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EFFECT OF POLLUTION ON PREY-PREDATOR SYSTEM WITH INFECTED PREDATOR

SUDIPA CHAUHAN, SUMIT KAUR BHATIA*, PREETI CHAUDHARY

Amity Institue of Applied Sciences, Amity University, Noida, India

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Abstract. In this paper, we consider effect of pollution on prey-predator model with infected predator. We have modified the prey-predator model by introducing pollutant in susceptible predator and infected predator population. The pollutant effects both susceptible and infected predator at the same rate. It is assumed that prey population grows with logistic growth rate. Local stability analysis is done for boundary and interior equilibrium point E^*. Further, we have proved that if the rate of infection and the rate of depletion of predator due to pollutant is less than some threshold i.e., $\beta < \beta^*$ and $g < g^*$ then E^* becomes unstable and periodic solutions bifurcates from E^* and thus, Hopf bifurcation occurs. Finally, persistence of the system is obtained and numerical simulations are done in support of our results using MATLAB software.

Keywords: prey-predator; infection; pollution; reproduction number; stability; Hopf-bifurcation; persistence.

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1. Introduction

Lot of research has already been conducted in the field of epidemiology. J. Graunt [1] was the first scientist who measured the causes of death in his book in 1662. He had gone through

*Corresponding author
E-mail address: sumit2212@gmail.com
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various bills that ultimately provided him the list of "causes and number of deaths". Further,according to various scientists, his analysis of various theories is accomplished well, in many epidemiologists in today's era. [2] carried out a technique to study the wide spread of diseases using mathematical modeling in 2001. His work actually led people know the concept of Germ theory. [3] work led to the origin of mathematical model in the area related with various infectious disease transmission process. Since then, lot of research had been conducted in the field of ecology as well as in epidemiology.

[4] were the first to combine the theory of ecology and epidemiology. Further, predator model was formulated where prey population was infected by various diseases. In following time, many theories have been studied with reference to prey-predator model having diseases present in them. Micro parasites are the parasites that reproduce frequently within the host population. They usually tend to have small generation time. Hosts acquire immunity from the infection, usually when recovered from infection for some duration or may be for a life time. The time period of infection is usually short with respect to the total life span of host with some exceptions. Most of viral, protozoan, fungal and bacterial parasites can be divided into category of micro parasites. In today's era, there is zero probability of survival of any species alone.Further, species that are responsible for spread of diseases always depend on others for food and as well as space or are attacked by predators. The relationship of prey-predator helps in balancing numbers of prey-predator. For this purpose, mathematical modeling became an important tool in determining the outbreak and control of various such diseases. Number of prey-predator models were proposed and studied in ecological system but less importance had been given to communicable diseases when two or more are in a ecological relationship.

According to survey, [5], [6], [7], [8], [9], [10], [11], [12], [13], [14], [15] had done a pioneer work in the area of "Effect of predation in the Epidemic". It was seen that, Auger et al. had noticed two kind of behavior of prey predator system when disease is introduced in it, the predator population either becomes extinct or the prey-predator population exist together. When the

predator population exists, then either it will tend to non disease state or disease endemic process. Mathematical epidemiology has not developed its approach in the area of predator control.

Many mathematical models indicates that the accomplished communication of prey-predator tends to get effected when the disease is induced in predator population. Further, micro parasites with their complete life cycles has been observed. Models of Macro parasites usually have a capacity to uncertain the dynamics generally but the synopsis of unstableness does not hold true always in this case. When the disease is induced in a system, it can have opposite effect, as it can stable the vibration of prey-predator dynamics.

It is also found that very less work had been done to combine ecotoxicology and epidemiology. In this [16], authors had studied the combined effect of environmental toxicant and disease on preypredator system. They assumed that the environmental toxicant affects both prey and predator population and the infected prey is assumed to be more vulnerable to the toxicant and predation compared to the sound prey individuals. Thresholds were identified which determined persistence and endemicity of the system. [17] had studied a mathematical model of the simultaneous effects of toxicants and infectious diseases on a competing species system. It was assumed that the competing populations were adversely affected by the toxicant and one of them was vulnerable to an infectious disease. The models were analyzed using stability theory, and conditions for the nonlinear stability of the interior equilibria were obtained.

Keeping in view the above discussions, a mathematical model is proposed with effect of pollutant on a prey-predator system with disease in predator.A mathematical model is proposed in section 4 followed by the existence and local stability analysis of boundary equilibrium points.Biological significance of reproduction numbers is discussed.In section 8, the local stability analysis and bifurcation analysis of interior equilibrium points based on rate of infection and rate of depletion of predator due to pollutant is done. Finally, in the last section, the conclusion is added along with graphs in support of our result.

2. Mathematical Model

Formulation of our model is done based on the following basic assumptions:

1. Let X denotes the prey population density, Y denotes the susceptible predator population density, Z denotes the infected predator population with respect to time.
2. The density of prey population is considered to have logistic growth rate in the absence of predators having intrinsic growth rate r ($r > 0$) and carrying capacity K.

$$\frac{dX}{dT} = rX(1 - \frac{X}{K}) \tag{1}$$

3. When the prey population $X(t)$ combines with susceptible predator population $Y(t)$ and infected predator population $Z(t)$ due to infection then the we can write the evolutionary equation as follows:

$$\frac{dX}{dT} = rX(1 - \frac{X}{K}) - \frac{c_1 X(Y + fZ)}{a_1 + X} \tag{2}$$

where a_1 defines half saturation constant, c_1 is the susceptible predator's rate of predation, $c_1 f$ defines the infected predator's rate of predation.

4. A susceptible predator population Y(t) tends to have negative effect when combined with infected predator population. Let the predators be attacked by parasites only and the disease is transmitted at the rate λ_1 in the predator population. Then, the evolutionary equation can be written as:

$$\frac{dY}{dT} = \frac{m_1 X(Y + fZ)}{a_1 + X} - d_1 Y - \lambda_1 YZ - r_2 YU \tag{3}$$

where m_1 defines the rate of transition factor for susceptible predator, $m_1 f$ is the rate of transition factor for the predators that are infected, d_1 defines non parasitic predator population and r_2 is the rate of decrease of susceptible predator population due to pollutant.

5. The equation for infected predator population is given in the following way:

$$\frac{dZ}{dT} = \lambda_1 YZ - (d_1 + \alpha_1)Z - r_2 ZU \tag{4}$$

where α_1 defines the rate at which infected predator dies out and r_2 defines the reducing rate of infected predator population due to pollutant.

6. If Q is exogenous pollutant in the environment then the equation for the concentration of pollutant becomes:

$$\frac{dP}{dT} = Q - hP \tag{5}$$

where h is the loss rate of toxicant.

7. Let U be the organism pollutant rate in the environment then we can present the equation as:

$$\frac{dU}{dT} = b_1 P + \frac{e_1 \eta \beta}{b_1} - (l_1 + l_2)U \tag{6}$$

where b_1 in the first term is the per unit mass consumption of environment pollutant by organism and in second term, it denotes pollutant consumption rate in the food. η is density of pollutant in the resource, β is food intake rate per unit mass by organism, e_1 is pollutant rate in food per unit mass organism, l_1 and l_2 are the ingestion and depuration rate of pollution in organism.

Thus, our formulated mathematical model is as follows:

$$\frac{dX}{dT} = rX(1 - \frac{X}{K}) - \frac{c_1 X(Y + fZ)}{a_1 + X} \tag{7}$$

$$\frac{dY}{dT} = \frac{m_1 X(Y + fZ)}{a_1 + X} - d_1 Y - \lambda_1 YZ - r_2 YU \tag{8}$$

$$\frac{dZ}{dT} = \lambda_1 YZ - (d_1 + \alpha_1)Z - r_2 ZU \tag{9}$$

$$\frac{dP}{dT} = Q - hP \tag{10}$$

$$\frac{dU}{dT} = b_1 P + \frac{e_1 \eta \beta}{b_1} - (l_1 + l_2)U \tag{11}$$

Now, consider the system :

$$\frac{dx}{dt} = f(x,t) \tag{12}$$

$$\frac{dy}{dt} = g(y) \tag{13}$$

Where f and g are continuous and locally lipschitz in x in R^n and the solutions exists for all positive time. Equation (13) is called asymptotically autonomous with limit equation (12) if $f(t,x) \to g(x)$ as $t \to \infty$uniformly for all x in R^n.Thieme [18] considered the situation in which e is a locally asymptotically stable equilibrium of (13) and ω is the ω-limit set of a forward-bounded solution $x(t)$ of (12). If ω contains a point y_0 such that the solution of (13) with $y(0) = y_0$ converges to e as $t \to \infty$, then $\omega = \{e\}$, that is, $x(t) \to e$ as $t \to \infty$.

