] proposed a predator-prey

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model with logistic growth in the prey population including an SIS parasitic infection in the prey and observed that the infected prey being more vulnerable to predation. They also found that the infection in the prey population could promote coexistence. In the references [11, 12, 13] the authors used Holling type-II predation rate function, the disease is not genetic, and no immunity was considered.

In this paper, we consider a predator-prey model in which both the population affected by disease. The predation rate is different for the susceptible predator and infected predator. The disease is not transmitted vertically in the predator. In nature this type actually exists. For example [14], the white spot viral disease (by white spot baculovirus)gets transmitted by contact from the Nauplius stage of shrimp to other shrimp [15] which consumes the Phaeocystis phytoplankton infected by phaeccystis pouchetii viral disease [16] considered a marine planktonic system where both phytoplankton (such as Cryptophyte) and zooplankton (such as Rotifers) are infected by some viral disease. To explain the scenario, we formulate a mathematical model that the viral disease could be infected both the predator and prey populations, but it cannot be transmitted between them.

In the present paper, we derive the condition for existence of positive equilibria. We also perform local stability analysis of the positive interior equilibrium.

2 The mathematical model

To construct the mathematical model, we make the following assumptions:
• The prey whose total population density is denoted by S and the predator whose total population density is denoted by P.
• In presence of disease total prey population is divided into susceptible prey X and infected prey Y. The total predator population is also divided into susceptible predator Z and infected predator W. So, at time t total prey population is $S = X + Y$ and total predator population is $P = Z + W$.
• Disease does not spread from prey to predator by feeding or any other ways. We assume that the disease only transmits from susceptible prey X to infected prey Y and susceptible predator Z to infected predator W.
• We assume that the susceptible prey is capable of reproducing according to the logistic law

$$\frac{dX}{dt} = rX\left(1 - \frac{X+Y}{K}\right).$$

with carrying capacity $K(>0)$ and intrinsic growth rate $r(>0)$.
• The susceptible prey X becomes infected followed by the mass action law with the rate of infection α and also the susceptible predator Z is infected according to the mass action law with the rate of infection β. We also assume that infected prey and predator do not recover from the disease.
• Both predators consume only infected prey following Holling type-I functional response at constant rate c.

Considering the above basic assumptions we have the following mathematical model.

$$\begin{aligned}
\frac{dX}{dt} &= rX\left(1-\frac{X+Y}{K}\right)-\alpha XY \equiv F_1(X,Y,Z,W),\\
\frac{dY}{dt} &= \alpha XY - cY(Z+eW)-dY \equiv F_2(X,Y,Z,W),\\
\frac{dZ}{dt} &= r_1 cY(Z+eW)-\beta ZW-d_1 Z \equiv F_3(X,Y,Z,W),\\
\frac{dW}{dt} &= \beta ZW - d_2 W \equiv F_4(X,Y,Z,W),
\end{aligned} \tag{2.1}$$

where r_1 is the conversion rate factor for the susceptible predator due to consumption of infected prey, e $(0<e<1)$ stands for the impact of disease on the predation rate, and the parameters d, d_1, d_2 $(d_1 < d_2)$ are the death rates of infected prey, susceptible predator and infected predator respectively.

The system (2.1) has to be analyzed with the following initial conditions,

$$X(0)>0,\ Y(0)>0,\ Z(0),\ W(0)>0. \tag{2.2}$$

3 Qualitative analysis of the system

3.1 Boundedness of the System

Theorem 3.1. All the solutions of the system (2.1) are bounded.

Proof: Consider the function $U(t)=X(t)+Y(t)+Z(t)+W(t)$.

Now, using the equations (2.1), we have

$$\begin{aligned}
\tfrac{dU}{dt} &= \frac{dX}{dt}+\frac{dY}{dt}+\frac{dZ}{dt}+\frac{dW}{dt}\\
&= rX(1-\frac{X+Y}{K})-c(1-r_1)Y(Z+eW)-dY-d_1Z-d_2W\\
&\leq rX-\frac{r}{K}X^2-\frac{r}{K}XY-dY-d_1Z-d_2W, \text{ if } r_1\leq 1.
\end{aligned}$$

Therefore, $\frac{dU}{dt}+\mu U \leq (r+\mu)X-\frac{r}{K}X^2-\frac{r}{K}XY-(d-\mu)Y-(d_1-\mu)Z-(d_2-\mu)W$.

Let $\mu=\min\{d,d_1,d_2\}$, then $\frac{dU}{dt}+\mu U \leq X(r+\mu-\frac{r}{K}Y)\leq \frac{K(r+\mu)^2}{4r}$.

If we take $r_1\leq 1$ and $\mu=\min\{d,d_1,d_2\}$, then for each $\mu\geq 0$ the above inequality becomes

$$\frac{dU}{dt}+\mu U \leq m, \text{ where } m=\frac{K(r+\mu)^2}{4r}.$$

Now by the theory of differential inequality (Birkhoff and Rota [17]), we have

$$0\leq U(t)\leq \frac{m}{\mu}(1-e^{-\mu t})+U(0)e^{-\mu t}.$$

As $t\to\infty$, then $0\leq U(t)\leq \frac{m}{\mu}$. Hence $U(t)$ is a bounded quantity.

Thus all the solutions of the system (2.1) are confined in the region

$$\Omega=\left\{(X,Y,Z,W): 0\leq X(t)+Y(t)+Z(t)+W(t)\leq \frac{m}{\mu}+\varepsilon, \text{ for all } \varepsilon>0\right\}.$$

3.2 Equilibria Analysis

- The equilibria $\mathbf{E}_0(\ 0,\ 0,\ 0,\ 0\ )$ and $\mathbf{E}_1(\ K,\ 0,\ 0,\ 0\ )$ exist for all parametric values.
- The predators free equilibrium point $\mathbf{E_2}(\ \overline{X},\ \overline{Y},\ 0,\ 0\ )$ exists if $R_0 < 1$, where $R_0 = \frac{d}{K\alpha}$, $\overline{X} = \frac{d}{\alpha}$ and $\overline{Y} = \frac{r(K\alpha - d)}{\alpha(r+K\alpha)}$.
- The infected predator free equilibrium point $\mathbf{E_3}(\ \widetilde{X},\ \widetilde{Y},\ \widetilde{Z},\ 0\ )$ exists if $R_1 < 1$, where $R_1 = \frac{d_1(r+K\alpha)}{Krr_1c} + \frac{d}{K\alpha}$
  $\widetilde{X} = K\left\{1 - \frac{d_1(r+K\alpha)}{Krr_1c}\right\}$, $\widetilde{Y} = \frac{d_1}{r_1c}$, $\widetilde{Z} = \frac{K\alpha}{c}\left\{1 - \frac{d_1(r+K\alpha)}{Krr_1c} - \frac{d}{K\alpha}\right\}$.
- The positive interior equilibrium point $\mathbf{E}^*(\ X^*,\ Y^*,\ Z^*,\ W^*\ )$ exists if

$$R_2 = \frac{d_2\alpha}{cerr_1}\frac{(K\alpha + r)}{(K\alpha + d)} < 1, R_3 = \frac{\beta Kdcerr_1 + ced_1d_2(r+K\alpha)}{d_2(r+K\alpha)(\beta d + cd_2)} < 1$$

, where $Y^* = \frac{r(K-X)}{(r+K\alpha)}$, $Z^* = \frac{d_2}{\beta}$ and $W^* = \frac{1}{ce}\left\{\alpha X^* - (d + \frac{cd_2}{\beta})\right\}$ and X^* is the positive root of the equation $AX^2 - BX - C = 0$, and the coefficients are given by

$$\begin{aligned} A &= \frac{\alpha\beta err_1}{r+K\alpha}, \\ B &= \beta err_1\frac{(K\alpha+d)}{r+K\alpha} - \alpha\beta\frac{d_2}{c}, \\ C &= \frac{d_2}{c}(\beta d + cd_2) - ed_1d_2 - \frac{\beta Kderr_1}{r+K\alpha}. \end{aligned}$$

3.3 Stability Analysis

The stability of the system (2.1) at each equilibria is obtained by using Routh-Hurwitz stability criterion.

For this we have to compute the variational matrix $V(X,Y,Z,W)$ of the system (2.1) and check the stability at each of the equilibrium.

Result 1. The equilibrium point $\mathbf{E_0} : (0,0,0,0)$ is unstable saddle as one eigenvalue of the variational matrix is positive.

Result 2. The equilibrium point $\mathbf{E_1} : (\ K,\ 0,\ 0,\ 0\ )$ is stable if $R_{01} < 1$, where $R_{01} = \frac{1}{R_0} = \frac{K\alpha}{d}$.

Result 3. The predators free equilibrium point $\mathbf{E_2} : (\ \overline{X},\ \overline{Y},\ 0,\ 0\ )$ is stable if $R_{02} = r_1cr\frac{(K\alpha-d)}{d_1(r+K\alpha)} < 1$.

Result 4. The infected predator free equilibrium point $\mathbf{E_3} : (\ \widetilde{X},\ \widetilde{Y},\ \widetilde{Z},\ 0\ )$ is locally asymptotically stable if $R_{03} = \frac{K\alpha\beta}{cd_2}(1 - R_1) < 1$.(For proof see Appendix.)

Stability analysis of the positive interior equilibrium and Hopf bifurcation

Theorem 3.2. The positive interior equilibrium point $E^*(\ X^*,\ Y^*,\ Z^*,\ W^*\ )$ is locally asymptotically stable if $\sigma_1 > 0, \sigma_4 > 0, \sigma_1\sigma_2 - \sigma_3 > 0, \sigma_3(\sigma_1\sigma_2 - \sigma_3) - \sigma_4\sigma_1^2 > 0$, where $\sigma_i's$ are given in the proof of the theorem.

