



Available online at <http://scik.org>

Commun. Math. Biol. Neurosci. 2022, 2022:3

<https://doi.org/10.28919/cmbn/6887>

ISSN: 2052-2541

STABILITY AND BIFURCATION ANALYSIS OF A CONTAMINATED SIR MODEL WITH SATURATED TYPE INCIDENCE RATE AND HOLLING TYPE-III TREATMENT FUNCTION

NAINA ARYA, SUMIT KAUR BHATIA*, AMRITA KUMAR

Department of Mathematics, Amity Institute of Applied Sciences, Amity University, Uttar Pradesh, Noida, India

Copyright © 2022 the author(s). This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract. The paper deals with an epidemic model which is contaminated. Logistic growth input of vulnerable is taken in our SIR model. Saturated type incidence rate alongside Holling type III treatment function and time delay in growth component has been considered. The stability analysis of equilibrium points is done and the basic reproduction number (R_0) is obtained. Using (R_0), Local Stability around disease-free equilibrium point has been acquired. When R_0 is less than one, it is locally stable which means disease doesn't exist in the environment anymore and when R_0 is greater than one, it is unstable which means disease still exists in the environment. We have shown that at $R_0 = 1$ the model exhibits forward bifurcation. The conditions for the local stability of endemic equilibrium point have also been obtained. Conditions for the existence of Hopf bifurcation along with its direction and stability has been determined. Numerical simulations have been performed utilizing MATLAB to confirm the logical outcomes acquired. We have seen that to diminish the effect of illness, contamination ought to be decreased. Also, suitable strategies should be chosen to expand the capacity of treatment to decrease the effect of infection.

Keywords: SIR model; delay differential equation; Holling type III treatment function; stability analysis; Hopf bifurcation.

2010 AMS Subject Classification: 92D05, 92D25, 92D40.

*Corresponding author

E-mail address: sumit2212@gmail.com

Received October 14, 2021

1. INTRODUCTION

With constant progress and increase of population in the urban areas, a wide range of problems related to environment such as air pollution, release of toxic materials, solid waste disposals, deforestation and so on, have gained attention much greater than ever before. Moreover, the amount of pollution in the environment can also be affected by the local species; the pollution gets absorbed, digested, and eventually gets metabolized by the species. Environment is one of the most important elements present to the society. The presence of pollution in the environment is a very broad topic and many authors have incorporated the effects and factors of pollution in their study [15, 5, 11, 19]. The major cause of pollution is the excessive use of environmental resources and chemicals, without taking into consideration the safety measures to control pollution. Hence, resulting pollution directly affects the humans' health. Many scientists have shown their concerns regarding the reduction of environmental pollution. Motivated by the same novelty here, we have developed an SIR model where we have considered the effect of contaminants on populace. Since epidemiology is a discipline which deals with infection in a population. It deals with all aspects of epidemic such as spread, control, vaccination strategy etc. Mathematical modeling is an important tool in epidemiology which analyze the spread and control of infection in the species. Based on this here, an epidemic model describing the disease dynamics is developed. In [4, 1, 2], the researchers in their models have considered constant recruitment rate of the susceptible with saturated incidence rate. However, logistic growth input of susceptible have been considered in many problems and is practical for infections with high mortality rate. A model was proposed by Hutchinson [7] in which he considered a logistic equation with delay in death rate term or carrying capacity which is as follows [10]:

$$\frac{dx}{dt} = rx \left(1 - \frac{x(t - \tau)}{K} \right)$$

Initially, the populace growth was exponential but it oscillated at higher levels of population. In mathematical community, his formulation on logistic equation involving the delay is know as *delayed logistic equation*.

Since, there are many examples involving delay in this organic world. One of them being, at the time of sexual reproduction, the time from the fertilization of eggs to the birth. Another

example involving delay is, time involved in the digestion of food and its conversion to biomass resources. In our paper, we have incorporated delay in growth term apart from interaction term. Since there are numerous reasons because of which infection is caused, the doctors and health organizations came up with various treatments to have control over the spread of disease among populace. These treatments involve antibiotics, vaccination, blood transfusion etc. Researchers who studied models based on epidemics worked on the assumption that treatment rate of disease is in direct relation to number of infective populace. It is also known in general that treatment affects the recovery rate. Authors like Wang and Ruan [21] studied the SIR model in which they examined the case where $T(I) = 0$ for $I = 0$ and is equal to r which is constant for all $I > 0$. Wang [20] in his further work on SIR model, incorporated piecewise continuous treatment function: $T(I) = \min(rI, rI_0)$, where the health care system reaches capacity at infective level I_0 , and $r > 0$ is constant. Zhang and Liu [24], A.Kumar and Nilam [3] used the saturated treatment function: $T(I) = \frac{rI}{1+\alpha I}$ for $r > 0, \alpha \geq 0$. Dubey et al. [13] incorporated the Holling type III treatment rate, that determines the case where recovery rate grows rapidly with the increase in infectives initially, and later on, it settles to a saturated value after developing gradually. In our paper, an SIR epidemic model has been developed with t_1 as time delay in growth component and saturated Holling type III treatment rate. We have also considered pollution in the system. The paper is organised as follows: Section (2) describes the model, and we have discussed the characteristics of the model, section(3) examines the disease-free equilibrium and its stability analysis, in section (4) we have studied the endemicity of the model, section (5) explores the existence of hopf bifurcation, in Section (6) the numerical simulations of the system are discussed and analysed.

2. MODEL DESCRIPTION AND BASIC PROPERTIES

We have presented the model where we have supposed that entire populace is denoted by $W(t)$ is distributed mainly into three compartments, viz. susceptible population $X(t)$ (who are supposed to be infected by the disease), infected population $Y(t)$ (who are capable of spreading the disease) and recovered population $Z(t)$ (who have recovered from the disease and no more infectious). We assumed that the susceptible populace follows logistic growth with intrinsic

growth rate r and environment carrying capacity K ([9]),

$$\frac{dX}{dt} = rX \left(1 - \frac{X(t-t_1)}{K} \right)$$

where t_1 is time delay and is derived by assuming that $\frac{X'}{X}$ i.e. the net per capita rate of change is dependent on state of system t_1 (time units) (Hutchinson [7]). Model assumes saturated incidence rate of the form:

$$F(X, Y) = \frac{\lambda XY}{1 + \varepsilon Y}$$

where λ is the highest disease transmission rate and the factor $1/(1 + \varepsilon Y)$ represents the inhibition effect when the number of infectives is large.