Corollary 2.1. If the solutions of the system are bounded and the equilibrium e of the limit system (13) is globally asymptotically stable then any solution x(t) of the system (12) satisfies $x(t) \to e$ as $t \to \infty$.

Since we know from (10)and (11) that, $\limsup_{t\to\infty} P(t) \leq P^* = \frac{Q}{h}$

$$\limsup_{t\to\infty} U(t) \leq U^* = \frac{b_1 P^* + \frac{e_1\eta\beta}{b_1}}{(l_1 + l_2)}$$

Thus using the above corollary in the model, we get the limiting system:

$$\frac{dX}{dT} = rX(1 - \frac{X}{K}) - \frac{c_1 X(Y + fZ)}{a_1 + X} \tag{14}$$

$$\frac{dY}{dT} = \frac{m_1 X(Y + fZ)}{a_1 + X} - d_1 Y - \lambda_1 YZ - r_2 YU^* \tag{15}$$

$$\frac{dZ}{dT} = \lambda_1 YZ - (d_1 + \alpha_1)Z - r_2 ZU^* \tag{16}$$

Now to reduce the parameters, we non-dimensionalize the system of equations by using the following equations:

$x = \frac{X}{K}, y = \frac{Y}{K}, z = \frac{Z}{K}, t = rT$

$$\frac{dx}{dt} = x(1-x) - \frac{ax(y+fz)}{1+bx} \tag{17}$$

$$\frac{dy}{dt} = \frac{cx(y+fz)}{1+bx} - dy - \beta yz - gyU^* \tag{18}$$

$$\frac{dz}{dt} = \beta yz - ez - gzU^* \tag{19}$$

where $b = \frac{K}{a_1}$, $a = \frac{c_1 K}{r a_1}$, $c = \frac{m_1 K}{r a_1}$,$d = \frac{d_1}{r}$, $\beta = \frac{\lambda_1 K}{r}$, $e = \frac{d_1 + \alpha_1}{r}$, $g = \frac{r_2}{r}$
The system is discussed with the initial conditions $x(0) > 0$, $y(0) > 0$, $z(0) > 0$.

3. Existence of Equilibria

We will consider four equilibrium points for our system. The equilibrium point $E_0(0,0,0)$, $E_1(1,0,0)$, disease-free equilibrium $E_2(\bar{x},\bar{y},0)$ where,

$$\bar{x} = \frac{d + gU^*}{c - b(d + gU^*)} \tag{20}$$

provided $c > b(d + gU^*)$ and

$$\bar{y} = \frac{c(c - (b+1)(d + gU^*)}{a((c - b(d + gU^*))^2} \tag{21}$$

provided $c > (b+1)(d + gU^*)$ and $c > b(d + gU^*)$ which further implies $R_1 > 1$ where $R_1 = \frac{c}{(b+1)(d+gU^*)}$ and the interior equilibrium point $E^*(x^*, y^*, z^*)$ is given by the following equation:

$$Q_1 x^2 + Q_2 x + Q_3 = 0 \tag{22}$$

where $Q_1 = (-\frac{c}{a} + \frac{b(e + gU^*)}{af})$
$Q_2 = \frac{c}{a} - \frac{(e + gU^*)}{af}(b-1)$
$Q_3 = \frac{(e + gU^*)}{af\beta}(-\beta + a(1 - df - gU^* f))$
and $y^* = \frac{e + gU^*}{\beta}$
$z^* = \frac{\beta(1 + bx^*)(1 - x^*) - a(e + gU^*)}{af\beta}$

4. Analysis of local stability of boundary equilibria

At any point $E_i(x,y,z)$, the jacobian matrix so formed for our system is given by:

$$\begin{bmatrix} 1-2x-\frac{a(y+fz)}{(1+bx)^2} & \frac{-ax}{1+bx} & \frac{-axf}{1+bx} \\ \frac{c(y+fz)}{(1+bx)^2} & \frac{cx}{1+bx}-d-\beta z-gU^* & \frac{cxf}{1+bx}-\beta y \\ 0 & \beta z & \beta y-e-gU^* \end{bmatrix} \tag{23}$$

The jacobian corresponding to $E_0(0,0,0)$ is:

$$\begin{bmatrix} 1 & 0 & 0 \\ 0 & -d-\beta-gU^* & 0 \\ 0 & 0 & -e-gU^* \end{bmatrix} \tag{24}$$

The corresponding eigen values with respect to the Jacobian are:

$\lambda_1 = 1$, $\lambda_2 = -(d+gU^*)$, $\lambda_3 = -(e+gU^*)$

Since one of the eigen value is positive, therefore the equilibrium point $E_0(0,0,0)$ is locally unstable. The jacobian corresponding to $E_1(1,0,0)$ is:

$$\begin{bmatrix} -1 & -\frac{a}{1+b} & \frac{-af}{1+b} \\ 0 & \frac{c}{1+b}-d-gU^* & \frac{cf}{1+b} \\ 0 & 0 & -e-gU^* \end{bmatrix} \tag{25}$$

The corresponding eigen values with respect to the jacobian are:

$\lambda_1 = -1$, $\lambda_2 = -(d+gU^*-\frac{c}{1+b})$, $\lambda_3 = -(e+gU^*)$

The equilibrium point $E_1(1,0,0)$ is locally asymptotically stable provided $d+gU^* > \frac{c}{1+b}$ i.e. $R_1 < 1$ where $R_1 = \frac{c}{(1+b)(d+gU^*)}$.

Thus, the trajectories will approach to $E_1(1,0,0)$ when $R_1 < 1$. The Jacobian corresponding to $E_2(\bar{x},\bar{y},0)$ is:

$$\begin{bmatrix} -\bar{x}-\frac{ab\bar{x}\bar{y}}{(1+bx)^2} & \frac{-a\bar{x}}{1+b\bar{x}} & \frac{-a\bar{x}f}{1+b\bar{x}} \\ \frac{c\bar{y}}{(1+bx)^2} & 0 & \frac{c\bar{x}f}{1+b\bar{x}}-\beta\bar{y} \\ 0 & 0 & \beta\bar{y}-e-gU^* \end{bmatrix} \tag{26}$$

The corresponding eigen values with respect to the jacobian are $\lambda = \beta\bar{y}-e-gU^*$ and the rest two eigen values are obtained from the following characteristic equation:

$$\lambda^2 + \bar{x}(1-\frac{a\bar{y}b}{(1+b\bar{x})^2})\lambda + \frac{ca\bar{x}\bar{y}}{(1+b\bar{x})^3} = 0$$

$E_2(\bar{x},\bar{y},0)$ is locally asymptotically stable provided $1-\frac{ab\bar{y}}{(1+b\bar{x})^2}>0$ i.e $(1+b\bar{x})^2>ab\bar{y}$ and $\beta\bar{y}-e-gU^*<0$ i.e. $R_2<1$ where $R_2=\frac{\beta\bar{y}}{e+gU^*}$ otherwise unstable if $R_2>1$.

Remark: The local stability analysis of boundary equilibria shows that if $R_1<1$ than the trajectories will approach to $E_1(1,0,0)$ and if $R_2<1$ than the trajectories will approach to $E_2(\bar{x},\bar{y},0)$.