Proof: The variational matrix of the system (2.1) around the the positive equilibrium point $E^*(\ X^*,\ Y^*,\ Z^*,\ W^*)$ is

$$V^* = \begin{bmatrix} -m_{11} & -m_{12} & m_{13} & m_{14} \\ m_{21} & m_{22} & -m_{23} & -m_{24} \\ m_{31} & m_{32} & -m_{33} & -m_{34} \\ m_{41} & m_{42} & m_{43} & m_{44} \end{bmatrix}$$

where $m_{11} = \frac{r}{K}X^* > 0,\ m_{12} = (\frac{r}{K} + \alpha)X^* > 0, m_{13} = m_{14} = 0; m_{21} = \alpha Y^* > 0,\ m_{22} = 0,\ m_{23} = cY^* > 0,\ m_{24} = ceY^* > 0; m_{31} = 0,\ m_{32} = r_1c(Z^* + eW^*) > 0,\ m_{33} = \frac{\beta er_1c}{d_2}Y^*W^* > 0,\ m_{34} = d_2 - r_1ceY^*; m_{41} = m_{42} = 0, m_{43} = \beta W^* > 0, m_{44} = 0.$

The characteristic equation of the variational matrix V^* at E^* is given by

$$\rho^4 + \sigma_1\rho^3 + \sigma_2\rho^2 + \sigma_3\rho + \sigma_4 = 0,$$

where

$\sigma_1 = (m_{11} + m_{33}) = \frac{r}{K}X^* + \frac{\beta ecr_1}{d_2}Y^*W^* > 0$

$$\begin{aligned}\sigma_2 &= m_{11}m_{33} + m_{34}m_{43} + m_{12}m_{21} + m_{23}m_{32} \\ &= \frac{\beta cerr_1}{d_2K}X^*Y^*W^* + \beta(d_2 - r_1ce\,Y^*)W^* + \alpha(\frac{r}{K} + \alpha)\,X^*\,Y^* + r_1c^2(Z^* + eW^*)Y^*\end{aligned}$$

$$\begin{aligned}\sigma_3 &= m_{21}m_{12}m_{33} + m_{34}m_{43}m_{11} + m_{11}m_{23}m_{32} + m_{24}m_{32}m_{43} \\ &= \frac{\alpha\beta cer_1}{d_2K}(\frac{r}{K} + \alpha)X^*\,Y^*\,Y^*\,W^* + \frac{r\beta}{K}(d_2 - r_1ce\,Y^*)\,X^*W^* \\ &+ \frac{rr_1c^2}{K}(Z^* + eW^*) + \beta ec^2r_1(Z^* + eW^*)Y^*\,W^*\end{aligned}$$

$$\begin{aligned}\sigma_4 &= m_{12}m_{21}m_{34}m_{43} + m_{11}m_{24}m_{32}m_{43} \\ &= \left\{\alpha\beta(\frac{r}{K} + \alpha)(d_2 - r_1ceY^*) + \frac{\beta rr_1ec^2}{K}(Z^* + eW^*)\right\}X^*\,Y^*\,W^*\end{aligned}$$

$$\begin{aligned}\sigma_1\sigma_2 - \sigma_3 &= m_{11}(m_{12}m_{21} + m_{11}m_{33}) + \\ &\quad m_{33}(m_{11}m_{33} + m_{23}m_{32} + m_{34}m_{43}) - m_{24}m_{32}m_{43}\end{aligned}$$

$$\begin{aligned}\sigma_3(\sigma_1\sigma_2 - \sigma_3) - \sigma_4\sigma_1^2 &= m_{11}m_{34}m_{43}(m_{11}^2m_{33} + m_{11}m_{33}^2 + m_{33}m_{23}m_{32} + m_{33}m_{34}m_{43}) + \\ &\quad m_{11}m_{32}m_{23}(m_{11}m_{12}m_{21} + m_{11}^2m_{33} + m_{11}m_{33}^2 + m_{33}m_{23}m_{32} + \\ &\quad m_{33}m_{34}m_{43}) + m_{24}m_{32}m_{43}(m_{11}m_{12}m_{21} + m_{11}^2m_{33} + m_{33}m_{32}m_{23} + \\ &\quad + m_{33}m_{34}m_{43}) + m_{12}m_{21}m_{33}(m_{11}m_{12}m_{21} + m_{11}m_{33}^2 + m_{33}m_{32}m_{23}) \\ &\quad - m_{24}m_{32}m_{43}(m_{11}m_{34}m_{43} + m_{11}m_{32}m_{23} + m_{24}m_{32}m_{43} + \\ &\quad + m_{12}m_{21}m_{33} + m_{11}^3 + 2m_{11}^2m_{33}) - 2m_{11}m_{33}m_{12}m_{21}m_{34}m_{43}\end{aligned}$$

Using the Routh-Hurwitz criteria, we observe that the system (2.1) is locally asymptotically stable around the interior equilibrium point E^* if $\sigma_1 > 0,\ \sigma_4 > 0,\ \sigma_1\sigma_2 - \sigma_3 > 0,\ \sigma_3(\sigma_1\sigma_2 - \sigma_3) - \sigma_4\sigma_1^2 > 0.$

Theorem 3.3. The Hopf-bifurcation of the equivalent equilibrium E^* occurs at $\lambda_1 = \lambda_1^* \in (0, \infty)$ if and only if

(i)$\Psi(\lambda_1^*) = 0$
(ii)$\sigma_1^3\sigma_2'\sigma_3(\sigma_1 - 3\sigma_3) > 2(\sigma_2\sigma_1^2 - 2\sigma_3^2)(\sigma_3'\sigma_1^2 - \sigma_1'\sigma_3^2)$.
and all other eigenvalues are of negative real parts, where $\rho(\lambda_1)$ is purely imaginary at $\lambda_1 = \lambda_1^*$.

Proof: By the condition $\Psi(\lambda_1^*) = 0$, the characteristic equation can be written as

$$(\rho^2 + \frac{\sigma_3}{\sigma_1})(\rho^2 + \sigma_1\rho + \frac{\sigma_1\sigma_4}{\sigma_3}) = 0.$$

If this equation has four roots, say $\rho_i (i = 1,2,3,4)$ with the pair of purely imaginary roots at $\lambda_1 = \lambda_1^*$ as $\rho_1 = \bar{\rho}_1$, then we have

$$\rho_3 + \rho_4 = -\sigma_1 \tag{3.1}$$

$$\omega^2 + \rho_3\rho_4 = \sigma_2 \tag{3.2}$$

$$\omega_0^2(\rho_3 + \rho_4) = -\sigma_3 \tag{3.3}$$

$$\omega_0^2\rho_3\rho_4 = \sigma_4 \tag{3.4}$$

where $\omega_0 = Im\rho_1(\lambda_1^*) = \frac{\sigma_3}{\sigma_1}$. Now, if ρ_3 and ρ_4 are complex conjugate, then from (3.3) it follows that $2Re\rho_3 = -\sigma_1$; if ρ_3 and ρ_4 are real roots, then by (3.3) and (3.4) $\rho_3 < 0$ and $\rho_4 < 0$. To complete the discussion, it remains to verify the transversality conditions.

As $\Psi(\lambda_1^*)$ is a continuous function of all its roots, so there exists an open interval $\lambda_1 \in (\lambda_1^* - \varepsilon, \lambda_1^* + \varepsilon)$ where ρ_1 and ρ_2 are complex conjugate for λ_1. Suppose, their general form in this neighbourhood are

$$\rho_1(\lambda_1) = \chi(\lambda_1) + i\nu(\lambda_1)$$

$$\rho_2(\lambda_1) = \chi(\lambda_1) - i\nu(\lambda_1)$$

Now, we shall verify the transversality condition

$$\left[\frac{dRe(\rho_j(\lambda_1))}{d\lambda_1}\right]_{\lambda_1=\lambda_1^*} > 0, j = 1,2.$$

$\rho_1(\lambda_1) = \chi(\lambda_1) \pm i\nu(\lambda_1)$, into the characteristic equation $D(\rho) = 0$ and calculating the derivative, we have

$$Q(\lambda_1)\chi'(\lambda_1) - L(\lambda_1)\nu'(\lambda_1) + M(\lambda_1) = 0 \tag{3.5}$$

$$Q(\lambda_1)\chi'(\lambda_1) + L(\lambda_1)\nu'(\lambda_1) + N(\lambda_1) = 0$$

where

$$\begin{aligned}
Q(\lambda_1) &= 4\chi^3 - 12\chi\nu^2 + 3\sigma_1(\chi^2 - \nu^2) + 2\sigma_2\chi + \sigma_3 \\
L(\lambda_1) &= 12\chi^2\nu + 6\sigma_1\chi\nu - 4\nu^3 + 2\sigma_2\nu \\
M(\lambda_1) &= \sigma_1\chi^3 - 3\sigma_1'\chi\nu^2 + \sigma_2'(\chi^2 - \nu^2) + \sigma_3'\chi \\
N(\lambda_1) &= 3\sigma_1'\chi^2\nu - \sigma_1'\nu^3 + 2\sigma_2'\chi\nu + \sigma_3'\nu
\end{aligned}$$

Solving for $\chi'(\lambda_1^*)$, we have

$$\begin{aligned}\chi'(\lambda_1)_{\lambda_1=\lambda_1^*} &= \left[\frac{d}{d\lambda_1}\left\{Re(\rho_j(\lambda_1))\right\}\right]_{\lambda_1=\lambda_1^*} \\ &= -\frac{L(\lambda_1^*)N(\lambda_1^*)+Q(\lambda_1^*)M(\lambda_1^*)}{Q^2(\lambda_1^*)+L^2(\lambda_1^*)} \\ &= \frac{\sigma_1^3\sigma_2'\sigma_3(\sigma_1-3\sigma_3)-2(\sigma_2\sigma_1^2-2\sigma_3^2)(\sigma_3'\sigma_1^2-\sigma_1'\sigma_3^2)}{\sigma_1^4(\sigma_1-3\sigma_3)^2+4(\sigma_2\sigma_1^2-2\sigma_3^2)^2} > 0, \\ &\quad \text{if } \sigma_1^3\sigma_2'\sigma_3(\sigma_1-3\sigma_3) > 2(\sigma_2\sigma_1^2-2\sigma_3^2)(\sigma_3'\sigma_1^2-\sigma_1'\sigma_3^2).\end{aligned}$$

Hence proves the theorem.