Therapies, surgeries, medications etc., are important tools of treatment of disease. Proper treatment effectively reduces the infection present in the populace. These treatments have the capacity to influence the recovery rate of the infectives. Therefore, proper treatment rate must be considered in the model. In our model, we have considered Holling type-III treatment function [2, 13] which is as follows:

$$T(Y) = \frac{aY^2(t)}{1 + bY^2(t)}$$

a is considered as the cure rate of infection and b is considered as the limitation rate in the treatment of infected.

Above assumptions leads to the following set of delay differential equations which includes the treatment function:

$$\begin{aligned} \frac{dX}{dt} &= rX \left(1 - \frac{X(t-t_1)}{K} \right) - \frac{\lambda XY}{1 + \varepsilon Y} \\ \frac{dY}{dt} &= \frac{\lambda XY}{1 + \varepsilon Y} - (\mu + d + \sigma)Y - \frac{aY^2(t)}{1 + bY^2(t)} \\ \frac{dZ}{dt} &= \sigma Y + \frac{aY^2(t)}{1 + bY^2(t)} - \mu Z \end{aligned}$$

Now in order to study the behavior of population under the pollutants we have considered certain assumptions:

- (i) Let us suppose that the pollutants penetrates into population through the intake of food and from the outside resource,
- (ii) Pollutants deprives as the time passes from the organism due to some metabolic processes

or any other way.

Let Q be the constant exogenous rate of input of toxicant in environment, then the composition of equation with respect to the concentration of environmental pollutant and concentration of toxicants for an organism is given by:

$$\begin{aligned}\frac{dA}{dt} &= Q - hA \\ \frac{dU}{dt} &= a_1A + \frac{d_1\eta\phi}{a_1} - (l_1 + l_2)U\end{aligned}$$

where $A(t)$ is the concentration of contaminants present in environment, $U(t)$ is the concentration of pollutant present in the organism, h denotes the rate by which toxicants are lost from the resource, a_1 denotes the rate of the resource pollutants per unit mass organism, η denotes level of concentration of pollutants in the environment, the mean rate of intake of food per unit mass organism is denoted by α_2 , d_1 is the uptake rate of toxicant in the food per unit mass organism, l_1 and l_2 are rate of net ingestion and depuration of pollutant in the organism. Thus our extended model is:

$$\left\{ \begin{aligned}\frac{dX}{dt} &= rX \left(1 - \frac{X(t-t_1)}{K} \right) - \frac{\lambda XY}{1 + \varepsilon Y} - \alpha_1 UX \\ \frac{dY}{dt} &= \frac{\lambda XY}{1 + \varepsilon Y} - (\mu + d + \sigma)Y - \frac{aY^2(t)}{1 + bY^2(t)} - \alpha_2 UY \\ \frac{dZ}{dt} &= \sigma Y + \frac{aY^2(t)}{1 + bY^2(t)} - \mu Z - \alpha_3 UZ \\ \frac{dA}{dt} &= Q - hA \\ \frac{dU}{dt} &= a_1A + \frac{d_1\eta\phi}{a_1} - (l_1 + l_2)U\end{aligned}\right.$$

Table 1. Meaning of the parameters

Parameters	Description
t_1	Time delay
λ	Transmission rate of infection
ε	Inhibition measures by the infectives
μ	Natural diminishing rate
d	Diminishing rate due to infection
σ	Recovery rate

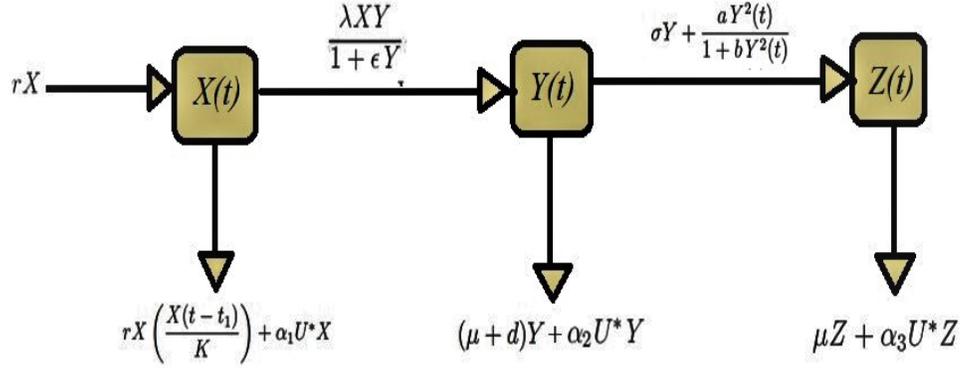


FIGURE 1. Transmission of disease in the infective populace from susceptible populace, with delay in growth component: The SIR model.

Using Corollary 2.1 in [15] the above model gets transformed into:

$$(1) \quad \begin{cases} \frac{dX}{dt} = rX \left(1 - \frac{X(t-t_1)}{K} \right) - \frac{\lambda XY}{1 + \epsilon Y} - \alpha_1 U^* X \\ \frac{dY}{dt} = \frac{\lambda XY}{1 + \epsilon Y} - (\mu + d + \sigma)Y - \frac{aY^2(t)}{1 + bY^2(t)} - \alpha_2 U^* Y \\ \frac{dZ}{dt} = \sigma Y + \frac{aY^2(t)}{1 + bY^2(t)} - \mu Z - \alpha_3 U^* Z \end{cases}$$

Now since the first two equations in the above system does not have Z in it, therefore our model reduces to:

$$(2) \quad \begin{cases} \frac{dX}{dt} = rX \left(1 - \frac{X(t-t_1)}{K} \right) - \frac{\lambda XY}{1 + \epsilon Y} - \alpha_1 U^* X \\ \frac{dY}{dt} = \frac{\lambda XY}{1 + \epsilon Y} - (\mu + d + \sigma)Y - \frac{aY^2(t)}{1 + bY^2(t)} - \alpha_2 U^* Y \end{cases}$$

with initial conditions

$$\begin{cases} X(\varpi) = \psi_1(\varpi), Y(\varpi) = \psi_2(\varpi), \\ \psi_i(\varpi) \geq 0, \varpi \in [-t_1, 0], \psi_i(0) > 0 (i = 1, 2), \end{cases}$$

where $(\psi_1(\varpi), \psi_2(\varpi)) \in \mathbb{C}([-t_1, 0], \mathbb{R}_2^+)$.

\mathbb{C} here represents the Banach space of continuous mapping in $[-t_1, 0]$ into \mathbb{R}_2^+ .

2.1. Basic Properties of the Model. For system (2) to be meaningful, all of $(X(t), Y(t), Z(t))$ must be nonnegative and $r, K, \lambda, \varepsilon, \mu, d, \sigma, a, b > 0$. We show that all solutions of (2) will continue to remain positive for $t \geq 0$ and also we will show that system is bounded.

Theorem 1: The variables $(X(t), Y(t))$ of the system (2) are non-negative.