5. Biological significance of R_1 and R_2

We have obtained two threshold parameters R_1 and R_2 for the equilibrium points $E_1(1,0,0)$ and $E_2(\bar{x},\bar{y},0)$ respectively with their different biological meaning. Now, we will determine the biological significance of these parameters. Our first threshold parameter R_1 is defined as:

$$R_1=\frac{c}{(1+b)(d+gU^*)}$$

where $\frac{c}{1+b}$ defines the birth rate of the susceptible predators and $\frac{1}{d+gU^*}$ defines the total life span of the susceptible predator.We can easily calculate the reproduction number which is simply the product of $\frac{c}{1+b}$ and $\frac{1}{d+gU^*}$ that gives the average number of new born predators from the single predator. Here $R_1<1$ indicates that the predator population dies out and the probability of getting the infection in predator population will be negligible which clearly means that the equilibrium point $E_1(1,0,0)$ will be stable.

Our second threshold parameter R_2 is defined as:

$R_2=\frac{\beta\bar{y}}{e+gU^*}$ where $\beta\bar{y}$ defines the infection rate arising from the newly infected predators in a susceptible predator population and $\frac{1}{e+gU^*}$ defines the average duration of infection induced from infected predators. The reproduction number R_2 can be calculated easily as product of $\beta\bar{y}$ and $\frac{1}{e+gU^*}$, which defines the total life span of the infected predator. Here $R_2<1$ indicates that the disease will die out due to extinction of infected predators which clearly means that the disease free equilibrium point will be stable, otherwise unstable if $R_2>1$.

6. Interior equilibrium point and its local stability

In this section, we will discuss the local stability analysis of interior equilibrium point E^*. The jacobian for the interior equilibrium point $E^*(x^*, y^*, z^*)$ is:

$$\begin{bmatrix} -x^* - \frac{abx^*(y^*+fz^*)}{(1+bx^*)^2} & \frac{-ax^*}{1+bx^*} & \frac{-ax^*f}{1+bx^*} \\ \frac{c(y^*+fz^*)}{(1+bx^*)^2} & \frac{cx^*}{1+bx^*} - d - \beta z^* - gU^* & \frac{cx^*f}{1+bx^*} - \beta y^* \\ 0 & \beta z^* & 0 \end{bmatrix} \tag{27}$$

Now,the above Jacobian can also be written as:

$$\begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{bmatrix} \tag{28}$$

where,

$A_{11} = -x^* - \dfrac{abx^*(y^*+fz^*)}{(1+bx^*)^2}, A_{12} = -\dfrac{ax^*f}{1+bx^*},$

$A_{13} = -\dfrac{ax^*f}{1+bx^*}, A_{21} = \dfrac{c(y^*+fz^*)}{(1+bx^*)^2},$

$A_{22} = \dfrac{cx^*}{1+bx^*} - d - \beta z^* - gU^*, A_{23} = \dfrac{cx^*f}{1+bx^*} - \beta y^*$

$A_{31} = 0, A_{32} = \beta z^*, A_{33} = 0$

we can form a characteristic equation of the jacobian in the following way:

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0$$

where,

$$\begin{aligned} \sigma_1 &= -(A_{11} + A_{22}) \\ \sigma_2 &= A_{11}A_{22} - A_{23}A_{32} - A_{12}A_{21} \\ \sigma_3 &= A_{12}A_{23}A_{32} - A_{13}A_{21}A_{32} \end{aligned} \tag{29}$$

Now we will evaluate the value of $(\sigma_1\sigma_2 - \sigma_3)$

$$\sigma_1\sigma_2 - \sigma_3 = -A_{11}^2A_{22} + A_{11}A_{12}A_{21} - A_{22}^2A_{11} + A_{22}A_{23}A_{32} + A_{12}A_{21}A_{22} + A_{13}A_{21}A_{32}$$

Using Routh Hurwitz criteria,

$\sigma_1 > 0, \sigma_1\sigma_2 - \sigma_3 > 0, \sigma_3 > 0$

i.e. $A_{11} \leq 0$ and $A_{22} \leq 0$

that implies,

$(1+bx^*)^2 > ab(y^*+fz^*)$ and $\frac{cx^*}{1+bx^*} < (d+\beta z^*+r_2U^*)$

Since the sufficient conditions of Routh Hurwitz criteria are satisfied,thus $E^*(x^*,y^*,z^*)$ is locally asymptotically stable.

Theorem 6.1. The system enters into Hopf-bifurcation around the positive equilibrium E^* for the parameter $\beta = \beta^*$ if the following condition holds:

(i) $\sigma_1(\beta^*) > 0$

(ii) $\sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0$

(iii) $(\sigma_1(\beta^*)\sigma_2(\beta^*))' < \sigma_3'(\beta^*)$

Proof. Let β be the rate of force of infection. We assume that the interior point E^* is asymptotically stable. Our interest is on the parameter β that whether E^* loses its stability with the change in the parameter β i.e we assume β as the bifurcation parameter, then there exists a critical value β^* such that $\sigma_1(\beta^*) > 0$, $\sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0$, $[\sigma_1(\beta^*)\sigma_2(\beta^*)]' < \sigma_3{}'(\beta^*)$ For the occurrence of Hopf bifurcation, the characteristic equation must be obtained of the kind,

$$(\lambda^2(\beta^*) + \sigma_2(\beta^*))(\lambda(\beta^*) + \sigma_1(\beta^*)) = 0 \tag{30}$$

which has corresponding eigen values $\lambda_1(\beta^*) = i\sqrt{\sigma_2(\beta^*)}$, $\lambda_2 = -i\sqrt{\sigma_2(\beta^*)}$, $\lambda_3 = -\sigma_1(\beta^*) < 0$.

We will check if Hopf bifurcation is likely to occur at $\beta = \beta^*$, for this we will verify the transversality criteria, i.e.,

$$[\frac{dRe(\lambda(\beta))}{d\beta}]_{\beta=\beta^*} \neq 0$$

The roots for all β are:

$\lambda_1(\beta) = \mu(\beta) + i\nu(\beta)$,

$\lambda_2(\beta) = \mu(\beta) - i\nu(\beta)$,

$\lambda_3(\beta) = -\sigma_1(\beta)$

Now, in order to verify the transversality condition, we substitute $\lambda_j(\beta) = \mu(\beta) \pm i\nu(\beta)$ in equation (30) and then derivative is calculated, which gives,

$$F(\beta)\mu'(\beta) - G(\beta)\nu'(\beta) + H(\beta) = 0 \tag{31}$$

$$G(\beta)\mu'(\beta) + F(\beta)\nu'(\beta) + I(\beta) = 0 \tag{32}$$

where,

$$F(\beta) = 3\mu^2(\beta) + 2\sigma_1(\beta)\mu(\beta) + \sigma_2(\beta) + \sigma_2(\beta) - 3\nu^2(\beta)$$

$$G(\beta) = 6\mu(\beta)\nu(\beta) + 2\sigma_1(\beta)\nu(\beta)$$

$$H(\beta) = \mu^2(\beta)\sigma'_(\beta) + \sigma'_2(\beta)\mu(\beta) + \sigma'_(\beta) - \sigma'_1(\beta)\nu^2(\beta)$$

$$I(\beta) = 2\mu(\beta)\nu(\beta)\sigma'_1(\beta) + \sigma'_2(\beta)\nu(\beta)$$

We Know that, $\mu(\beta^*) = 0$, $\nu(\beta^*) = \sqrt{\sigma_2(\beta^*)}$, which results in,

$$F(\beta^*) = -2\sigma_2(\beta^*)$$

$$G(\beta^*) = 2\sigma_1(\beta^*)\sqrt{\sigma_2(\beta^*)}$$

$$H(\beta^*) = \sigma'_3(\beta^*) - \sigma'_1(\beta^*)\sigma_2(\beta^*)$$

$$I(\beta^*) = \sigma'_2(\beta^*)\sqrt{\sigma_2(\beta^*)}$$

We now determine the value for $\mu'(\beta^*)$ using equations (31),(32) and we get,

$$[\frac{dRe(\lambda_j(\beta))}{d\beta}]_{\beta=\beta^*} = \mu^{'}(\beta)_{\beta=\beta^*} = -\frac{G(\beta^*)I(\beta^*) + F(\beta^*)H(\beta^*)}{F^2(\beta^*) + G^2(\beta^*)}$$

$$= \frac{\sigma'_3(\beta^*) - \sigma^{'}_1(\beta^*)\sigma_2(\beta^*) - \sigma_1(\beta^*)\sigma^{'}_2(\beta^*)}{\sigma_1^2(\beta^*) + \sigma_2(\beta^*)} > 0$$

If $[\sigma_1(\beta^*)\sigma_2(\beta^*)]^{'} < \sigma^{'}_3(\beta^*)$ and $\lambda_3(\beta^*) = -\sigma_1(\beta^*) < 0$ then it clearly implies the transversality condition holds. Therefore,at $\beta = \beta^*$, Hopf bifurcation occurs.