4 Biological interpretation

(a) $R_{01} = \frac{K\alpha}{d}$, this is the parameter which determines the local stability of the equilibrium point $E_1(K,0,0,0)$. Here, $K\alpha$ is the birth rate of infected prey at E_1 and d is the removal of infected prey due to disease induced mortality. So, R_{01} is the disease basic reproduction number at E_1. Thus when $R_{01} < 1$, predators will become extinct, and so will infected prey. The condition result in E_1 being stable. Therefore, stability of E_1 imply that E_2 does not exist.
(b) $R_{02} = r_1 cr\frac{(K\alpha-d)}{d_1\alpha(r+K\alpha)}$, this is the parameter which determines the local stability of the steady state $E_2(\overline{X},\overline{Y},0,0)$. Here, $r_1 cr\frac{(K\alpha-d)}{(r+K\alpha)}$ is the birth rate of susceptible predator at the equilibrium point E_2 and $\frac{1}{d_1}$ is the mean life span of susceptible predator at E_2. So their product R_{02} is the mean number new born predators, which is considered as ecological basic reproduction number at E_2.Thus $R_{02} < 1$ implies the predators will become extinct. The condition result in E_2 being stable.
(c) $R_{03} = \frac{K\alpha\beta}{cd_2}(1-R_1)$, determines the local stability of the equilibrium point $E_3(\widetilde{X},\ \widetilde{Y},\ \widetilde{Z},\ 0)$. R_{03} is the ecological basic reproduction number at E_3. Thus $R_{03} < 1$ implies the infected predator will become extinct. The condition result in E_3 being stable.

5 Numerical simulations

Parameter	Definition	Default Value
r	Growth rate of susceptible prey	1
K	Carrying capacity	300
α	The infectious rate in prey populations	0.032
c	Consume rate of susceptible predator	0.013
e	The impact of disease on predation rate, where $0 < e < 1$	0.55
r_1	Conversion rate of predator due to consumption of prey	0.9
β	The infectious rate in predator populations	0.016
d	Mortality rate of infected prey	0.01
d_1	Mortality rate of susceptible predator	0.001
d_2	Mortality rate of infected predator	0.21

Table 1 A set of parametric values.

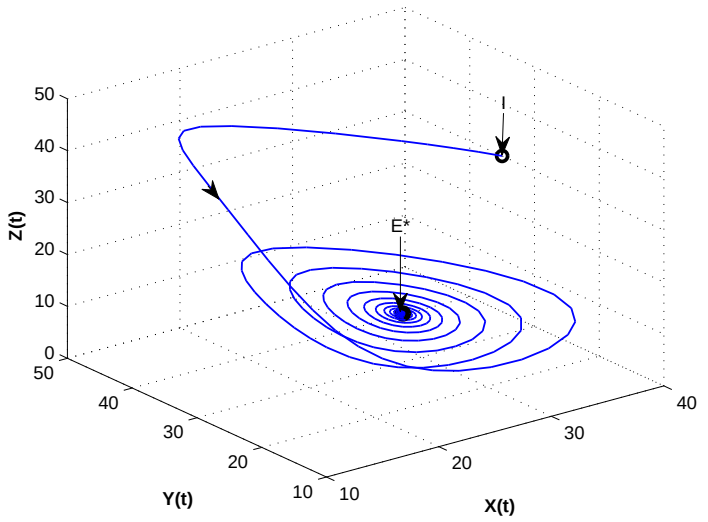

Fig. 1 The equilibrium point E^* is locally asymptotically stable for the set of parameter in the Table 1.

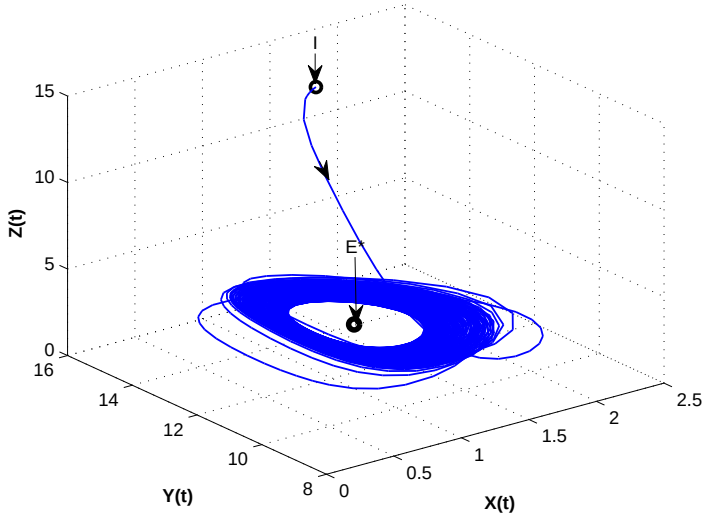

Fig. 2 The figures depicts oscillatory behaviour of susceptible prey, infected prey and susceptible predator populations for $\alpha = .085$ and $\beta = .056$, with same set of parameter as given in the Table1.

In this section, our study focused on the occurrence and termination of the disease. We begin with a set of parameter (see Table1) for which the existence conditions of the coexistence equilibrium point E^* is satisfied and the coexistence equilibrium point $E^* = (25.9197, 25.8566, 13.1250, 90.7419)$ is locally asymptotically stable in the form of a stable focus with eigenvalues $-0.0580 \pm i\ 0.9839, -01.0574, -0.0634$ (see Figure1). Keeping the other parameters fixed and increasing the value of the parameters α from 0.032 to 0.085 and β from 0.016 to 0.056,
we observe that the solution of (2.1) changes from stable behavior to oscillatory behavior (see Figure 2). Next, to observe the effects of some parameters on system (2.1), we first consider $\alpha = 0.004$ and

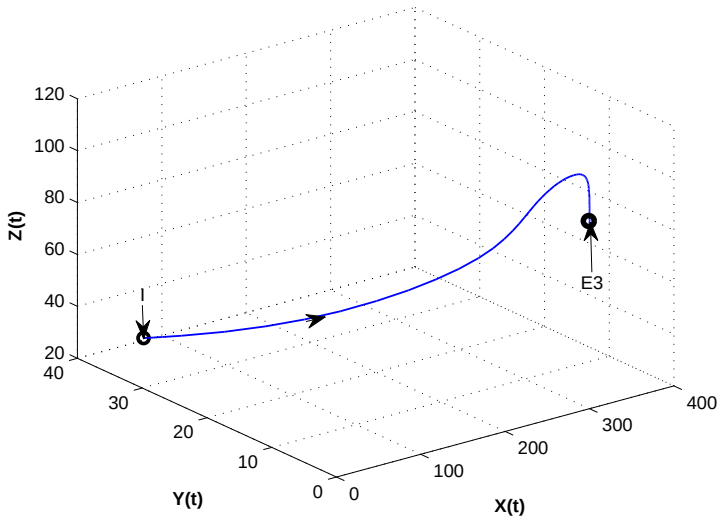

Fig. 3 The figure depicts that for $\alpha = .004$ and $\beta = .0014$, E^* approaches infected predator free equilibrium E_3 with other parametric values kept fixed in the Table 1.

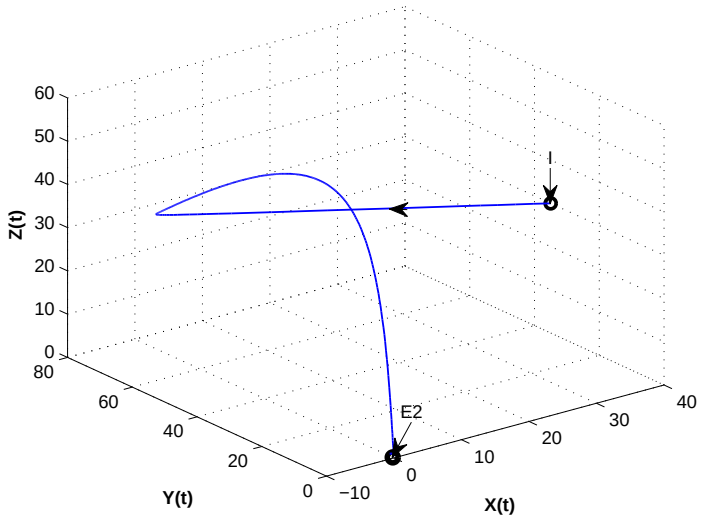

Fig. 4 The figure depicts that for $\alpha = 2.6$ and $\beta = .01$, E^* approaches susceptible and infected predator free equilibrium E_2 with other parametric values kept fixed in the Table 1.

$\beta = 0.0014$ and observe that the infected predator population goes to extinction (see Figure 3). Taking $\alpha = 2.6$ and $\beta = 0.01$, we observe that the system (2.1) goes to predator free equilibrium point (see Figure 4). Also, for $\alpha = 0.00001$ and $\beta = 0.015$, system (2.1) goes to infected prey and predator free equilibrium point (see Figure 5). Finally, for a clear understanding of the dynamical changes of system (2.1) due to changing the value of the parameter α, from 0.04 to 0.09; a bifurcation diagram is plotted as shown in (Figure 6).

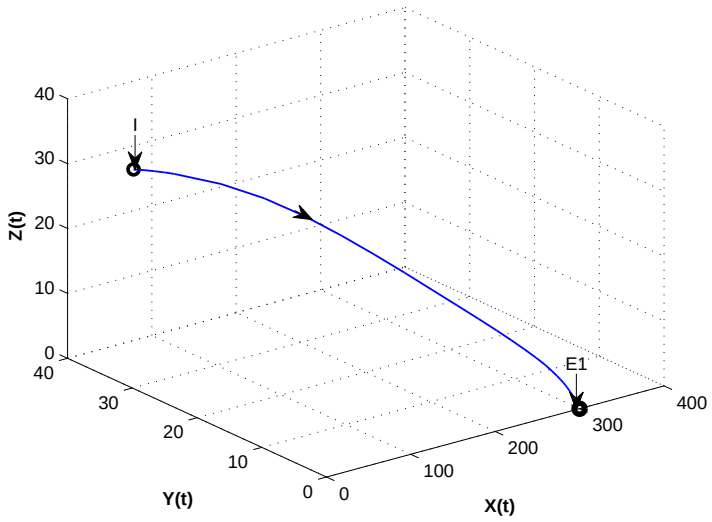

Fig. 5 The figure depicts that for $\alpha = .00001$ and $\beta = .015$, E^* approaches infected prey, susceptible predator and infected predator free equilibrium E_1 with other parametric values kept fixed in the Table 1.