Proof: As the system (2) can be written as:

$$T = \text{col}(X, Y) \in \mathbb{R}_2^+,$$

$$(\psi_1(\theta), \psi_2(\theta)) \in ([-t_1, 0], \mathbb{R}_2^+) \text{ and } \psi_i(0) > 0 (i = 1, 2)$$

$$G(T) = \begin{pmatrix} G_1(T) \\ G_2(T) \end{pmatrix} = \begin{pmatrix} rX \left(1 - \frac{X(t-t_1)}{K}\right) - \frac{\lambda XY}{1+\varepsilon Y} - \alpha_1 U^* X \\ \frac{\lambda XY}{1+\varepsilon Y} - (\mu + d + \sigma)Y - \frac{aY^2(t)}{1+bY^2(t)} - \alpha_2 U^* Y \end{pmatrix}$$

Then the model system (2) becomes:

$$(3) \quad \frac{dT}{dt} = G(T)$$

with $T(\theta) = (\psi_1(\theta), \psi_2(\theta)) \in ([-t_1, 0], \mathbb{R}_2^+)$ and $\psi_i(0) > 0 (i = 1, 2)$. It can be easily verified in the model (3) that whenever $T(\theta) \in \mathbb{R}_+$ is considered such that X, Y are both equals to zero, then

$$G_i(T)|_{x_i=0, T \in \mathbb{R}_2^+} \geq 0,$$

with $x_1(t) = X(t), x_2(t) = Y(t)$. From [Yang et al., [23]], using the lemma, any solution of (3) with $T(\theta) \in \mathbb{C}_+$, say $T(t) = T(t, T(\theta))$, is such that $T(t) \in \mathbb{R}_2^+$ for all t greater than or equals to zero. Therefore, the solution of (2) exist in \mathbb{R}_2^+ and remain positive for all t greater than zero. Following lemma will show the boundedness of the above model.

Lemma 1: The region

$$B = \left\{ (X, Y) \in \mathbb{R}_2^+ : X + Y \leq \frac{(r+1)M}{\eta}, X \leq M \right\}$$

where $\eta = \min[1, \mu + d + \sigma + \alpha_2 U^*]$ and $M = \max[X(0), K - \frac{\alpha_1 U^*}{r}]$ is an attracting region for the disease transmission model which is positively invariant and is given by (2) with initial conditions in R_+^2

Proof: From first equation of the model (2), we have

$$\frac{dX}{dt} \leq rX \left(1 - \frac{X}{K} - \frac{\alpha_1 U^*}{r} \right)$$

Hence, it yields $\limsup_{t \rightarrow \infty} X(t) \leq M$.

Let $L(t) = X(t) + Y(t)$ then

$$\begin{aligned} \frac{dL}{dt} &= rX \left(1 - \frac{X}{K} - \frac{\alpha_1 U^*}{r} \right) - (\mu + d + \sigma + \alpha_2 U^*)Y - \frac{aY^2}{1 + bY^2} \\ \frac{dL}{dt} &\leq rX \left(1 - \frac{X}{K} - \frac{\alpha_1 U^*}{r} \right) - X^2 - (\mu + d + \sigma + \alpha_2 U^*)Y \\ \frac{dL}{dt} &\leq (r+1)X - X - (\mu + d + \sigma + \alpha_2 U^*)Y \\ \frac{dL}{dt} &\leq (r+1)M - \eta W \end{aligned}$$

This follows that $0 \leq L(t) \leq \frac{(r+1)M}{\eta}$ for sufficiently large t .

The solutions of (2) along with initial conditions are bounded in the region \mathbb{R}_2^+ and enters D as $t \rightarrow \infty$. Moreover from the above lemma, we get that the model (2) is well formulated, both epidemiologically as well as mathematically .

3. STABILITY OF THE DISEASE-FREE EQUILIBRIUM

Here, we deal with the stability analysis of $E = \left(\frac{K(r - \alpha_1 U^*)}{r}, 0 \right)$ (the disease-free equilibrium). We begin by obtaining a key parameter R_0 which is the basic reproduction number. It is basically the threshold quantity for control of infection. It represents the number of infectives who got infected because of existing single infective [14]. The equilibrium solutions of system with no delay and of the system with delay are both same. (Tipsri and Chinviriyasit [18]). Here R_0 is computed as,

$$R_0 = \frac{\lambda K(r - \alpha_1 U^*)}{r(\mu + d + \sigma + \alpha_2 U^*)}$$

The Jacobian of the system with no delay, evaluated at $E = \left(\frac{K(r - \alpha_1 U^*)}{r}, 0 \right) = (X_0, 0)$ is as follows:

$$J(E) = \begin{bmatrix} -(r - \alpha_1 U^*) & \frac{-\lambda K(r - \alpha_1 U^*)}{r} \\ 0 & (\mu + d + \sigma + \alpha_2 U^*)(R_0 - 1) \end{bmatrix}$$

then, we have the following characteristic equation as:

$$P(\zeta) = \det(J(E) - \zeta I)$$

$$= (\zeta + (r - \alpha_1 U^*))(\zeta - (\mu + d + \sigma + \alpha_2 U^*)(R_0 - 1)) = 0$$

Thus, there are two eigenvalues: $\zeta_1 = -(r - \alpha_1 U^*)$ and $\zeta_2 = (\mu + d + \sigma + \alpha_2 U^*)(R_0 - 1)$. It is clear that ζ_1 is always negative, while $\zeta_2 < 0$ when $R_0 < 1$ and $\zeta_2 > 0$ when $R_0 > 1$. We know by Hartman-Grobman's theorem [12] that if $\zeta_2 \neq 0$, the solutions of the system (2) and its linearization equivalent near E . Then, we have the theorem as:

Theorem 2: E is a stable node when $R_0 < 1$, and it is a saddle when $R_0 > 1$.

3.1. Analysis at $R_0 = 1$. Here, we will analyse the stability of the undelayed model at $E(X_0, 0)$ and $R_0 = 1$.

Theorem 3: The system (2) will undergo forward bifurcation at disease free equilibrium E when R_0 crosses one.

Proof: When $R_0 = 1$ we get $\frac{\lambda K(r - \alpha_1 U^*)}{r(\delta + \alpha_2 U^*)} = 1$. Let the bifurcation parameter be $\lambda = \frac{r(\delta + \alpha_2 U^*)}{K(r - \alpha_1 U^*)} = \lambda^*$.