Theorem 6.2. The system enters into Hopf-bifurcation around the positive equilibrium E^* for the parameter $g = g^*$ if the following condition holds:

(i) $\sigma_1(g^*) > 0$

(ii) $\sigma_1(g^*)\sigma_2(g^*) - \sigma_3(g^*) = 0$

(iii) $(\sigma_1(g^*)\sigma_2(g^*))' < \sigma_3'(g^*)$

Proof. The Proof of this theorem can be proved on the similar lines as above.

Remark: If there exist critical values of rate of infection β and rate of pollutant g such that $\sigma_1(g^*) > 0$, $\sigma_1(g^*)\sigma_2(g^*) - \sigma_3(g^*) = 0$, $(\sigma_1(g^*)\sigma_2(g^*))' < \sigma_3'(g^*)$ and $\sigma_1(\beta^*) > 0$, $\sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0$, $(\sigma_1(\beta^*)\sigma_2(\beta^*))' < \sigma_3'(\beta^*)$, then when $\beta > \beta^*$ and $g > g^*$, than E^* is stable, E^* looses its stability at $\beta = \beta^*$ and $g = g^*$ and Hopf bifurcation occurs. At $\beta < \beta^*$ and $g < g^*$, E^* becomes unstable and periodic solutions bifurcates from E^*.

7. Persistence

Persistence of a system means the survival of all the population of the system in future time. In this section, we show that strictly positive solutions do not have omega limit point on boundary of the non negative cone.

Theorem 7.1. The system is uniformly persistent if $R_1 > 1$ and further there exists finite number of periodic solutions $x = \varphi_r(t)$, $y = \chi_r(t)$, $r = 1, 2, \ldots, n$ in the $x - y$ plane provided for each periodic solutions of period T,

$$\eta_r = -(e + gU^*) + \frac{1}{T}\int_0^T \beta \chi_r dt > 0 \tag{33}$$

Proof. Suppose k be a point in the positive cone and $o(k)$ be orbit through k and Ω be the omega limit set of the orbit through k. Note that $\Omega(k)$ is bounded.

We claim that E_0 does not belong to $\Omega(k)$. Let us suppose on the contrary, that $E_0 \in \Omega(k)$, then there exists a point l in $\Omega(k) \cap W^s(E_0)$, where $W^s(E_0)$ denotes the stable manifold of E_0. Since, $o(l)$ lies in $\Omega(k)$ and $W^s(E_0)$ is the $y - z$ plane, we conclude that $o(l)$ is unbounded, which is a contradiction.

We claim that E_1 does not belong to $\Omega(k)$. Let us suppose on the contrary, that $E_1 \in \Omega(k)$, then as E_1 is a saddle point, which follows from the condition $R_1 > 1$, thus by the Butler McGhee Lemma, there exists a point l in $\Omega(k) \cap W^s(E_1)$, where $W^s(E_1)$ denotes the stable manifold of E_1. Since, $o(l)$ lies in $\Omega(k)$ and $W^s(E_1)$ is the $x - z$ plane, we conclude that $o(l)$ is unbounded, which is again a contradiction.

Finally, we have to show that there is no periodicity in $x-y$ plane or $E_2 \in \Omega(k)$. Let $r_i,\ i = 1,2\ldots n$ denote the closed orbit of the periodic solution $(\varphi_r(t),\ \chi_r(t))$ in $x-y$ plane such that r_i lies inside r_{i-1}. Let the jacobian matrix J given in (23) corresponding to r_i is denoted by $J_r(\varphi_r(t),\chi_r(t),0)$. We compute the fundamental matrix of the linear periodic system which is as follows:

$$X^{'} = J_r(t)X,\ X(0) = I \tag{34}$$

We see that $e^{\eta_r T}$ is the floquet multiplier in the z direction. From [19], we conclude that no r_i lies in $\Omega(k)$. Thus, $\Omega(k)$ lies in the positive cone and system (17 - 19) is persistent. Finally we conclude that only the closed orbits and the equilibria from the omega limit set of the solutions are on the boundary of R^3_+ and the system (17 - 19) is dissipative. Now using a theorem of [3] , we conclude that the system is uniformly persistent.

Theorem 7.2. If the conditions $R_1 > 1$ and $R_2 > 1$ are satisfied and if there exists no limit cycle in the $x-y$ plane, then system (17 - 19) is uniformly persistent.

Proof. The proof is on the same line as above and hence omitted.

8. Numerical Example

We know that infection and pollutant has a very important role in the dynamics of any system. But our keen interest is what happens to the dynamics of the system if pollutant enters into it, which is already infected. We have considered some hypothetical set of parameters:
$a = 2.8,\ f = 0.01,\ b = 2.8,\ c = 0.336,\ d = 0.03,\ e = 0.09$. The graphs are plotted for two cases. In case I, when no pollutant is present in the system and the system is affected only by infection, we have taken the infection rate $\beta = 0.24$ and we discovered that the population of prey and susceptible predator is showing an oscillatory behavior whereas infected predator is going to extinction (Figure 1, 2, 3, 4). As we increase the infection rate $\beta = 0.25$, the system starts stabilizing.(Figure 11, 12, 13, 14)and it remains stable as we increase the infection rate β to $\beta = 0.30$(Figure 15). In case II, we see that, if we incorporate the effect of pollutant by considering $g = 0.14\ U = 0.2$, then the prey population and susceptible predator population which was showing an oscillatory behavior at $\beta = 0.24$ suddenly stabilizes by including the

pollutant (Figure 5, 6, 7).Thus, g also plays an important role in stabilizing the dynamical system. We further observed that as we decrease g to 0.13 then again the system is showing an oscillatory behavior (Figure 8, 9, 10). Hence, β and g can be considered as bifurcation parameters for the system.

The introduction of disease and pollutant not only control or eradicate the infected predator, but also allow the prey species to recover. For example, polluted parasites can be introduced to control so called predators such as stoats, ship rats so as to protect the endangered endemic birds such as kiwi for their survival in New Zealand.

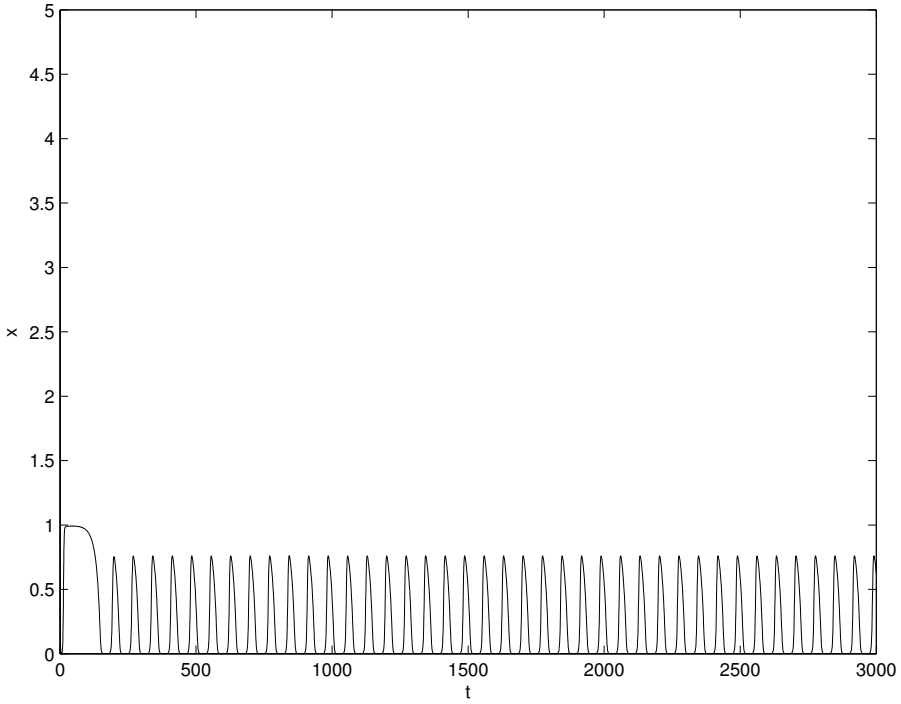

FIGURE 1. Oscillatory behavior of prey population without pollutant $\beta = 0.24$