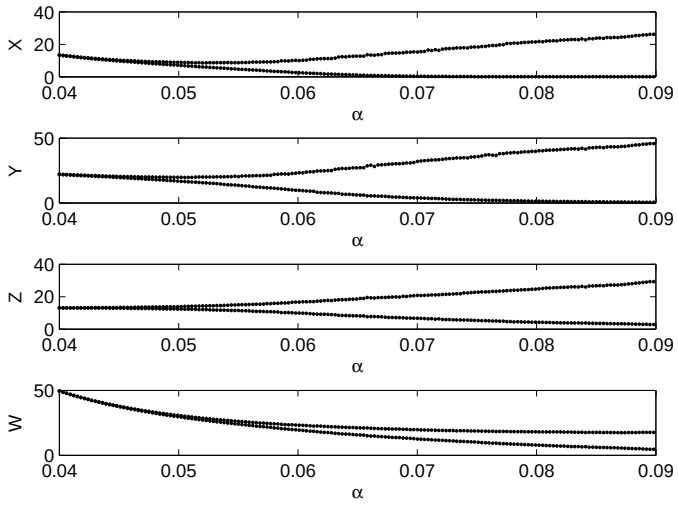

Fig. 6 The bifurcation diagram of all the populations with α as the bifurcation parameter.

6 Conclusion

In this paper, we discuss a predator-prey model with infectious disease which can transmit both in predator and prey, but cannot transmit between predator and prey. We find that lots of diseases are not transmitted vertically [15], so we assume that the disease in the predator is not genetic. This is the major difference with [13]. The non-linear differential equations are set up. Through the discussion on the stability of equilibrium points, we obtain four stable equilibrium points corresponding to four existence statuses with different species. Furthermore, we discuss the basic reproduction numbers

which are obtained analytically. And the threshold parameters which are combined to meaningful biological conditions are given too.

7 Appendix

Since one of the eigenvalues of the variational matrix computed around E_0 is $r > 0$, so the equilibrium point E_0 is always unstable node for all parametric values.
The characteristic roots of the variational matrix V_1 computed around E_1 are $-r\,, -(d-K\alpha)\,, -d_1\,, -d_2$. Hence E_1 is stable if $R_{01} < 1$, where $R_{01} = \frac{1}{R_0} = \frac{K\alpha}{d}$.

The characteristic roots of the variational matrix V_2 computed around E_2 are $-d_2,\ \frac{crr_1(K\alpha-d_1)}{\alpha(K\alpha+r)} - d_1$ and the negative roots of the equation

$$\mu^2 + \frac{rd}{K\alpha}\mu + \frac{dr}{K\alpha}(K\alpha - d) = 0.$$

Hence E_2 is stable if $\frac{crr_1(K\alpha-d)}{(K\alpha+r)} - d_1 < 0$ which imply the condition $R_{02} < 1$
where $R_{02} = \frac{crr_1(K\alpha-d)}{d_1(K\alpha+r)}$.

The variational matrix of the system (2.1) around the equilibrium point $E_3(\ \widetilde{X},\ \widetilde{Y},\ \widetilde{Z},\ 0\ )$ is

$$V_3 = \begin{bmatrix} m_{11} & m_{12} & m_{13} & m_{14} \\ m_{21} & m_{22} & m_{23} & m_{24} \\ m_{31} & m_{32} & m_{33} & m_{34} \\ m_{41} & m_{42} & m_{43} & m_{44} \end{bmatrix}$$

where

$$m_{11} = -\frac{r}{K}\widetilde{X} < 0,\ m_{12} = -(\frac{r}{K} + \alpha)\widetilde{X} < 0, m_{13} = m_{14} = 0;$$

$$m_{21} = \alpha\widetilde{Y} > 0,\ m_{22} = 0,\ m_{23} = -c\widetilde{Y} < 0,\ m_{24} = -ce\widetilde{Y} < 0;$$

$$m_{31} = 0,\ m_{32} = r_1 c\widetilde{Z} > 0,\ m_{33} = 0,\ m_{34} = r_1 ce\widetilde{Y} - \beta\widetilde{Z};$$

$$m_{41} = m_{42} = m_{43} = 0, m_{44} = \beta\widetilde{Z} - d_2 > 0;$$

It is found that the characteristic roots of the variational matrix V_2 at E_2 are
$\frac{K\alpha\beta}{c}\left\{1 - \frac{d_1(r+K\alpha)}{Krr_1c} - \frac{d}{K\alpha}\right\} - d_2$ and the roots of the equation

$$y^3 + Q_1 y^2 + Q_2 y + Q_3 = 0$$

where $Q_1 = -m_{11} > 0$ (since $m_{11} < 0$) $Q_2 = -m_{32}m_{23} + m_{12}m_{21} > 0$ (since $m_{23} < 0, m_{12} < 0$); $Q_3 = m_{11}m_{32}m_{23} > 0$ (since $m_{11} < 0, m_{23} < 0$) and $Q_1Q_2 - Q_3 = m_{11}m_{12}m_{21} > 0$
Hence the Routh -Hurwitz criteria are satiesfied. So, E_3 is locally asymptotically stable if
$\frac{K\alpha\beta}{c}\left\{1 - \frac{d_1(r+K\alpha)}{Krr_1c} - \frac{d}{K\alpha}\right\} - d_2 < 0$
which imply the condition $R_{03} < 1$.
where $R_{03} = \frac{K\alpha\beta}{cd_2}(1 - R_1)$.

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An SEIT Epidemic Model with new modulated Saturated Incidence and time Delay

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Abstract. In this paper, we have considered an SEIT epidemic model with new modulated saturated incidence and discrete time delay. From our motivation first we prepare the mathematical model and the effect of time delay is investigated. Our main consideration that the disease is transmitted only by contact with the infected individuals of the same species. The model convey two equilibria namely, a disease free equilibrium and an endemic equilibrium. With the help of basic reproductive number we discuss the transmission dynamics of the disease. Analyzing the model we have found out the mathematical results like invariant region boundedness and local stability of both the delayed and non delayed system. From our details study we can say that bifurcation occurs due to discrete time delay τ. Also we are trying to examine what will happen if we increase the discrete time delay. The stability of the endemic equilibrium point breaks of the delay system and Hopf bifurcation occurs. When the bifurcation parameter passes through the critical value, E^* falls its stability and a family of periodic solutions bifurcate from E^*. With the support of competent value of the parameter we have calculated the value of basic reproductive number. The proposed model has been solved numerically and it verify the analytical results.

1 Introduction

At that conjuncture time all over the World population were faced various kind of life killing diseases, such as Ebola, HIV/AIDS, Breast cancer, influenza, rubella, dengue and chicken pox. Basically such type of disease is transmit through horizontally and vertically. Horizontal transmission means the disease spread by contact and vertical transmission means by birth that is pregnant mother to new born child. All of you are know that some of the infectious disease are extent by direct touch with the

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same species and some of them are transmitted by viral agent, such as measles, rubella and dengue. In this paper we like take up only those diseases which spread only by horizontal transmission. Mathematical point of view the model which is related to epidemiology gives an idea to understanding the mechanisms of spread and control of the contagious disease. In 1927 Kermack-Mckendrick introduce a SIR epidemic model and he used the mass action term in the form $\frac{\beta SI}{N}$, where β, S and I denotes the transmission rate, susceptible population, infected population and N is the total population. So many epidemiological models have been developed with or without delay [1],[2],[3],[4],[5] and [6]. So many works have been done already on the disease transmission [7]. Innumerable papers have been published through the use of non-linear incidence rate [8],[9],[10],[11],[12],[13], [14],[15] and [16].

The above mention incidence rate plays a vital role to formulate a mathematical model. If the number of infected individuals is increase then the infection force is decrease. According to biological aspect there are so many reasons to initiate the time delays in mathematical model of disease transmission. Various paper have been published with discrete time delay and non-linear incidence rate [17] and [18]. Following different biological mechanisms came out from the various time delays epidemiological models; $a)$. Delay due to temporary immunity: Some kind of infections take steps or time lag to recovered individuals with a short or long immunity against reinfection. Some type of diseases provide life long immunity [19] and [20]. $b)$. Delay caused by the latency in a vector: We know that various infectious diseases are spread from one host to another by vector that is through agent. Malaria, dengue fever is such types of disease which spread by vector and the disease take some time to spread. According to various organisations report all the countries of the World the transmission rate of such types of disease is very high. In this paper we proposed a delayed SEIT epidemic model with new modulated saturated incidence rate i.e. $\frac{\beta SI}{1+\alpha_1 I+\alpha_2 I^2}$ and the disease is transmitted horizontally that is direct or indirect contact. The direct contact means touching, bitting, licking and indirect contact means vector agent. In this model we would extend the non-linear incidence rate of the model [21], [22] and [23]. The main focuses of my study on a SEIT epidemic model of disease transmission with new modulated saturated incidence rate and the effect of time delay. It is very essentials to discus all the parameters of the proposed mathematical model. The such types of disease transmission totally depends on the basic reproduction number R_0. The disease will persists or fade out it totally depends on the basic reproductive number R_0. If R_0 lies between zero and one then the disease dies out and it will be persists when R_0 is greater than one. Analyze the stability of our proposed model and it solved numerically which justify the analytical result.

2 The mathematical model

Here we consider a delay SEIT epidemic model with new modulated saturated incidence. For our study suppose that the total population is constant and it is denoted by N(t). We divide the total population into four subclasses such that susceptible class S(t), exposed class E(t), infected class I(t) and treatment class or recovery class T(t). Consequent to our assumption the disease is spread only by contact.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta_1 f(S(t-\tau)I(t-\tau)) - \beta_2 f(S(t-\tau)E(t-\tau)) - \mu S + \gamma T,\\
\frac{dE}{dt} &= \beta_1 f(S(t-\tau)I(t-\tau)) + \beta_2 S(t-\tau)E(t-\tau) - \varepsilon E - \mu E,\\
\frac{dI}{dt} &= \varepsilon E - \theta I - \mu I,\\
\frac{dT}{dt} &= \theta I - \gamma T - \mu T.
\end{aligned} \tag{2.1}$$

where, $f\{S(t-\tau),I(t-\tau)\} = \frac{S(t-\tau)I(t-\tau)}{1+\alpha_1 I(t-\tau)+\alpha_2 I^2(t-\tau)}$

$f\{S(t-\tau),E(t-\tau)\} = \frac{S(t-\tau)E(t-\tau)}{1+\alpha_1 I(t-\tau)+\alpha_2 I^2(t-\tau)}$

β_1 is the per capita contact rate between susceptible class and infected class, β_2 is the per capita contact rate between infected class and exposed class, α_1 and α_2 are two positive constants, μ is the mortality rate of all adults class, $\tau > 0$ is the latent time delay and it make a susceptible infected after interaction with exposed class and infected class. ε is the rate by which the exposed classes are recruited to infected class. Λ the rate at which the susceptible classes are recruited. θ the rate at which the infected class are recruited to treatment class. γ is the at which the individuals leave the compartment T and enter the compartment S. We consider a new modulated saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 I+\alpha_2 I^2}$ which indicates that infection force are decreases when the number of infective are increasing. Let $\Psi(\vartheta) = [S(\vartheta),E(\vartheta),I(\vartheta),T(\vartheta)]$, $\vartheta \in [-\tau,0]$, with the norm of Ψ defined as $\|\Psi\| = \sup_{-\tau\le\vartheta\le 0} |\Psi(\vartheta)|$ where the symbol $||.||$ is denote the norm in $\in \mathbf{R}_+{}^4$.