We can notice one of the eigenvalue of the system (2) vanishes and another eigenvalue is negative when evaluated at $R_0 = 1$ and $\lambda = \lambda^*$. Here, stability behavior of equilibrium point at R_0 equals to one is done by center manifold theory [Sastry [17]] as linearization is not applicable. For this purpose we define $X = x$ and $Y = y$, then the system (2) becomes:

$$(4) \quad \begin{cases} \frac{dx}{dt} = rx \left(1 - \frac{x(t-t_1)}{K}\right) - \frac{\lambda xy}{1+\epsilon y} - \alpha_1 U^* x \\ \frac{dy}{dt} = \frac{\lambda xy}{1+\epsilon y} - (\mu + d + \sigma)y - \frac{ay^2}{1+by^2} - \alpha_2 U^* y \end{cases}$$

Let us denote $\lambda = \lambda^*$ as bifurcation parameter and J^* as Jacobian matrix at $R_0 = 1$. which is as

follows:

$$(J^*) = \begin{bmatrix} -(r - \alpha_1 U^*) & \frac{-\lambda^* K(r - \alpha_1 U^*)}{r} \\ 0 & 0 \end{bmatrix}$$

let $v = [v_1, v_2]^T$ be the right and $u = [u_1, u_2]$ be the left eigenvector of jacobian corresponding to 0 eigenvalue. Then we have $u_1 = 0, u_2 = 1, v_1 = \frac{-\lambda^* K}{r}, v_2 = 1$

Now we evaluate the nonzero partial derivatives associated with the functions of system (4) at R_0 and $\lambda = \lambda^*$

$$\begin{aligned} \left(\frac{\partial^2 f_2}{\partial x \partial y}\right)_E &= \lambda^*, \quad \left(\frac{\partial^2 f_2}{\partial y \partial x}\right)_E = \lambda^* \\ \left(\frac{\partial^2 f_2}{\partial y^2}\right)_E &= -2 \left(\frac{K \varepsilon \lambda^* (r - \alpha_1 U^*)}{r} + a\right), \quad \left(\frac{\partial^2 f_2}{\partial x_2 \partial \lambda^*}\right)_E = \frac{K(r - \alpha_1 U^*)}{r} \end{aligned}$$

Then the bifurcation constants p_1 and q_1 from Castillo-Chavez and Song's [6] theorem 4.1, are as follows:

$$p_1 = v_1^2 \frac{\partial^2 f_2}{\partial x^2}(E, \lambda^*) + 2v_1 v_2 \frac{\partial^2 f_2}{\partial x \partial y}(E, \lambda^*) + v_2^2 \frac{\partial^2 f_2}{\partial y^2}(E, \lambda^*),$$

$$p_1 = \frac{\lambda^{*2} K^2}{r^2} \cdot 0 + 2 \cdot \frac{-K \lambda^*}{r} \cdot \lambda^* + 1 \cdot 1 \cdot \frac{-2b \lambda^* K(r - \alpha_1 U^*)}{r} - 2a$$

$$\begin{aligned} p_1 &= \frac{-2K \lambda^{*2}}{r} - \frac{2\varepsilon \lambda^* K(r - \alpha_1 U^*)}{r} - 2a \\ &= -2 \left(\frac{K \lambda^{*2}}{r} + \frac{K \varepsilon \lambda^* (r - \alpha_1 U^*)}{r} + a \right) < 0. \end{aligned}$$

and

$$q_1 = v_1 \frac{\partial^2 f_2}{\partial x \partial \lambda^*}(E, \lambda^*) + v_2 \frac{\partial^2 f_2}{\partial y \partial \lambda^*}(E, \lambda^*)$$

$$q_1 = \frac{K(r - \alpha_1 U^*)}{r} > 0$$

Since q_1 is positive and p_1 is negative, the forward bifurcation occurs at E when R_0 crosses unity.

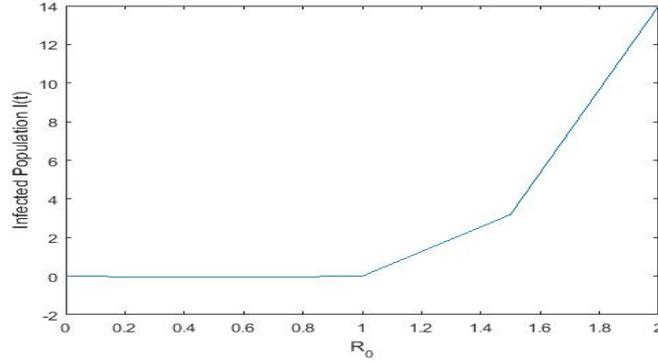


FIGURE 2. The system exhibits forward bifurcation at $R_0 = 1$ in (R_0, I) plane.

4. ENDEMICITY OF DISEASE

In epidemiology, endemicity of the infection and existence of the host populace are the two faces of persistence of infectious disease [8]. In our model (2), the disease persists in the system when $(R_0 > 1)$.

In this segment, we focus on the existence of E^* of (2) that is when disease persist in the system. Let endemic equilibrium point be denoted by $E^* = (X^*, Y^*)$. Substitute $\frac{dX}{dt} = 0$ to find the value

of X^* , which is: $X^* = K \left(1 - \frac{\lambda I}{r(1+\epsilon I)} - \frac{\alpha_1 U^*}{r} \right)$

Next substitute the value of X^* in the equation $\frac{dY}{dt} = 0$ to find the value of Y^* at $t_1 = 0$ and we get,

$$(5) \quad g_4 I^{*4} + g_3 I^{*3} + g_2 I^{*2} + g_1 I^* + g_0 = 0,$$

where

$$g_4 = r\epsilon^2 b(\delta + \alpha_2 U^*)$$

$$g_3 = K\epsilon\lambda b(\alpha_1 U^* - r) + K\lambda^2 b + 2r\epsilon b(\delta + \alpha_2 U^*) + r\epsilon^2 a$$

$$g_2 = rb(\delta + \alpha_2 U^*)(1 - R_0) + r\epsilon^2(\delta + \alpha_2 U^*) + 2ra\epsilon$$

$$g_1 = K\lambda^2 + 2r\epsilon(\delta + \alpha_2 U^*) + ra + K\lambda\epsilon(\alpha_1 U^* - r)$$

$$G_0 = r(\delta + \alpha_2 U^*)(1 - R_0)$$

Theorem 4: In the system, when R_0 is greater than unity, there is either a unique or there are three endemic equilibria which are non-negative. If all equilibria are simple roots and if R_0 is

less than unity then the endemic equilibrium does not exist in the system.