9. Conclusion

A prey-predator system is considered in our paper where parasitic infection impacts the predator population. The existence, analysis of local stability of all the boundary and interior equilibrium points are analyzed. Further, a bifurcation parameters β and g are obtained. Reproduction thresholds R_1 and R_2 are also obtained which also determines the stability of the boundary

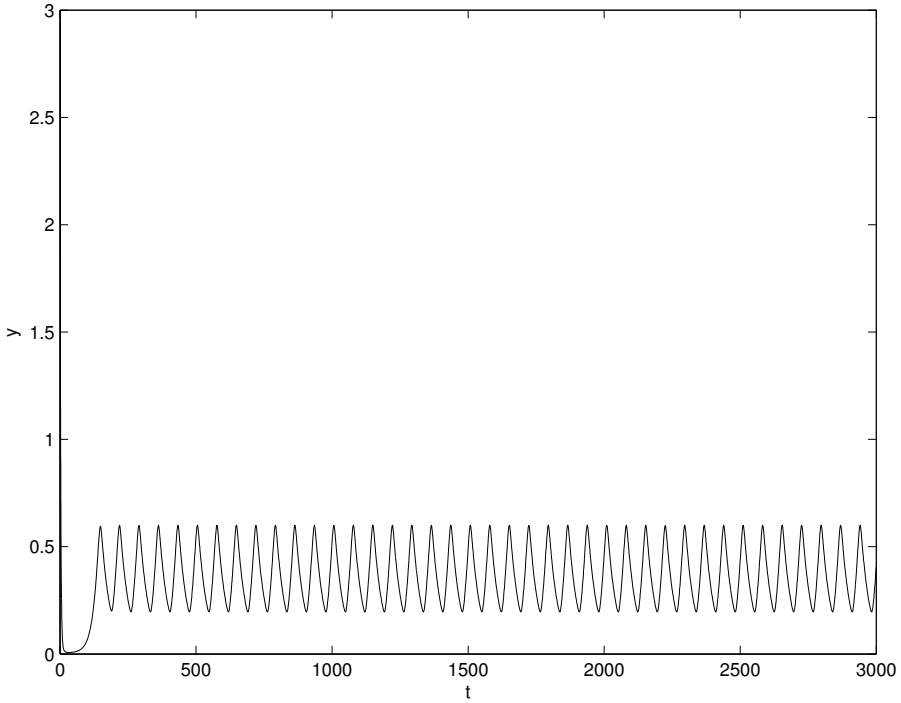

FIGURE 2. Oscillatory behavior of susceptible predator population without pollutant $\beta = 0.24$

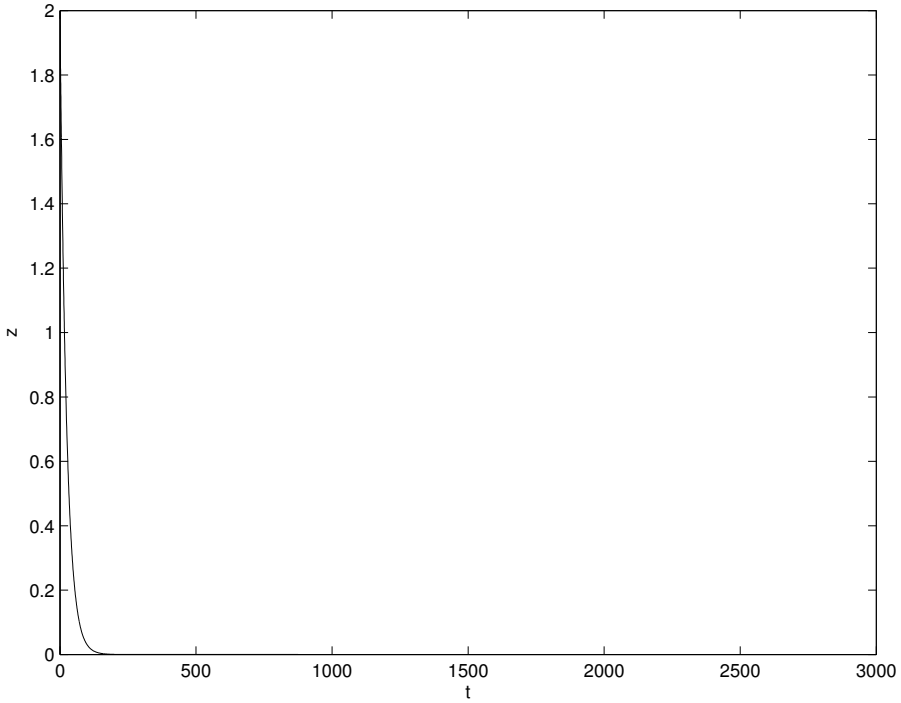

FIGURE 3. Behavior of infected predator population without pollutant $\beta = 0.24$