Therefore the initial condition for system of equation (2.1) is $\Psi(\vartheta) = [S(\vartheta),E(\vartheta),I(\vartheta),T(\vartheta)]$, where $S(\vartheta) \ge 0$, $E(\vartheta) \ge 0$, $I(\vartheta) \ge 0$ and $T(\vartheta) \ge 0$, for all $\vartheta \in [-\tau,0]$. Equation (2.1) subject to the above assumption has a unique solution [7].

3 Invariant region

Lemma 1. All the non-negative solutions of the system (2.1) starting in R_+^4 are bounded and eventually enter the attracting set $\Psi = \left\{(S,E,I,T) \in R_+^4 : 0 \le S+E+I+T \le \frac{\Lambda}{\mu}\right\}$.

Proof: Let all parameters of the system (2.1) are non negative. Here $N = S+E+I+T$ $\frac{dN}{dt} = \Lambda - \mu(S+E+I+T)$ or, $\frac{dN}{dt} = \Lambda - \mu N$. Since $N(t) \ge 0$ on $[-\tau,0]$ by consideration, $N(t) \ge 0$ for all $t \ge 0$. Hence $\lim_{t\to\infty} \sup (S+E+I+T) \le \frac{\Lambda}{\mu}$. Hence all the positive solutions of the model (2.1) starting in R_+^4 are bounded and eventually enter the invariant and compact set
$\Psi = \left\{(S,E,I,T) \in R_+^4\ 0 \le S+E+I+T \le \frac{\Lambda}{\mu}\right\}$.

Lemma 2. All the solutions of the system (2.1) is positive $\forall\, t > 0$ with the help of initials conditions $S(0) \ge 0$, $E(0) \ge 0$, $I(0) \ge 0$, $T(0) \ge 0$, and $t \in [-\tau,0]$.

Proof: From the first equation of equation (2.1), we have
$\frac{dS}{dt} = \Lambda - \frac{\beta_1 SIe^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} - \frac{\beta_2 SEe^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} - \mu S + \gamma T$ $\frac{dS}{dt} = \Lambda + \gamma T - \left[\frac{\beta_1 Ie^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} + \frac{\beta_2 Ee^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} + \mu\right] S$
$\frac{dS}{dt} \ge -\left[\frac{\beta_1 Ie^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} + \frac{\beta_2 Ee^{-\lambda\tau}}{1+\alpha_1 I+\alpha_2 I^2} + \mu\right] S$
$S(t) > S(0)e^{-\int\left\{\frac{\beta_1 I(\xi)e^{-\lambda\tau}}{1+\alpha_1 I(\xi)+\alpha_2 I^2(\xi)} + \frac{\beta_2 E(\xi)e^{-\lambda\tau}}{1+\alpha_1 I(\xi)+\alpha_2 I^2(\xi)} + \mu\right\}d\xi} \ge 0$. Similarly from the second, third and fourth equation of the system (2.1) we can prove that

$E(t) \geq \frac{E(0)}{e^{(\mu+\varepsilon)t}} \geq 0$, $I(t) \geq \frac{I(0)}{e^{(\mu+\theta)t}} \geq 0$ and $T(t) \geq \frac{T(0)}{e^{(\mu+\gamma)t}} \geq 0$. Therefore all solutions of the system (2.1) are positive $\forall\, t \geq 0$ in the region Ψ.

4 Qualitative Analysis of the Model (2.1)

The system (2.1) consists two positive equilibria, namely
(i) the disease free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$,
(ii) the endemic equilibrium $E^* = (S^*, E^*, I^*, T^*)$, where
$S^* = \frac{(\mu+\theta)(\mu+\varepsilon)(1+\alpha_1 I^*+\alpha_2 I^{*2})}{e^{-\lambda\tau}[\beta_1\varepsilon+\beta_2(\mu+\theta)]} = \frac{\Lambda(1+\alpha_1 I^*+\alpha_2 I^{*2})}{\mu R_0}$, $E^* = \frac{(\mu+\theta)I^*}{\varepsilon}$, $T^* = \frac{\theta I^*}{(\mu+\gamma)}$ and I^* is given as a root of the quadratic equation

$$A_1 I^{*2} + B_1 I^* + C_1 = 0 \tag{4.1}$$

, where $A_1 = \alpha_2\mu\varepsilon(\mu+\theta)(\mu+\varepsilon)(\mu+\gamma) > 0$,
$B_1 = \alpha_1\mu\varepsilon(\mu+\theta)(\mu+\varepsilon)(\mu+\gamma) + \beta_1\varepsilon(\mu+\theta)(\mu+\varepsilon)(\mu+\gamma) + \beta_2(\mu+\theta) - \gamma\varepsilon\theta e^{-\lambda\tau}[\beta_1\varepsilon+\beta_2(\mu+\theta)]$,
$C_1 = \mu\varepsilon(\mu+\theta)(\mu+\varepsilon)(\mu+\gamma)(1-R_0) > 0$,
where R_0 is a basic reproduction number given as follows

$$R_0 = \frac{\Lambda e^{-\lambda\tau}[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu(\mu+\theta)(\mu+\varepsilon)}.$$

When $R_0 > 1$, then $C_1 < 0$. The above result ensure that while $R_0 > 1$ the above quadratic equation has a positive root. Therefore $I^* = \frac{-B_1 \pm \sqrt{(B_1^2 - 4A_1C_1)}}{2A_1}$.

4.1 Disease free-equilibrium E_0

Theorem 1. The disease free equilibrium E_0 is locally asymptotically stable if $R_{01} < 1$, $(\mu+\varepsilon+\theta) > \frac{\Lambda\beta_2}{\mu}$ and $\tau \geq 0$.

Proof. The variational matrix of the model system (2.1) around the disease free equilibrium point E_0 is given by

$$V_0 = \begin{pmatrix} -\mu & -\frac{\Lambda\beta_2 e^{-\lambda\tau}}{\mu} & -\frac{\Lambda\beta_1 e^{-\lambda\tau}}{\mu} & \gamma \\ 0 & -\frac{\Lambda\beta_2 e^{-\lambda\tau}}{\mu} - (\mu+\varepsilon) & \frac{\Lambda\beta_1 e^{-\lambda\tau}}{\mu} & 0 \\ 0 & \varepsilon & -(\mu+\theta) & 0 \\ 0 & 0 & \theta & -(\mu+\gamma) \end{pmatrix}$$

The characteristic equation is
$(\mu+\lambda)(\mu+\gamma+\lambda)\left[\lambda^2 + \left[(\mu+\varepsilon)+(\mu+\theta) - \frac{\Lambda\beta_2 e^{-\lambda\tau}}{\mu}\right]\lambda + (\mu+\varepsilon)(\mu+\theta) - \frac{\Lambda e^{-\lambda\tau}[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu}\right] = 0$
Two eigenvalues are $-\mu$, $-(\mu+\gamma)$ and the other two eigenvalues of the quadratic equation

$$\lambda^2 + \left[(\mu+\varepsilon)+(\mu+\theta) - \frac{\Lambda\beta_2 e^{-\lambda\tau}}{\mu}\right]\lambda + (\mu+\varepsilon)(\mu+\theta) - \frac{\Lambda e^{-\lambda\tau}[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu} = 0 \tag{4.2}$$

For $\tau = 0$, equation (4.2) becomes

$$\lambda^2 + \left[(\mu+\varepsilon)+(\mu+\theta) - \frac{\Lambda\beta_2}{\mu}\right]\lambda + (\mu+\varepsilon)(\mu+\theta) - \frac{\Lambda[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu} = 0, \tag{4.3}$$

$$i.e.\ \ P_1\lambda^2 + Q_1\lambda + R_1 = 0, \tag{4.4}$$

where $P_1 = 1$, $Q_1 = [(\mu+\varepsilon)+(\mu+\theta)-\frac{\Lambda\beta_2}{\mu}] > 0$, if $(\mu+\varepsilon+\theta) > \frac{\Lambda\beta_2}{\mu}$ and $R_1 = (\mu+\varepsilon)(\mu+\theta)(1-R_{01}) > 0$, if $R_{01} < 1$, where $R_{01} = \frac{\Lambda[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu(\mu+\theta)(\mu+\varepsilon)}$

Hence all the roots of the quadratic equation (4.4) are negative if $R_{01} < 1$ and $(\mu+\varepsilon+\theta) > \frac{\Lambda\beta_2}{\mu}$. Therefore when $R_{01} < 1$, $(\mu+\varepsilon+\theta) > \frac{\Lambda\beta_2}{\mu}$, and $\tau = 0$ then the characteristic equation (4.2) has four negative eigenvalues and the disease free equilibrium E_0 is locally asymptotically stable.