Proof: Let $R_0 > 1$. The coefficient g_4 is non-negative and g_0 is negative when $R_0 > 1$. From (5), we have the following polynomial:

$$Q(Y) := g_0 + g_1Y + g_2Y^2 + g_3Y^3 + g_4Y^4 = 0.$$

Then for g_1, g_2 , and g_3 , there exists the following possibilities of the signs:

- (1) H1 : $g_1 > 0, g_2 > 0$, and $g_3 > 0$,
- (2) H2 : $g_1 < 0, g_2 < 0$, and $g_3 > 0$,
- (3) H3 : $g_1 < 0, g_2 > 0$, and $g_3 > 0$,
- (4) H4 : $g_1 < 0, g_2 < 0$, and $g_3 < 0$,
- (5) H5 : $g_1 > 0, g_2 > 0$, and $g_3 < 0$,
- (6) H6 : $g_1 > 0, g_2 < 0$, and $g_3 > 0$,
- (7) H7 : $g_1 > 0, g_2 < 0$, and $g_3 < 0$,
- (8) H8 : $g_1 < 0, g_2 > 0$, and $g_3 < 0$.

Then, by Descartes' rule [22], $Q(Y)$ can have either three positive roots or $Q(Y)$ can have a unique root. There exists unique endemic equilibrium point, if any of the H1–H4 holds, whereas H5–H8 conditions must satisfy, for the existence of three endemic equilibria. For $R_0 < 1$ it is easy to check that all the coefficients are greater than zero therefore, endemic equilibrium point does not exist in the system. Also when $R_0 = 1$, the coefficient g_0 is zero and other coefficients g_1, g_2, g_3 and g_4 all are positive, then by fundamental theorem of algebra, $Q(Y)$ can not have real root which is non-negative. Hence for $R_0 \leq 1$, system (2) will not have any endemic equilibrium point however, when $R_0 > 1$ endemic equilibrium exists. Hence, we have the theorem.

For our paper, we will consider the case where any of H1–H4 holds, and $R_0 > 1$, then the system (2) admits a unique endemic equilibrium.

4.1. Local stability of Endemic Equilibrium Point. From the previous section, it was noted that the system has unique $E^*(X^*, Y^*)$ which is greater than zero. Now the local stability of E^* will be analyzed.

Characteristic equation of system at E^* is as follows:

$$\begin{vmatrix} -\frac{rX^*}{K}e^{-\zeta t_1} & \frac{-\lambda X^*}{(1+\varepsilon Y^*)^2} \\ \frac{\lambda Y^*}{1+\varepsilon Y^*} & \frac{\lambda X^*}{(1+\varepsilon Y^*)^2} - \delta - \alpha_2 U^* - \frac{2aY^*}{(1+bY^2)^2} \end{vmatrix} = 0$$

where $\delta = (\mu + d + \sigma)$.

The second degree transcendental equation is

$$(6) \quad \zeta^2 + m_0 \zeta + n_0 + (m_1 \zeta + n_1)e^{-\zeta t_1} = 0$$

where

$$\begin{aligned} m_0 &= \delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} - \frac{\lambda X^*}{(1+\varepsilon Y^*)^2}, \\ n_0 &= \frac{\lambda^2 X^* Y^*}{(1+\varepsilon Y^*)^3}, \\ m_1 &= \frac{rX^*}{K}, \\ n_1 &= \frac{rX^*}{K} \left(\delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} - \frac{\lambda X^*}{(1+\varepsilon Y^*)^2} \right) \end{aligned}$$

Theorem 5: E^* is locally stable for $t_1 = 0$, if $K\lambda Y^* \geq rX^*(1+\varepsilon Y^*)$ and $r(1+\varepsilon Y^*) \geq K\lambda$ are both simultaneously satisfied.

Proof: The characteristic equation at E^* and $t_1 = 0$ is given by

$$\zeta^2 + m_0 \zeta + n_0 + (m_1 \zeta + n_1)e^{-\zeta t_1} = 0$$

It is easy to see that if $m_0 + m_1 > 0$ and $n_0 + n_1 > 0$ then both the roots of the above equation will be less than zero (i.e. negative) and hence, E^* will be locally stable. Consider,

$$m_0 + m_1 = \delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} + \frac{-\lambda X^* K + rX^*(1+\varepsilon Y^*)^2}{K(1+\varepsilon Y^*)^2}$$

Thus, if $r(1+\varepsilon Y^*) \geq K\lambda$ then $m_0 + m_1 > 0$. Also,

$$n_0 + n_1 = \frac{K\lambda^2 X^* Y^* - \lambda rX^{*2}(1+\varepsilon Y^*)}{K(1+\varepsilon Y^*)^3} + \frac{rX^*}{K} \left(\delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} \right)$$

Thus, if $K\lambda Y^* \geq rX^*(1+\varepsilon Y^*)$ then $n_0 + n_1 > 0$. Hence, the theorem.

Theorem 6: Let (E^*) of the system (2) exists for t_1 greater than zero, E^* is locally stable when M and N are both greater than zero, where M and N are given by (19) and (20) respectively.

Proof: Characteristic equation for t_1 greater than zero at E^* is given by

$$(7) \quad \zeta^2 + m_0\zeta + n_0 + (m_1\zeta + n_1)e^{-\zeta t_1} = 0$$

Let $\zeta = \iota\omega, \omega > 0$ be the solution of characteristic equation (7), now let us first substitute $\zeta = \iota\omega$ in equation (7), we get

$$(8) \quad -\omega^2 + n_0 + m_1\omega \sin \omega t_1 + n_1 \cos \omega t_1 + \iota(m_1\omega \cos \omega t_1 - n_1 \sin \omega t_1 + m_0\omega) = 0$$

Separating the imaginary part and the real part of equation (8), we get

$$(9) \quad m_1\omega \sin \omega t_1 + n_1 \cos \omega t_1 = \omega^2 - m_0,$$

$$(10) \quad m_1\omega \cos \omega t_1 - n_1 \sin \omega t_1 = -m_0\omega,$$

Now squaring the above equations and then adding them, we get:

$$(11) \quad \omega^4 + ((m_0)^2 - 2n_0 - (m_1)^2)\omega^2 + ((n_0)^2 - (n_1)^2) = 0$$

Letting $\omega^2 = s$ the above equation changes to

$$(12) \quad s^2 + Ms + N = 0,$$

where $M = (m_0^2 - 2n_0 - m_1^2)$ and $N = (n_0^2 - n_1^2)$.

$$M = (m_0^2 - 2n_0 - m_1^2) = \left(\delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} - \frac{\lambda X^*}{(1+\epsilon Y^*)^2} \right)^2 - 2 \left(\frac{\lambda^2 X^* Y^*}{(1+\epsilon Y^*)^3} \right) - \frac{r^2 X^{*2}}{K^2}$$

$$N = (n_0^2 - n_1^2) = \left(\frac{\lambda^2 X^* Y^*}{(1+\epsilon Y^*)^3} \right)^2 - \left(\frac{rX^*}{K} \left(\delta + \alpha_2 U^* + \frac{2aY^*}{(1+bY^{*2})^2} - \frac{\lambda X^*}{(1+\epsilon Y^*)^2} \right) \right)^2 > 0$$

By Routh-Hurwitz Criterion, if $M > 0, N > 0$ are satisfied simultaneously then the equation (7) will always consists roots having real part which is negative. Hence, endemic equilibrium of the system (2) is locally stable for t_1 greater than zero.