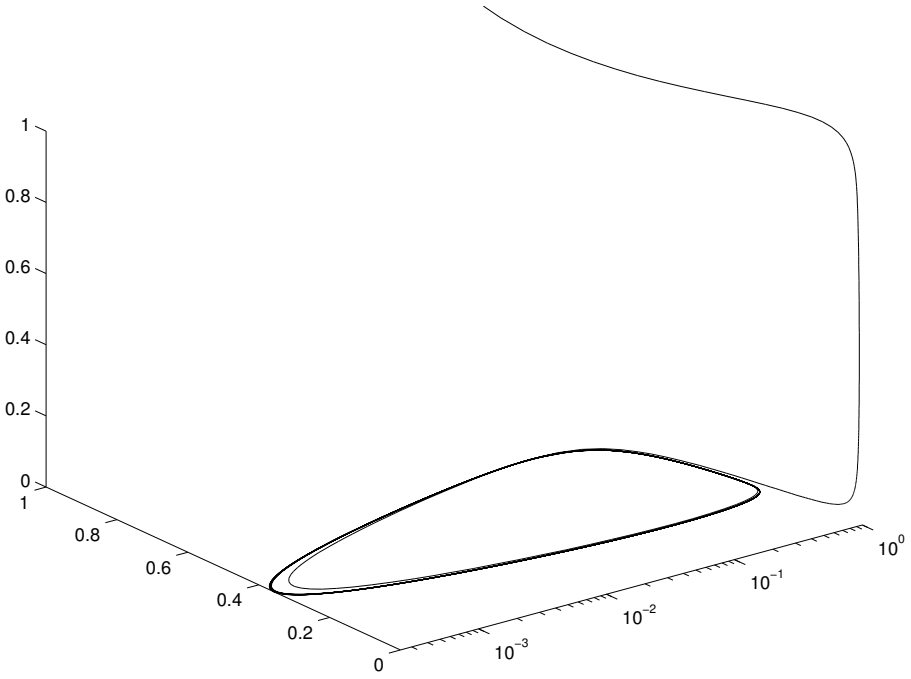

FIGURE 4. Behavior of the system without pollutant $\beta = 0.24$

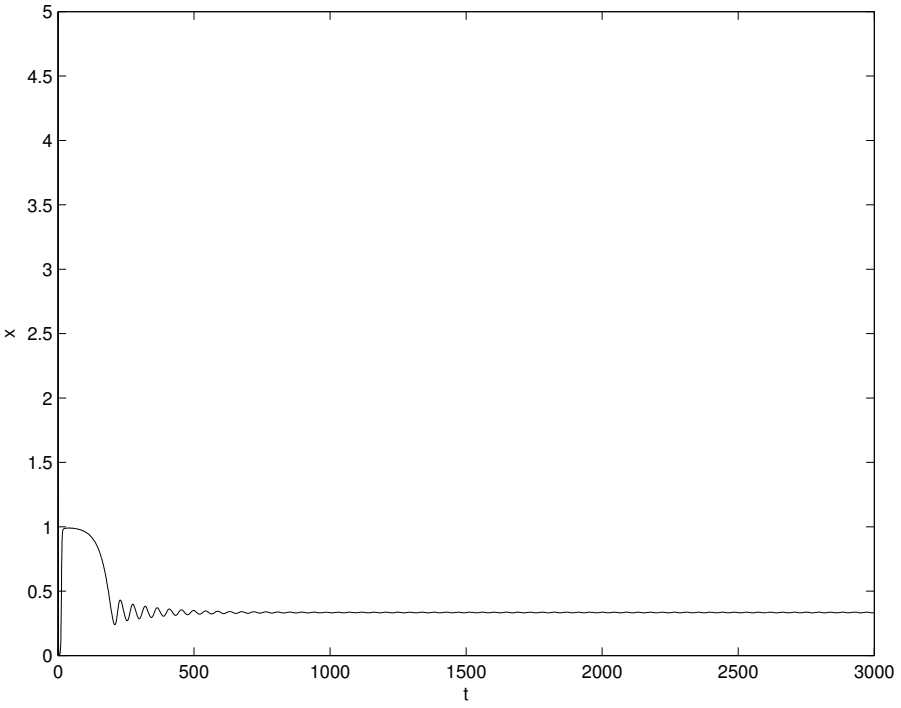

FIGURE 5. Behavior of prey poplutaion with pollutant $\beta = 0.24$, $g = 0.14$

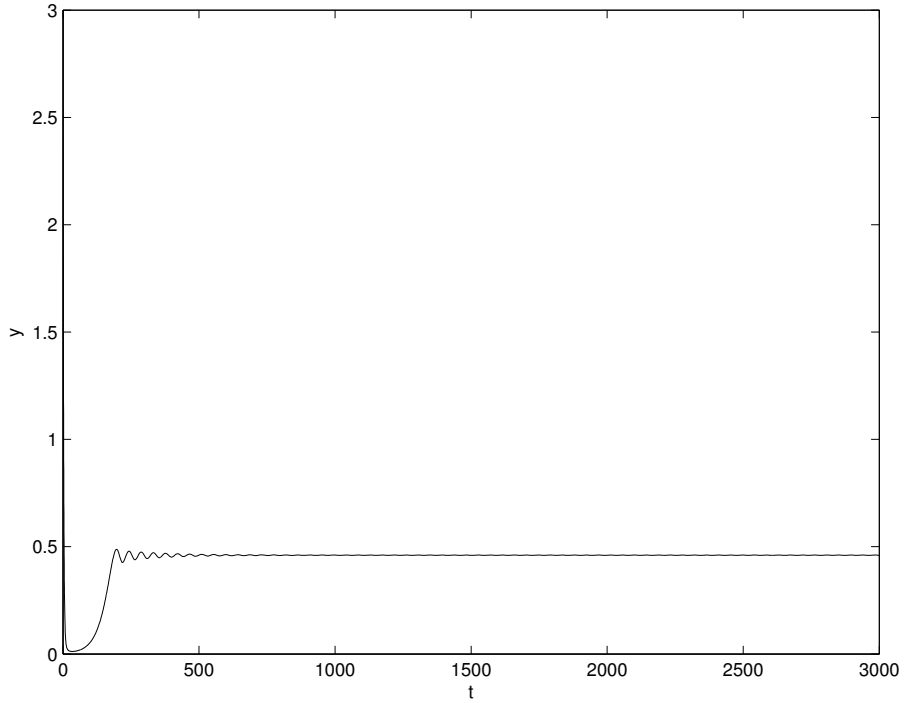

FIGURE 6. Behavior of susceptible predator with pollutant $\beta = 0.24$, $g = 0.14$

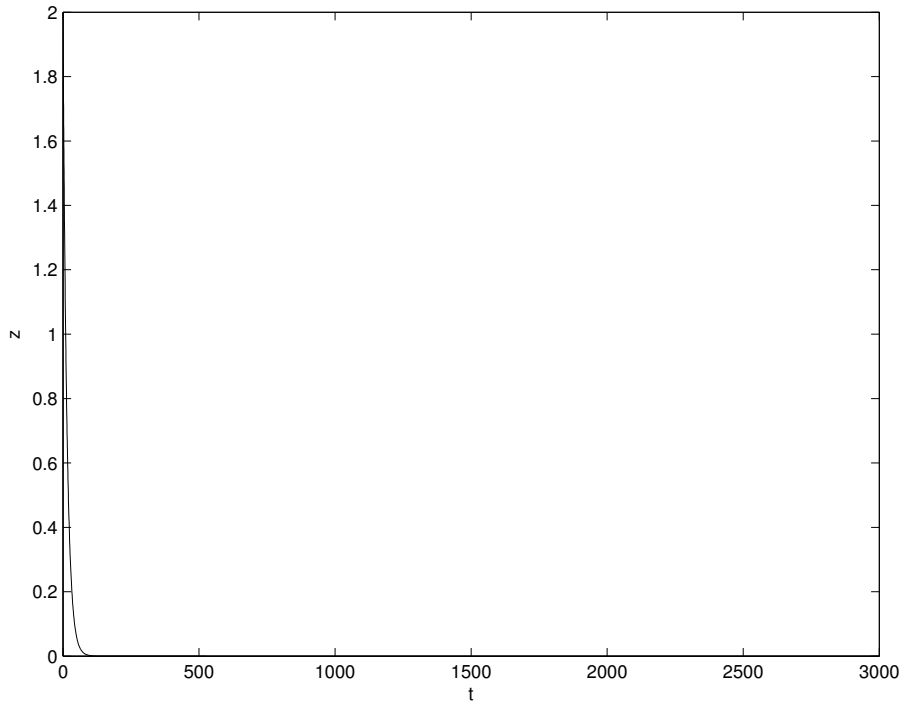

FIGURE 7. Behavior of infected predator with pollutant $\beta = 0.24$, $g = 0.14$

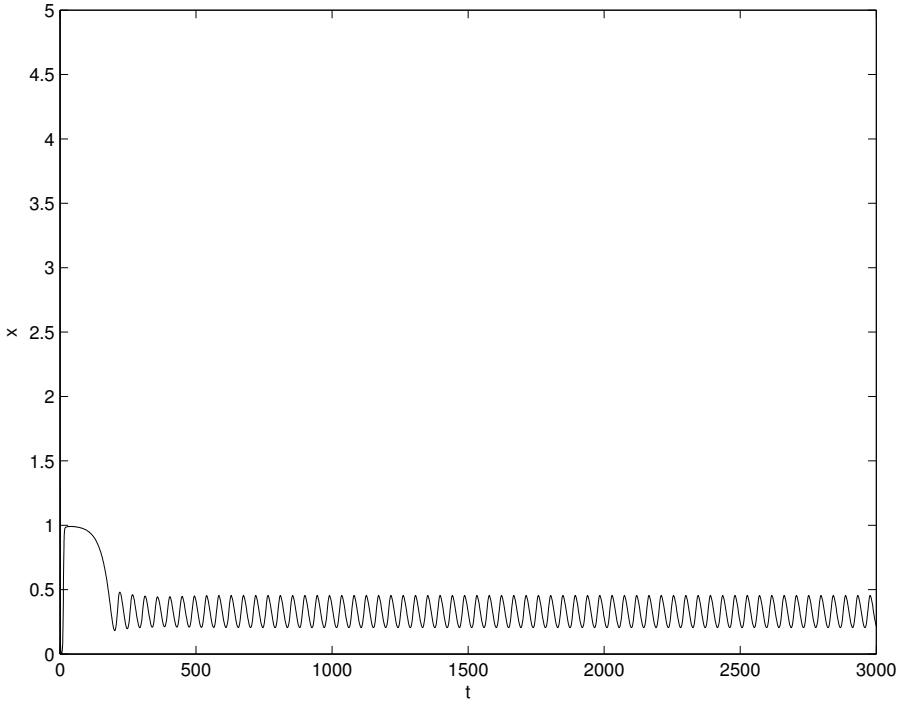

FIGURE 8. Oscillatory behavior of prey population with pollutant $\beta = 0.24$, $g = 0.13$