Now suppose $\tau \neq 0$, i. e. $\tau > 0$ by Rouche's theorem [24], Theorem 9.17.4, it follows that if instability occurs for a particular value of the delay τ, a characteristic root of (4.2) must intersect the imaginary axis. Let $\lambda = \varphi i$, $\varphi > 0$ is a roots of the above quadratic equation (4.2). Putting the value $\lambda = \varphi i$ into the equation (4.2) we get

$$\begin{aligned} -\varphi^2 + i\left[\varphi(\mu+\theta)(\mu+\varepsilon) - \frac{\varphi\beta_2\Lambda\cos\varphi\tau}{\mu} + \frac{\Lambda\sin\varphi\tau[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu}\right] \\ - \frac{\varphi\Lambda\beta_2\sin\varphi\tau}{\mu} - \frac{\Lambda\cos\varphi\tau[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu} + (\mu+\theta)(\mu+\varepsilon) = 0 \end{aligned} \tag{4.5}$$

Separating the real and imaginary part, gives

$$-\varphi^2 + (\mu+\theta)(\mu+\varepsilon) = \frac{\varphi\Lambda\beta_2\sin\varphi\tau}{\mu} + \frac{\Lambda\cos\varphi\tau[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu} \tag{4.6}$$

$$\varphi(\mu+\theta)(\mu+\varepsilon) = \frac{\varphi\beta_2\Lambda\cos\varphi\tau}{\mu} - \frac{\Lambda\sin\varphi\tau[\beta_1\varepsilon+\beta_2(\mu+\theta)]}{\mu} \tag{4.7}$$

Squaring and adding equation (4.6) and (4.7) we have

$$\varphi^4 + \varphi^2\left[(\mu+\theta)^2 + (\mu+\varepsilon)^2 - \frac{\beta^2\Lambda^2}{\mu^2}\right] + (\mu+\theta)^2(\mu+\varepsilon)^2(1-R_{01}^2) = 0 \tag{4.8}$$

It follows from Routh-Hurwitz Criteria that the roots of the equation (4.8) all have negative real parts if $R_{01} < 1$ and when $\tau > 0$. Hence we say that the disease free equilibrium point E_0 of the given system is locally asymptotically stable if and only if $\tau \geq 0$, $R_{01} < 1$ and satisfies the condition $(\mu+\varepsilon+\theta) > \frac{\Lambda\beta_2}{\mu}$.

4.2 Local Stability of Endemic Equilibrium :

Theorem 2. For $R_0 > 1$, the endemic equilibrium point E^* is locally asymptotically stable, if and only if $\tau \geq 0$.

Proof. The Jacobian matrix V^* about the endemic equilibrium point E^* is given by,

$$V^* = \begin{pmatrix} -\beta_1 I^* a e^{-\lambda\tau} - \beta_2 a E^* e^{-\lambda\tau} - \mu & -\beta_2 a S^* e^{-\lambda\tau} & -\beta_1 S^* a^2 e^{-\lambda\tau} & \gamma \\ \beta_1 a I^* e^{-\lambda\tau} + \beta_2 a E^* e^{-\lambda\tau} & \beta_2 a S^* e^{-\lambda\tau} & \beta_1 S^* a^2 e^{-\lambda\tau} & 0 \\ 0 & \varepsilon & -(\mu+\theta) & 0 \\ 0 & 0 & \theta & -(\mu+\gamma) \end{pmatrix}$$

The characteristic equation is

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 + e^{-\lambda\tau}(b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4), where \tag{4.9}$$

$a_1 = \mu + (\mu+\gamma) + (\mu+\varepsilon) + (\mu+\theta)$,
$a_2 = \mu(\mu+\gamma) + \mu(\mu+\varepsilon) + \mu(\mu+\theta) + (\mu+\varepsilon)(\mu+\gamma) + (\mu+\theta)(\mu+\gamma) + (\mu+\varepsilon)(\mu+\theta)$,
$a_3 = \mu(\mu+\gamma)(\mu+\varepsilon) + \mu(\mu+\gamma)(\mu+\theta) + \mu(\mu+\theta)(\mu+\varepsilon) + (\mu+\gamma)(\mu+\theta)(\mu+\varepsilon)$,
$a_4 = \mu(\mu+\gamma)(\mu+\theta)(\mu+\varepsilon) + a\beta_2 E(\mu+\gamma)(\mu+\theta)(\mu+\varepsilon)$,
$b_1 = a\beta_1 I^* + a\beta_2 E^* - a\beta_2 S^*$,
$b_2 = a\beta_2 E^*(\mu+\theta) + a\beta_2 E^*(\mu+\gamma) + a\beta_2 E^*(\mu+\varepsilon) + a\beta_1 I^*(\mu+\theta) + a\beta_1 I^*(\mu+\gamma) + a\beta_1 I^*(\mu+\varepsilon) - a\beta_2\mu S^* - a\beta_2(\mu+\gamma)S^* - a\beta_2(\mu+\theta)S^* - a^2\beta_1 S^* E^*$,
$b_3 = a\beta_2 E^*(\mu+\theta)(\mu+\varepsilon) + a\beta_1 I^*(\mu+\gamma)(\mu+\theta) + a\beta_1 I^*(\mu+\varepsilon)(\mu+\theta) + a\beta_1 I^*(\mu+\gamma)(\mu+\varepsilon) + a\beta_2 E^*(\mu+\gamma)(\mu+\varepsilon) + a\beta_2 E^*(\mu+\gamma)(\mu+\theta) - a\beta_2 S^*\mu(\mu+\gamma) - a\beta_2 S^*\mu(\mu+\theta) - a\beta_2 S^*(\mu+\theta)(\mu+\gamma) - a^2\beta_1 S^*\mu\varepsilon - a^2\beta_1 S^*(\mu+\gamma)\varepsilon$,
$b_4 = a\beta_1(\mu+\varepsilon)(\mu+\theta)(\mu+\gamma)I^* - a\beta_2 S^*\mu(\mu+\gamma)(\mu+\theta) - a\beta_1 I^*\gamma\varepsilon\theta - a\beta_2 E^*\gamma\varepsilon\theta - a^2\beta_1 S^*\varepsilon\mu(\mu+\gamma)$,
where $a = \frac{1}{1+\alpha_1 I^* + \alpha_2 I^{*2}}$.
For $\tau = 0$, equation (4.9) becomes

$$\lambda^4 + c_1\lambda^3 + c_2\lambda^2 + c_3\lambda + c_4 = 0, \ where \tag{4.10}$$

$c_1 = a_1 + b_1 = \mu + (\mu+\gamma) + (\mu+\varepsilon) + (\mu+\theta) + a\beta_1 I^* + a\beta_2 E^* - a\beta_2 S^*$,
$c_2 = a_2 + b_2 = \mu(\mu+\gamma) + \mu(\mu+\varepsilon) + \mu(\mu+\theta) + (\mu+\varepsilon)(\mu+\gamma) + (\mu+\theta)(\mu+\gamma) + (\mu+\varepsilon)(\mu+\theta) + a\beta_2 E^*(\mu+\theta) + a\beta_2 E^*(\mu+\gamma) + a\beta_2 E^*(\mu+\varepsilon) + a\beta_1 I^*(\mu+\theta) + a\beta_1 I^*(\mu+\gamma) + a\beta_1 I^*(\mu+\varepsilon) - a\beta_2\mu S^* - a\beta_2(\mu+\gamma)S^* - a\beta_2(\mu+\theta)S^* - a^2\beta_1 S^* E^*$,
$c_3 = a_3 + b_3 = \mu(\mu+\gamma)(\mu+\varepsilon) + \mu(\mu+\gamma)(\mu+\theta) + \mu(\mu+\theta)(\mu+\varepsilon) + (\mu+\gamma)(\mu+\theta)(\mu+\varepsilon) + a\beta_2 E^*(\mu+\theta)(\mu+\varepsilon) + a\beta_1 I^*(\mu+\gamma)(\mu+\theta) + a\beta_1 I^*(\mu+\varepsilon)(\mu+\theta) + a\beta_1 I^*(\mu+\gamma)(\mu+\varepsilon) + a\beta_2 E^*(\mu+\gamma)(\mu+\varepsilon) + a\beta_2 E^*(\mu+\gamma)(\mu+\theta) - a\beta_2 S^*\mu(\mu+\gamma) - a\beta_2 S^*\mu(\mu+\theta) - a\beta_2 S^*(\mu+\theta)(\mu+\gamma) - a^2\beta_1 S^*\mu\varepsilon - a^2\beta_1 S^*(\mu+\gamma)\varepsilon$,
and $c_4 = a_4 + b_4 = \mu(\mu+\gamma)(\mu+\theta)(\mu+\varepsilon) + a\beta_2 E(\mu+\gamma)(\mu+\theta)(\mu+\varepsilon) + a\beta_1(\mu+\varepsilon)(\mu+\theta)(\mu+\gamma)I^* - a\beta_2 S^*\mu(\mu+\gamma)(\mu+\theta) - a\beta_1 I^*\gamma\varepsilon\theta - a\beta_2 E^*\gamma\varepsilon\theta - a^2\beta_1 S^*\varepsilon\mu(\mu+\gamma)$.
Therefore, with the help of Routh-Hurwitz criterion, all the roots of the equation (4.10) have negative real parts if $c_i > 0,\ i = 1,\ 2,\ 3,\ 4,\ c_1c_2 - c_3 > 0$ and $c_3(c_1c_2 - c_3) - c_1^2c_4 > 0$. So we can conclude that E^* is locally asymptotically stable for $\tau = 0$. Now putting $\lambda = \varphi' i'$, $\varphi' > 0$ into the equation (4.9) and separating the real and imaginary parts we get

$$\varphi'^4 i'^4 + a_1\varphi'^3 i'^3 + a_2\varphi'^2 i'^2 + a_3\varphi' i' + a_4 + (\cos\varphi'\tau - i'\sin\varphi'\tau)(b_1\varphi'^3 i'^3 + b_2\varphi'^2 i'^2 + b_3\varphi' i' + b_4) = 0 \tag{4.11}$$

$$-b_2\varphi'^2\cos\varphi'\tau - b_1\varphi'^3\sin\varphi'\tau + b_4\cos\varphi'\tau + b_3\varphi'\sin\varphi'\tau = -\varphi'^4 + a_2\varphi'^2 - a_4 \tag{4.12}$$

$$b_1\varphi'^3\cos\varphi'\tau - b_2\varphi'^2\sin\varphi'\tau - b_3\varphi'\cos\varphi'\tau + b_4\sin\varphi'\tau = a_3\varphi' - a_1\varphi'^3 \tag{4.13}$$