5. HOPF BIFURCATION ANALYSIS

Here, we study the existence of Hopf Bifurcation. Characteristic equation at E^* is as follows:

$$(13) \quad \zeta^2 + m_0\zeta + n_0 + (m_1\zeta + n_1)e^{-\zeta t_1} = 0$$

There exists unique positive s_0 satisfying equation (12) if $T < 0$ i.e., a purely imaginary pair of roots $\pm s_0$ exists to equation (13).

The t_{1j} corresponding to s_0 can be obtained from equations (9) and (10) is as follows:

$$t_{1j} = \frac{1}{s_0} \left\{ \arccos \left(\frac{(n_0 - m_0 m_1) s_0^2 - n_0 n_1}{m_1^2 s_0^2 + n_1^2} \right) + 2j\pi \right\}$$

where $j = 0, 1, 2, \dots$

For $t_1 < t_0$, E^* is stable if $\frac{d}{dt_1}(Re(\zeta))|_{\zeta=ts_0} > 0$

Thus, we have the theorem as:

Theorem 7: When $t_1 \in (0, t_0)$, positive equilibrium of the system (2) is locally stable and when $t_1 > t_0$, it is unstable, that is, the system will undergo hopf bifurcation at the positive equilibrium when $t_1 = t_0$.

Proof: Differentiating equation (13) with respect to t_1 we get,

$$(14) \quad \left(2\zeta + m_0 m_1 e^{-\zeta t_1} - (m_1 \zeta + n_1) t_1 e^{-\zeta t_1} \right) \frac{d\zeta}{dt_1} = \zeta (m_1 \zeta + n_1) e^{-\zeta t_1}$$

$$\begin{aligned} \left(\frac{d\zeta}{dt_1} \right)^{-1} &= \frac{2\zeta + m_0 + m_1 e^{-\zeta t_1} - (m_1 \zeta + n_1) t_1 e^{-\zeta t_1}}{\zeta (m_1 \zeta + n_1) e^{-\zeta t_1}} \\ &= \frac{2\zeta + m_0}{\zeta (m_1 \zeta + n_1) e^{-\zeta t_1}} + \frac{m_1}{\zeta (m_1 \zeta + n_1) e^{-\zeta t_1}} - \frac{t_1}{\zeta} \\ &= \frac{2\zeta + m_0}{-\zeta (\zeta^2 + m_0 \zeta + n_0)} + \frac{m_1}{\zeta (m_1 \zeta + n_1) e^{-\zeta t_1}} - \frac{t_1}{\zeta} \end{aligned}$$

Now $\frac{d}{dt_1}(Re(\zeta))^{-1}|_{\zeta=ts_0} = Re \left(\frac{d\zeta}{dt_1} \right)^{-1} |_{\zeta=ts_0}$

$$\begin{aligned} &= Re \left(\frac{2ts_0 + m_0}{-ts_0(-s_0^2 + tm_0s_0 + n_0)} + \frac{m_1}{ts_0(tm_1s_0 + n_1)} - \frac{t_1}{ts_0} \right) \\ &= Re \left(\frac{1}{s_0} \left(\frac{2ts_0 + m_0}{(s_0^2 - m_0)t + m_0s_0} + \frac{m_1}{-m_1s_0 + tn_1} + tt_1 \right) \right) \end{aligned}$$

$$\begin{aligned}
&= \frac{1}{s_0} \left(\frac{2s_0(s_0^2 - n_0) + m_0^2 s_0}{(s_0^2 - n_0)^2 + (m_0 s_0)^2} - \frac{m_1^2 s_0}{(m_1 s_0)^2 + n_1^2} \right) \\
&= \frac{2s_0^2 + (m_0^2 - 2n_0 - m_1^2)}{(m_1 s_0)^2 + n_1^2} (\because (s_0^2 - n_0)^2 + (m_0 s_0)^2 = (m_1 s_0)^2 + n_1^2)
\end{aligned}$$

Hence, by following the condition $m_0^2 - 2n_0 - m_1^2 > 0$, we have $\frac{d}{dt_1}(Re(\zeta))^{-1}|_{\zeta=i s_0} > 0$.

Therefore the condition of transversality holds and at $t_1 = t_0$, hopf bifurcation occurs.

6. DIRECTION AND STABILITY OF HOPF BIFURCATION

In the past segment, we got certain conditions under which the given system goes under Hopf-Bifurcation, where time delay is considered as the being the critical parameter. In this part, by taking into account the normal form theory and the center manifold theorem, we will introduce the formula for deciding the direction of Hopf-Bifurcation and will get conditions for the stability of bifurcating periodic solutions, too. Since Hopf-Bifurcation happens at t_1' of t_1 , there exists a pair of pure imaginary root $\pm i \sigma(t_1')$ of the characteristic equation.

Next let, $e_1 = X - X^*$, $e_2 = Y - Y^*$ (where X^*, Y^* are the endemic equilibrium point).

Thus, the given system of equations gets transformed into the following system:

$$\begin{aligned}
\frac{de_1}{dt} &= \kappa_1 e_1(t) + \kappa_2 e_2(t) + \kappa_3 e_1(t - t_1) + \kappa_4 e_1(t) e_2(t) + \kappa_5 e_2(t) e_1(t) + \kappa_6 e_1(t) e_1(t - t_1) + \\
&\quad \kappa_7 e_1(t - t_1) e_1(t) \\
\frac{de_2}{dt} &= \kappa_8 e_1(t) + \kappa_9 e_2(t) + \kappa_{10} e_1(t) e_2(t) + \kappa_{11} x_2^2(t)
\end{aligned}$$

where,

$$\begin{aligned}
\kappa_1 &= r - \frac{rX^*}{K} - \frac{\lambda Y}{1+\varepsilon Y} - \alpha_1 U^*, \quad \kappa_2 = \frac{-\lambda X}{1+\varepsilon Y}, \quad \kappa_3 = \frac{-rX^*}{K}, \quad \kappa_4 = \frac{\lambda}{(1+\varepsilon Y)^2}, \quad \kappa_5 = \frac{-\lambda}{1+\varepsilon Y}, \quad \kappa_6 = \frac{-r}{K}, \\
\kappa_7 &= \frac{-r}{K}, \quad \kappa_8 = \frac{\lambda Y}{(1+\varepsilon Y)}, \quad \kappa_9 = \frac{-(\mu+d+\sigma+\alpha_2 U^*)(a+bY^2)^2(1+\varepsilon Y)^2}{(1+\varepsilon Y)^2+(1+bY^2)^2}, \quad \kappa_{10} = \frac{-\lambda}{(1+\varepsilon Y)^2}, \quad \kappa_{11} = \frac{-2\lambda X \varepsilon}{(1+\varepsilon Y)^3} + \\
&\quad \frac{2aby^2(3by^2+2)-2a}{(1+by^2)^4}.
\end{aligned}$$