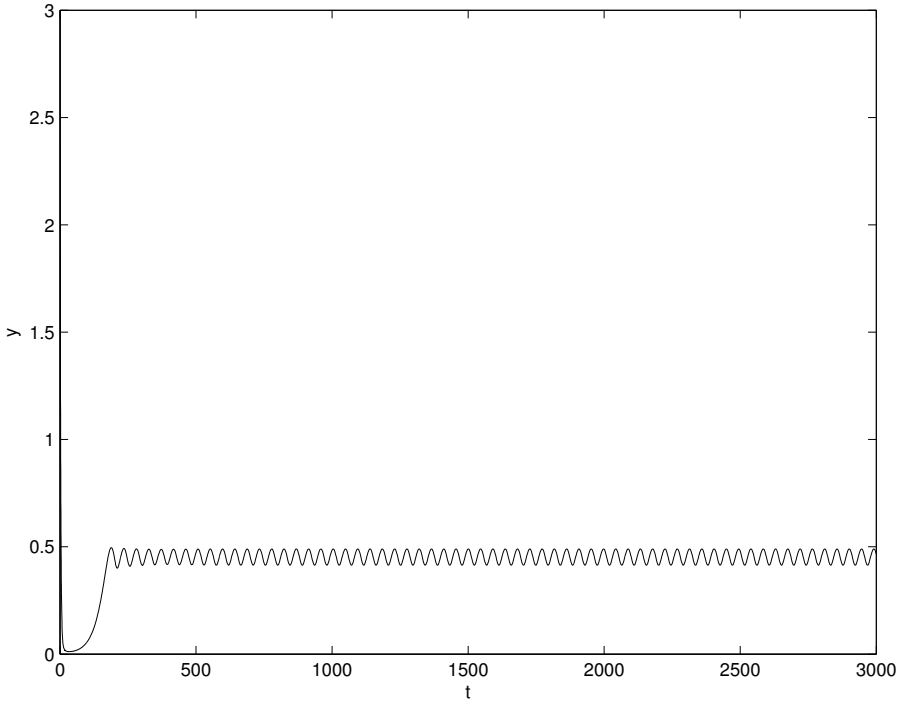

FIGURE 9. Oscillatory behavior of susceptible predator with pollutant $\beta = 0.24$, $g = 0.13$

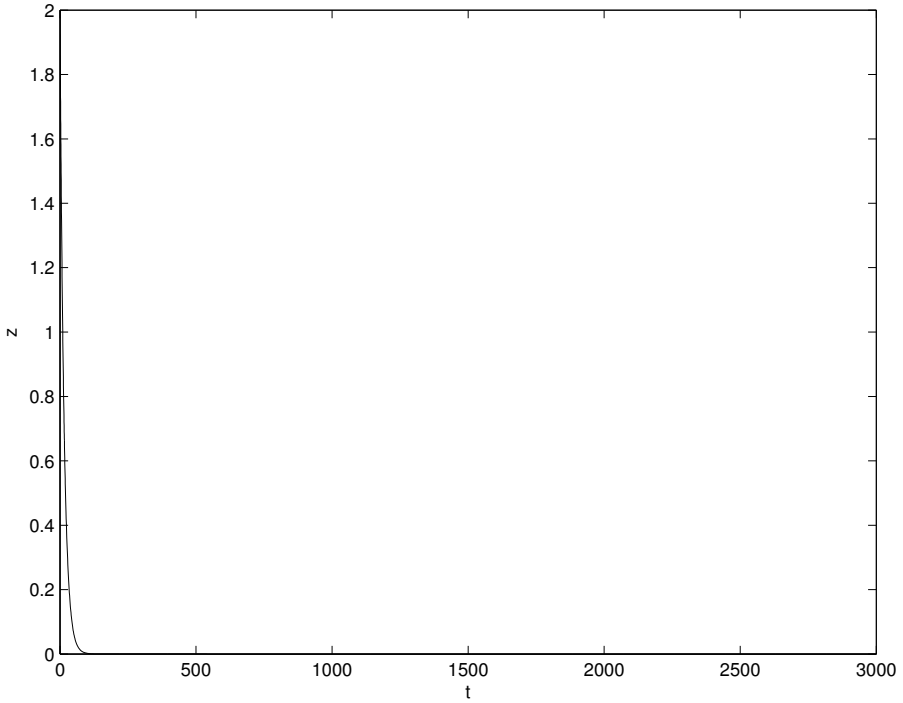

FIGURE 10. Behavior of infected predator with pollutant $\beta = 0.24$, $g = 0.13$