Squaring and adding equation no (4.12) and (4.13), we have
$[-\varphi'^4 + a_2\varphi'^2 - a_4]^2 + [a_3\varphi' - a_1\varphi'^3]^2 = [-b_2\varphi'^2\cos\varphi'\tau - b_1\varphi'^3\sin\varphi'\tau + b_4\cos\varphi'\tau + b_3\varphi'\sin\varphi'\tau]^2 + [b_1\varphi'^3\cos\varphi'\tau - b_2\varphi'^2\sin\varphi'\tau - b_3\varphi'\cos\varphi'\tau + b_4\sin\varphi'\tau]^2$
$\varphi'^8 + \varphi'^6(a_1^2 - 2a_2 - b_1^2) + \varphi'^4(a_2^2 + 2a_4 - 2a_1a_3 - b_2^2 + 2b_1b_3) + \varphi'^2(a_3^2 - 2a_2a_4 - b_3^2 + 2b_2b_4)$

$$+a_4^2 - b_4^2 = 0 \tag{4.14}$$

Let $u_1 = \varphi'^2$, then equation (4.14) can be rewritten as

$$u_1^4+\rho_0 u_1^3+\rho_1 u_1^2+\rho_2 u_1+\rho_3=0, where \tag{4.15}$$

$\rho_0=(a_1^2-2a_2-b_1^2)$, $\rho_1=(a_2^2+2a_4-2a_1a_3-b_2^2+2b_1b_3)$, $\rho_2=(a_3^2-2a_2a_4-b_3^2+2b_2b_4)$ and $\rho_3=a_4^2-b_4^2$. Therefore, by using the Routh-Hurwitz criterion, all the roots of the equation (4.15) have negative real parts if $\rho_i>0,\ i=1,\ 2,\ 3,\ 4$, $\rho_0\rho_1-\rho_2>0$ and $\rho_2(\rho_0\rho_1-\rho_2)-\rho_0^2\rho_3>0$. Therefore we can notify that the endemic equilibrium point E^* is locally asymptotically stable for $\tau\geq 0$.

4.3 Existence of Hopf bifurcation

From the above analysis for the local stability of E^*, it is noted that the equation (4.15) has a positive root φ_0'. This implies that the equation (4.15) has a pair of imaginary roots $\pm i\varphi_0'$. Let $\lambda=u(\tau)+i\varphi'(\tau)$ be the eigen values of equation (4.9) such that $u(\tau_0)=0$, $\varphi'(\tau_0)=0$. From equation (4.12) and (4.13) we get the corresponding $\tau_k>0$ such that the characteristic equation (4.9) has a pair of imaginary roots.

$$\tau_k=\frac{1}{\varphi_0'}\cos^{-1}\left\{\frac{(b_2-a_1b_1)\varphi_0'^6-(a_2b_2-a_3b_1-a_1b_3+b_4)\varphi_0'^4+(a_2b_4+a_4b_2-a_3b_3)\varphi_0'^2-a_4b_4}{b_1^2\varphi_0'^6+(b_2^2-2b_1b_3)\varphi_0'^4+(b_3^2-2b_2b_4)\varphi_0'^2+b_4^2}\right\}+\frac{2k\pi}{\varphi_0'},$$

$k=0,1,2,3,.....$, assuming $a_i,\ b_i\ (i=1,\ 2,\ 3,\ 4)$ to be bounded function of τ if the following condition is satisfied $\frac{d(Re\lambda)}{d\tau}>0,\ at\ \tau=\tau_k$

This will signify that there exists at least one eigenvalue with positive real part for $\tau>\tau_k$ and for this root of characteristic equation (4.9) crosses the imaginary axis from the left to the right as τ continuously varies from a numberless than τ_k to one greater than τ_k. The real part of $\lambda(\tau)$ becomes positive when $\tau>\tau_0$ by continuity and the steady state becomes unstable. Since the loss of stability corresponds to a root $\lambda(\tau)=i\varphi'(\tau)$ of the characteristic equation, there are solutions of the model heaving like the real part of $e^{\varphi'(\tau)i}$, that is there are periodic solutions, [25]. This is exactly what happens according to the Hopf bifurcation theorem, which says that while roots of the characteristic polynomial cross the imaginary axis a stable periodic orbit arise [26] and [27].

5 Sensitivity Analysis on R_{01}

In our propose model we have considered only such types of disease which is only spread by contact. For this concern we have some responsibilities to find out a desirable way to decrease the disease transmission. We are trying to observe those parameters, which are responsible for increase the disease transmission. Our concentration to carry out the sensitivity of those parameters formerly which have been given in Table:2 on R_{01}. In epidemiology basically, disease transmission and control are entirely depends on the basic reproduction number R_{01}. First we investigate the sensitivity of R_{01} with respect to the parameters of the without delay system by the method of [28], using the formula,

$$S_{v_j}^{R_{01}}=\frac{\partial R_{01}}{\partial v_j}\times\frac{v_j}{R_{01}}$$

Table1: Sensitivity indicates of R_{01} with respect to the parameters

Parameter v_j	$\wedge$	β_1	β_2	μ	θ	ε
Value	1.3	0.001	0.2	0.001	0.03	0.12
$S_{v_j}^{R_{01}}$	1	0.01899	0.98118	−0.01009	−0.18378	−0.97288

From Table:1 it is clear that, the value of R_{01} will either increase or decrease it depends on the parameters $\wedge$, β_2 and β_1. Therefore if the values of recruitment rate of susceptible population, contact rate of susceptible population with infected and exposed class will increase (decrease) then the values of R_{01} will also increase (decrease). On the other hand μ, θ and ε have inversely proportional relationship with R_{01}. If we increases the value of θ and ε then the value of R_{01} decreases. Numerically it has been shown in Figure 6.

6 Numerical Simulation and Conclusion:

Table2: A set of parameter values

Parameter	*Definition*	*Value*
Λ	Recruitment rate of susceptible class	1.3
β_1	Per capita contact rate between susceptible class and infected class	0.001
β_2	Per capita contact rate between infected class and exposed class	0.2
μ	Mortality rate of all adults class	0.001
α_1	Positive constant	0.001
α_2	Positive constant	0.01
τ	Latent time delay	9.9
ε	Rate by which the exposed classes are recruited to infected class	0.12
θ	Rate at which the infected class are recruited to treatment class	0.03
γ	Treatment rate	0.02

Now we investigate the behavior of the equilibria E_0 and E^* of the system (2.1) using numerical simulation. For such investigation we choose set of parameter values satisfying the existence criteria of E_0 and E^*. The values of different parameters are given in the following table: With this set of parameters values we show that the endemic equilibrium point E^* of the without delay system is stable and with delay system is unstable (see Figure 1) If we decrease the value of delay time from 9.9 to 9.0 then we have Figure 2, which indicates that the endemic equilibrium point of both the system is stable. Figure 3 shows that when we decrease the value of θ from 0.03 to 0.02 then the endemic equilibrium point of the delay system goes from unstable situation to stable situation and all other parameters values as in Table 2. Figure 4 indicates the bifurcation figure for the parameter τ. Figure 5 displays that if we can decreases the contact rate of susceptible populations with infected populations and exposed populations then the value of basic reproductive number will decreases. Therefore the disease transmission rate will also decrease.

7 Conclusion:

We have introduced and analyzed an SEIT epidemic model to study the effect of discrete time delay. It is observed that the disease free equilibrium point is locally asymptotically stable if $(\mu+\theta+\varepsilon) > \frac{\Lambda\beta_2}{\mu}$ and the basic reproductive number satisfies the relation $0 < R_0 < 1$. While this two relations $R_0 > 1$ and $\tau > 0$ are fulfilled then the endemic equilibrium point is locally asymptotically stable. In our final analysis we have observed that, when we increase discrete delay time then the stable situation of the endemic equilibrium point of the delayed model switches and Hopf bifurcation occurs. We have also shown that if we decrease the value of treatment rate of the infected population from 0.03 to 0.02 then the stability behavior of the endemic equilibrium of the delay system is also changed and its goes

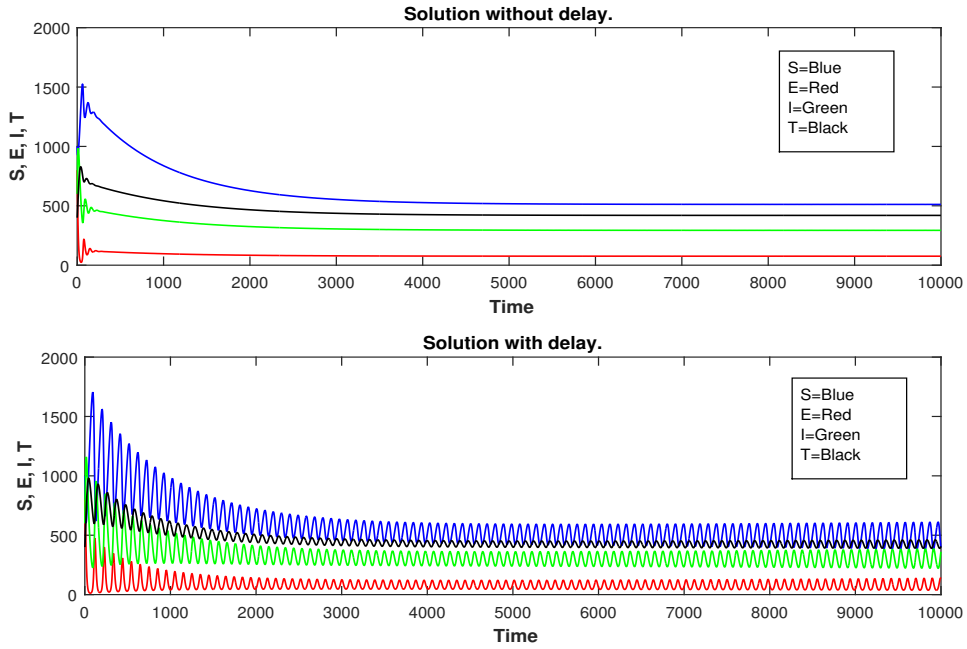

Fig. 1 The endemic equilibrium point E^* of the without delay system is stable and with delay system is unstable.