We also let $t \rightarrow t_1 t$, and $t_1 = t_1' + \mu$. Then, the system finally takes the form of an FDE in $C = C([-1, 0], R^2)$ as:

$$(15) \quad \dot{e}(t) = M_\mu(e_t) + N(\mu, e_t)$$

where $e(t) = (e_1(t), e_2(t)(t))^T \in R^2$ and $M_\mu : C \rightarrow R^2$, $N : C \times R \rightarrow R^2$ are given respectively by:

$$(16) \quad M_\mu(\psi) = M_1 \psi(0) + M_2 \psi(-t_1)$$

and

$$N(\mu, \psi) = (t_1' + \mu)N_1$$

where,

$$M_1 = \begin{bmatrix} \kappa_1 & \kappa_2 \\ \kappa_8 & \kappa_9 \end{bmatrix}$$

and,

$$M_2 = \begin{bmatrix} \kappa_3 & 0 \\ 0 & 0 \end{bmatrix}$$

and,

$$N_1 = \begin{bmatrix} \kappa_4 \psi_1(0) \psi_2(0) \\ \kappa_{10} \psi_1(0) \psi_2(0) \end{bmatrix}$$

We also have that, $\psi = (\psi_1, \psi_2)^T \in C$, and $e_t(\varrho) = e(t + \varrho)$ for $\varrho \in [-t_1, 0]$.

By the Riesz Representation theorem, there exists a function $\eta(\varrho, \mu)$ of bounded variation for $\varrho \in [-t_1, 0]$, such that

$$(17) \quad M_\mu(\psi) = \int_{-t_1}^0 d\eta(\varrho, \mu) \psi(\varrho)$$

This equation holds for $\psi \in C$.

In fact, we can take

$$\eta(\varrho, \mu) = \mu M_1 \delta(\varrho) + \mu M_2 \delta(\varrho + t_1)$$

where, M_1, M_2 have already been given above, and $\delta(\varrho)$ is Dirac delta function.

Next, for $\psi \in C^1([-t_1, 0], R^2)$, we define the following :

$$A(\mu)\psi = \begin{cases} \frac{d\psi(\varrho)}{d\varrho}, & \varrho \in [-t_1, 0) \\ \int_{-t_1}^0 d\eta(s, \mu) \psi(s), & \varrho = 0 \end{cases}$$

and,

$$R(\mu)\psi = \begin{cases} 0, & \varrho \in [-t_1, 0) \\ N(\mu, \psi), & \varrho = 0 \end{cases}$$

Then, the system is equivalent to,

$$(18) \quad \dot{e}_t = A(\mu)e_t + R(\mu)e_t$$

where, $e_t(\vartheta) = e(t + \vartheta)$ for $\vartheta \in [-t_1, 0]$.

Next, for $\varphi \in C^1([0, 1], R^2)$, the adjoint operator A^* of A can be defined as,

$$A^* \varphi(s) = \begin{cases} \frac{-d\varphi(s)}{ds}, & s \in (0, t_1] \\ \int_{-t_1}^0 d\eta^T(t, 0)\varphi(-t), & s = 0 \end{cases}$$

and hence for $\psi \in ([-t_1, 0], R^2)$, $\varphi \in ([0, t_1], R^2)$ a bilinear inner product, in order to normalize the eigenvalues of A and A^* can be defined as follows:

$$(19) \quad \langle \varphi(s), \psi(\vartheta) \rangle = \bar{\varphi}(0)\psi(0) - \int_{-t_1}^0 \int_{s=0}^{\vartheta} \bar{\varphi}(s - \vartheta) d\eta(\vartheta) \psi(s) ds$$

where $\eta(\vartheta) = \eta(\vartheta, 0)$, and $\bar{\varphi}$ is the complex conjugate of φ . It can be verified that the operators A and A^* are adjoint operators with respect to this bilinear form. Thus, since $\pm i\sigma' t_1'$ are eigenvalues of $A(0)$, they are the eigenvalues of A^* as well.

Suppose that $b \in C^1$ is an eigenvector of $A(0)$ associated with $i\omega_0$ such that $b(\vartheta) = (1, b_2)^T e^{i\omega_0 \vartheta}$. Then $A(0)b(\vartheta) = i\omega_0 b(\vartheta)$ for all $\vartheta \in [-t_1, 0]$. When $\vartheta = 0$, we can use (16) and (17) to obtain

$$M_1 b(0) + M_2 b(-t_1, *) = i\omega_0 b(0)$$

which yields

$$b_2 = \frac{\kappa_8}{i\omega_0 - \kappa_9}$$

Similarly, suppose that the eigenvector, b^* of A^* corresponding to $-i\omega_0$ takes the form $b^*(s) = \frac{1}{\bar{D}}(1, b^*)^T e^{i\omega_0 s}$. Then,

$$M_1 b^*(0) + M_2 b^*(-t_1, *) = i\omega_0 b^*(0)$$

which yields

$$b_2^* = \frac{-\kappa_2}{\kappa_9 + i\omega_0}$$

Now, in order to ensure that $\langle b^*, b \rangle = 1$, we will determine the value \bar{D} . By (19) we have,

$$\langle b^*, b \rangle = \bar{b}^{*T}(0)b(0) \int_{\varrho=-t_1^*}^0 \int_{s=0}^{\varrho} \bar{b}^{*T}(s-\varrho) d\eta(\varrho)b(s) ds$$

$$\text{Solving it we get, } \langle b^*, b \rangle = \frac{1}{\bar{D}} [1 + b_2 \bar{b}_2^* + t_1^* e^{-i\omega_0 t_1^*} \kappa_3]$$

Hence,

$$\bar{D} = 1 + b_2 \bar{b}_2^* + t_1^* e^{-i\omega_0 t_1^*} \kappa_3$$

Therefore, we obtain $\langle b^*, b \rangle = 1$

In the remaining part of this section, we now compute the coordinates in order to describe the center manifold C_0 at $\mu = 0$. Let e_t be the solution of (18) when $\mu = 0$.