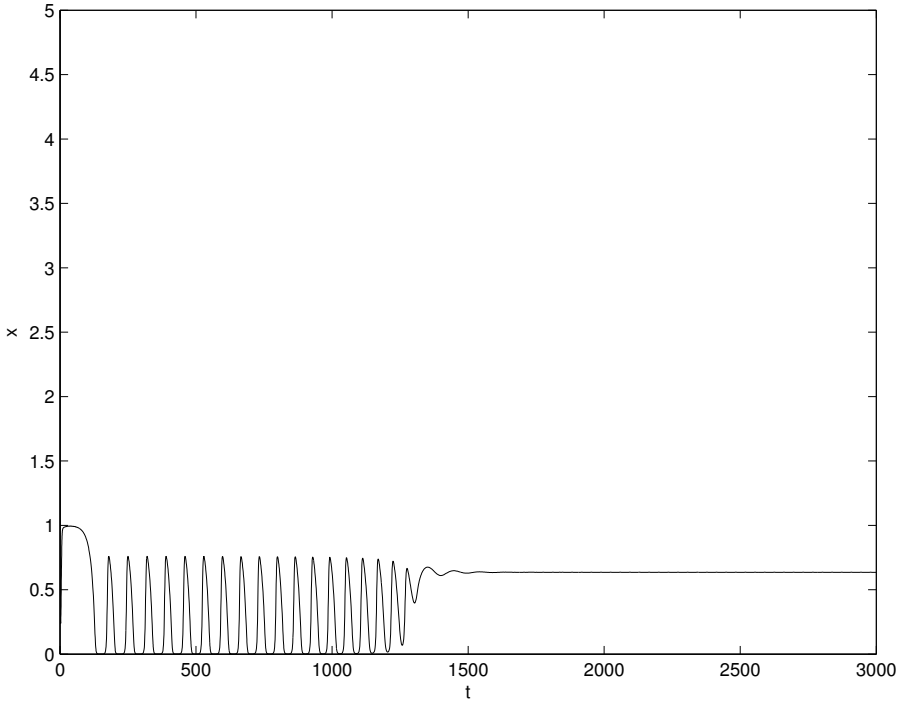

FIGURE 11. Behavior of prey population without pollutant at $\beta = 0.25$

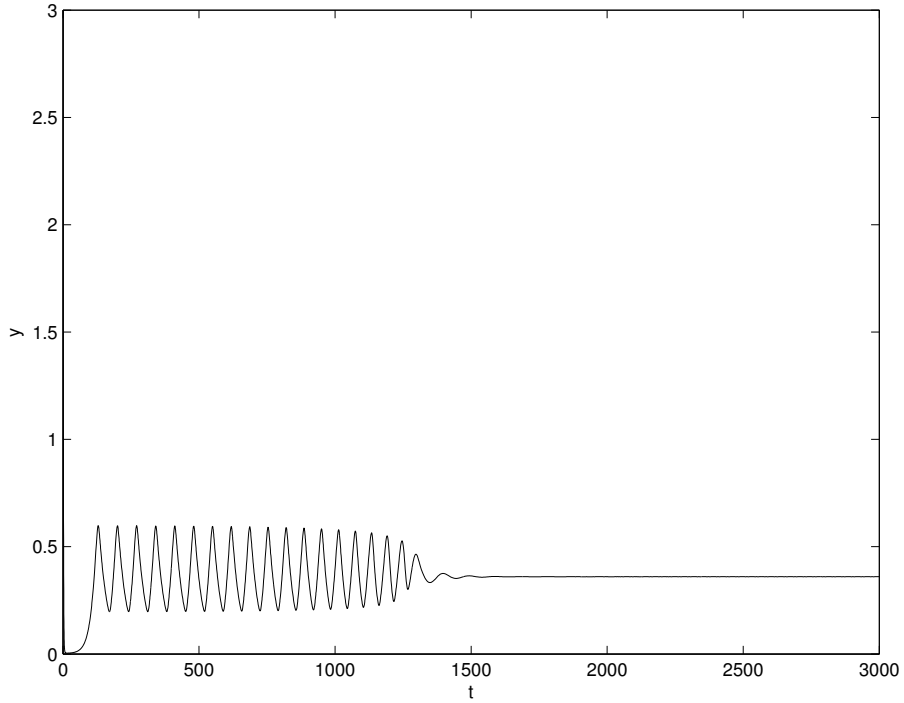

FIGURE 12. Behavior of susceptible predator without pollutant $\beta = 0.25$

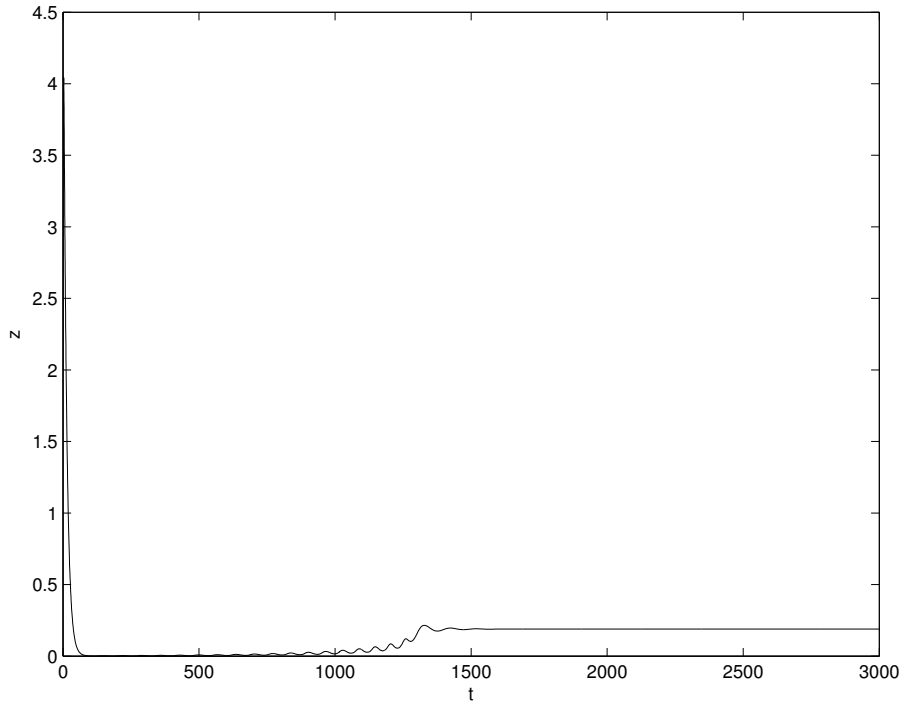

FIGURE 13. Behavior of infected predator without pollutant $\beta = 0.25$

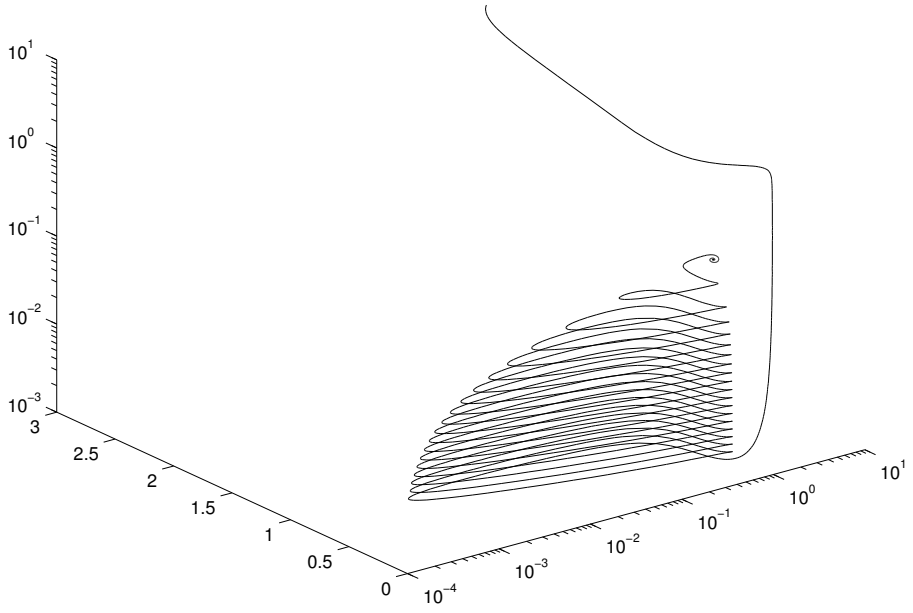

FIGURE 14. Behavior of system without pollutant $\beta = 0.25$

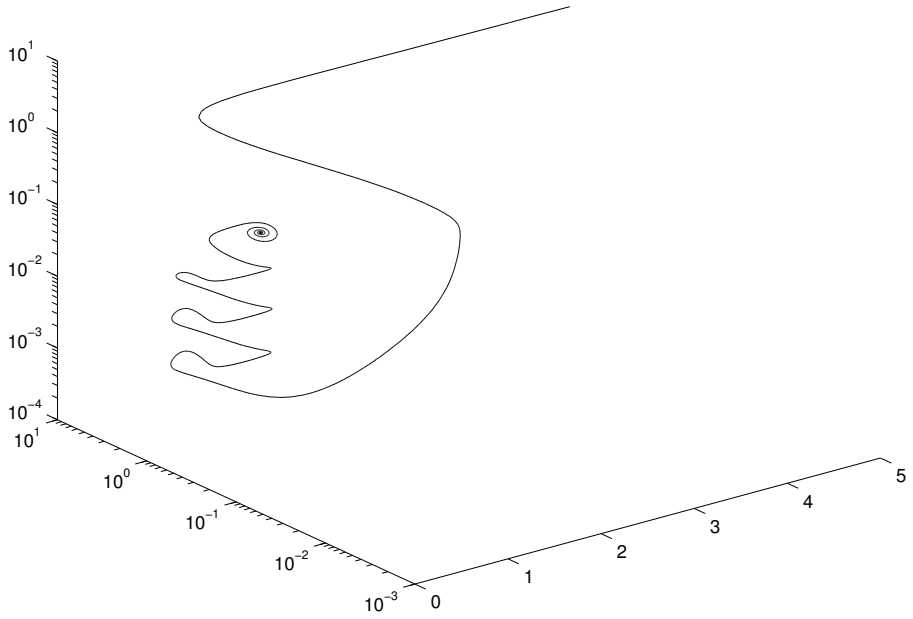

FIGURE 15. Behavior of the system without pollutant $\beta = 0.30$

equilibrium points. The biological significance of reproduction numbers are explained. The focus of the paper is totally different from the previous papers studied earlier. The highlights of the paper is what is the importance of including pollutant in any dynamical system. The two major possible outcomes upon introduction of pollutant are :

(1) the host population can be driven to extinction.

(2) the system can be stabilized which was earlier unstable.

It is observed that increasing the infection and pollutant parameters β and g stabilizes the system. The system shows an oscillatory behavior at $\beta = 0.24$ in the absence of pollutant but stabilizes after the addition of pollutant $g = 0.14$. Further, it can again destabilize if the parameter g is reduced to $g = 0.13$. Thus β and g are the bifurcation parameters. This result is very interesting because it is not necessary that adverse effects in any dynamical system will always destabilize the system, sometimes it helps in stabilizing the system by eradicating the infected population. To analyze the above points, we have gone through extensive numerical simulations in MATLAB for different set of parameters. Finally, the persistence of all the population is also obtained.

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