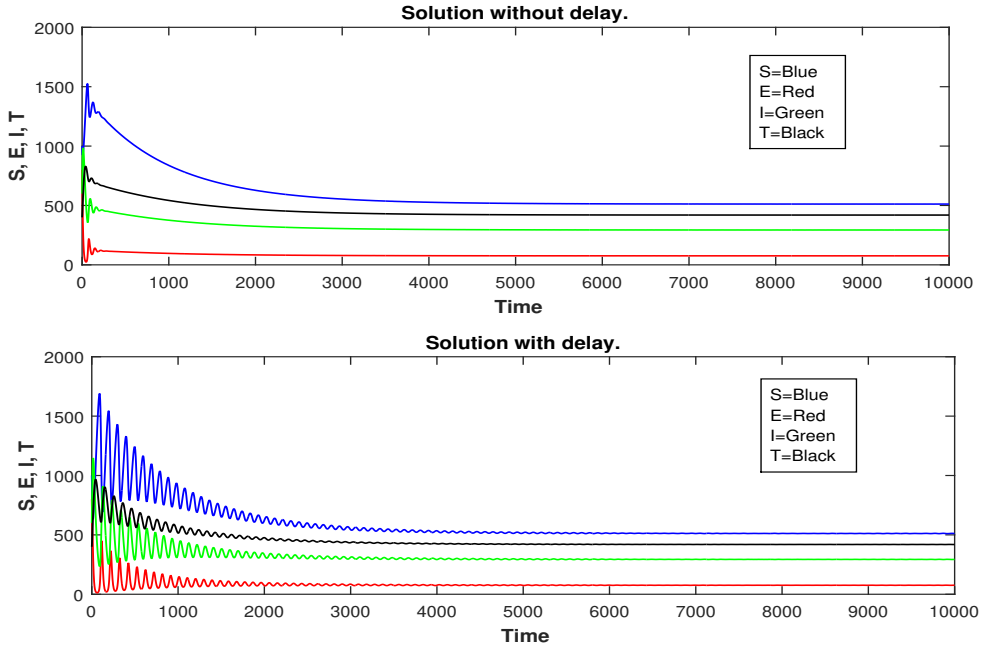

Fig. 2 The equilibrium point E^* of both the system is stable when τ= 9.

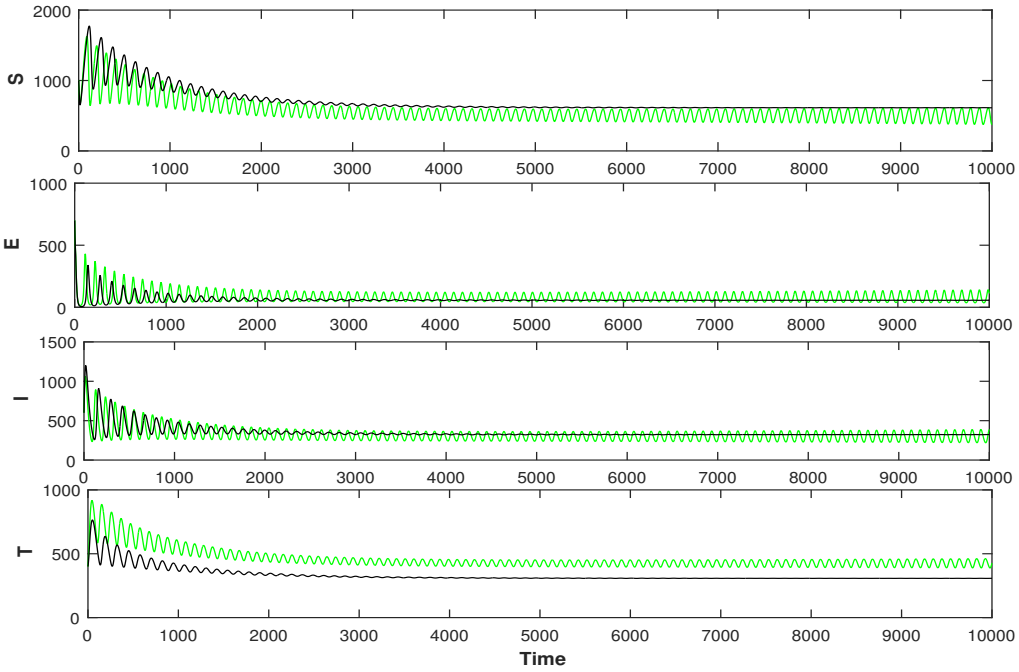

Fig. 3 Endemic equilibrium point of the delay system is stable when θ= 0.02 and unstable when θ= 0.03 with the other parametric values as given in Table 2.

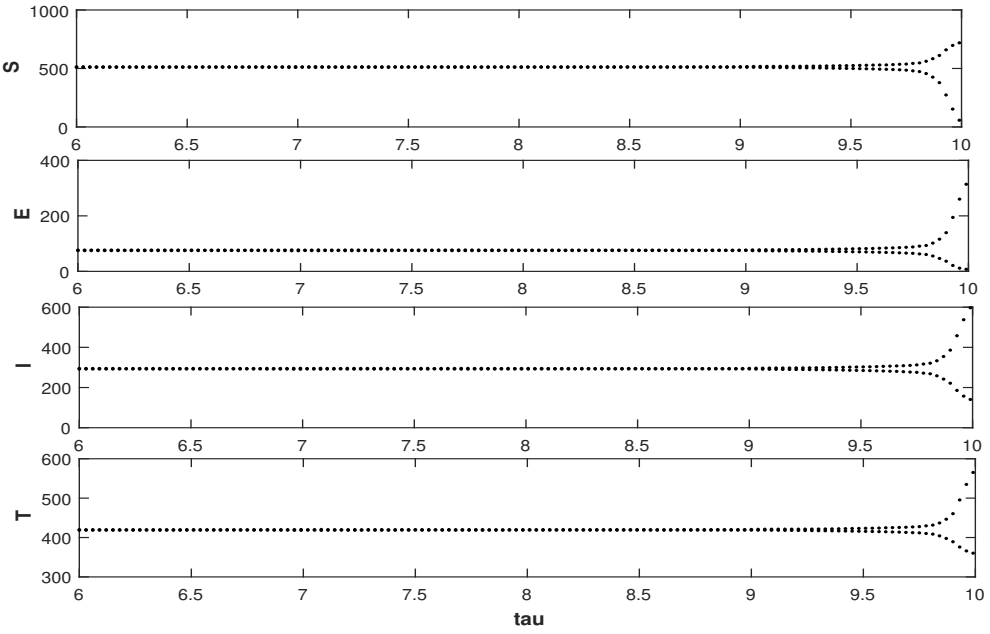

Fig. 4 Bifurcation figure for the parameter τ and all other parameters values as in Table 2.

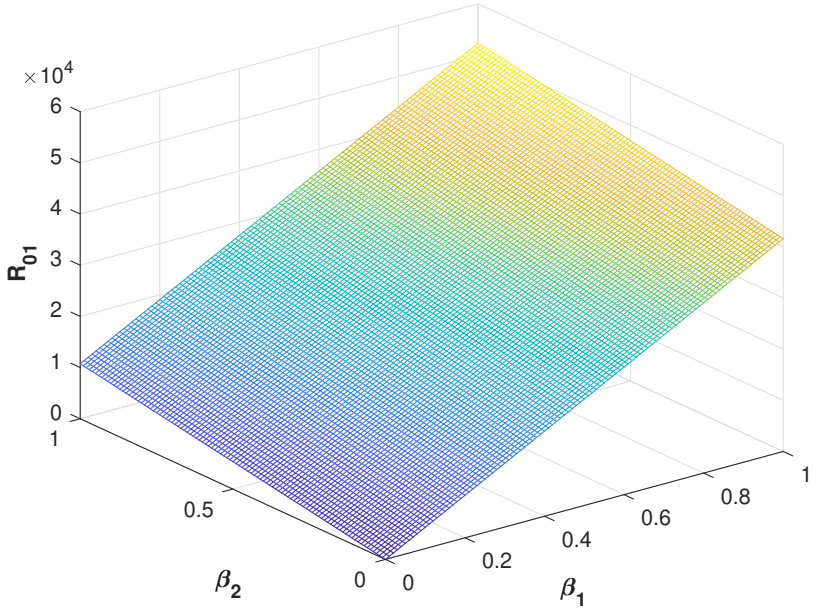

Fig. 5 Relation among β_1, β_2 and R_{01}

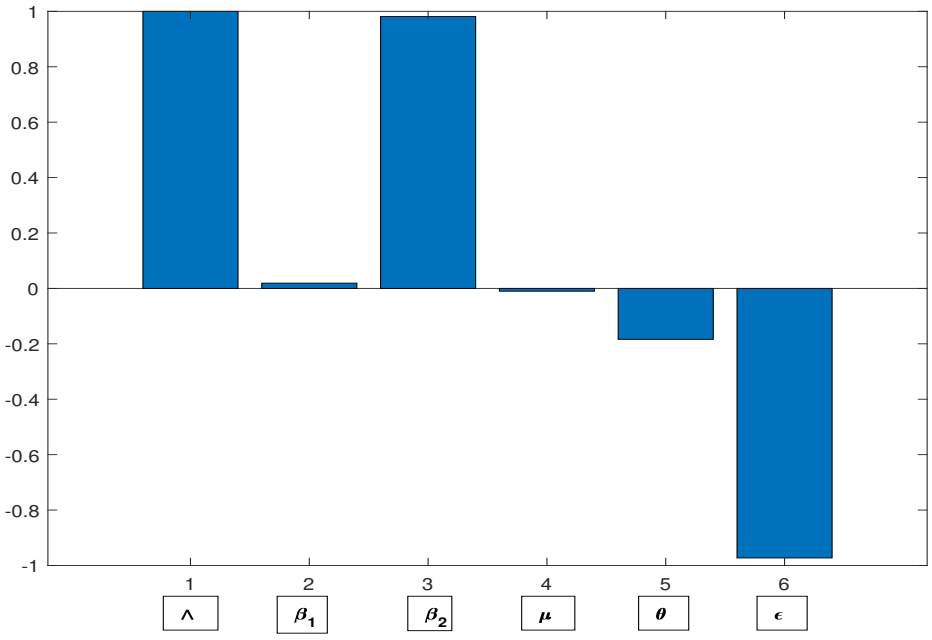

Fig. 6 Sensitivity analysis on R_{01} with respect to the parameters

from unstable to stable. Therefore from our study we must say that if we will increases the value of treatment rate of infected population and the progression rate of infected population then the value of R_{01} will decreases which ensure that the transmission rate of disease will decrease. Our computed analytical outcome also being verified and tested by using numerical technique.

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