Next, define

$$(20) \quad \tilde{z}(t) = \langle b^*, e_t \rangle, W(t, \varrho) = e_t - 2\text{Re}[\tilde{z}(t)b(\varrho)]$$

Now, on the center manifold C_0 , we have

$$W(t, \varrho) = W(\tilde{z}(t), \bar{\tilde{z}}(t), \varrho) = W_{20}(\varrho) \frac{\tilde{z}^2}{2} + W_{11}(\varrho) \tilde{z} \bar{\tilde{z}} + W_{02}(\varrho) \frac{\bar{\tilde{z}}^2}{2} + \dots$$

where \tilde{z} and $\bar{\tilde{z}}$ are local coordinates for the center manifold C_0 in the direction of b^* and \bar{b}^* . We note that W is real if e_t is real and we will be considering the real solutions only.

From (20) we have,

$$(21) \quad \begin{aligned} \dot{\tilde{z}}(t) &= \langle b^*, \dot{e}_t \rangle \\ &= \langle b^*, A(0)e_t + R(0)e_t \rangle \\ &= \langle A^* b^*, e_t \rangle + \langle \bar{b}^{*T}(0) f_0(z, \bar{z}) \rangle \end{aligned}$$

where, $f_0(z, \bar{z}) = F(0, W(z, \bar{z}, \varrho))$

Therefore, we can write (21) as,

$$(22) \quad \dot{z}(t) = i\omega_0 z(t) + g(z, \bar{z})$$

with,

$$(23) \quad g(\tilde{z}, \bar{\tilde{z}}) = \bar{b}^{*T}(0) N_0(\tilde{z}, \bar{\tilde{z}}) = g_{20} \frac{\tilde{z}^2}{2} + g_{11} \tilde{z} \bar{\tilde{z}} + g_{02} \frac{\bar{\tilde{z}}^2}{2} + g_{21} \frac{\tilde{z}^2 \bar{\tilde{z}}}{2} + \dots$$

Substituting (18) and (21) into $\dot{W} = \dot{e}_t - \dot{z}b - \bar{\tilde{z}}b$, we have,

$$\dot{W} = \begin{cases} AW - 2\text{Re}[\bar{b}^{*T}(0) f_0(\tilde{z}, \bar{\tilde{z}}) b(\varrho)], & \varrho \in [-t_1, 0) \\ AW - 2\text{Re}[\bar{b}^{*T}(0) f_0(\tilde{z}, \bar{\tilde{z}}) b(\varrho)] + f_0(\tilde{z}, \bar{\tilde{z}}), & \varrho = 0 \end{cases}$$

Therefore, let

$$(24) \quad \dot{W} = AW + \tilde{H}(\tilde{z}, \bar{\tilde{z}}, \wp)$$

where,

$$\tilde{H}(\tilde{z}, \bar{\tilde{z}}, \wp) = \tilde{H}_{20}(\wp) \frac{\tilde{z}^2}{2} + \tilde{H}_{11}(\wp) \tilde{z} \bar{\tilde{z}} + \tilde{H}_{02}(\wp) \frac{\bar{\tilde{z}}^2}{2} + \dots$$

On the other hand, on the center manifold C_0 near the origin, $\dot{W} = W_{\tilde{z}} \dot{\tilde{z}} + W_{\bar{\tilde{z}}} \dot{\bar{\tilde{z}}}$

Using (5.10) to compare the coefficients, we finally arrive at the following,

$$(A - 2i\omega_0)W_{20}(\wp) = -\tilde{H}_{20}(\wp), AW_{11}(\wp) = -\tilde{H}_{11}(\wp), (A - 2i\omega_0)W_{02}(\wp) = -\tilde{H}_{02}(\wp)$$

Since, $e_t(t + \wp) = \tilde{W}(\tilde{z}, \bar{\tilde{z}}, \wp) + z + \bar{z}\bar{b}$, we have,

$$e_1(t + \wp) = ze^{i\omega_0\wp} + \bar{z}e^{-i\omega_0\wp} + W_{(20)}^{(1)}(\wp) \frac{z^2}{2} + W_{(11)}^{(1)}(\wp) z\bar{z} + W_{(02)}^{(1)}(\wp) \frac{\bar{z}^2}{2}$$

$$e_2(t + \wp) = zb_2e^{i\omega_0\wp} + \bar{z}\bar{b}_2e^{-i\omega_0\wp} + W_{(20)}^{(2)}(\wp) \frac{z^2}{2} + W_{(11)}^{(2)}(\wp) z\bar{z} + W_{(02)}^{(2)}(\wp) \frac{\bar{z}^2}{2}$$

and thus,

$$f_0(z, \bar{z}) = \begin{bmatrix} S_{11}z^2 + S_{12}z\bar{z} + S_{13}\bar{z}^2 + S_{14}z^2\bar{z} \\ S_{12}z^2 + S_{22}z\bar{z} + S_{23}\bar{z}^2 + S_{24}z^2\bar{z} \end{bmatrix}$$

where,

$$S_{11} = \kappa_4 b_2$$

$$S_{12} = \kappa_4 (b_2 + \bar{b}_2)$$

$$S_{13} = \kappa_4 \bar{b}_2$$

$$S_{14} = \kappa_4 (W_{(11)}^{(2)}(0) + W_{(11)}^{(1)}(0)b_2)$$

$$S_{21} = \kappa_{10} b_2$$

$$S_{22} = \kappa_{10} (b_2 + \bar{b}_2)$$

$$S_{23} = \kappa_{10} \bar{b}_2$$

$$S_{24} = \kappa_{10} (W_{(11)}^{(2)}(0) + W_{(11)}^{(1)}(0)b_2)$$

Since $b^*(0) = \frac{1}{D}(1, b_2^*)^T$, we have

$$g(z, \bar{z}) = \bar{b}^*(0)^T f_0(z, \bar{z})$$

$$= \frac{1}{D}(1, \bar{b}_2) f_0(z, \bar{z})$$

$$= \frac{1}{D} [(K_{11} + K_{21}\bar{b}_2^*)z^2 + (K_{12} + K_{22}\bar{b}_2^*)z\bar{z} + (S_{13} + S_{23}\bar{b}_2^*)\bar{z}^2 + (S_{14} + S_{24}\bar{b}_2^*)z^2\bar{z}]$$

Comparing the coefficients of the above equations with (23), we get,

$$g_{20} = \frac{2}{D}(S_{11} + S_{21}\bar{b}_2^*)$$

$$g_{11} = \frac{1}{D}(S_{12} + S_{22}\bar{b}_2^*)$$

$$g_{02} = \frac{2}{D}(S_{13} + S_{23}\bar{b}_2^*)$$

$$g_{21} = \frac{1}{D}(S_{14} + S_{24}\bar{b}_2^*)$$

Following the procedure same as that of [16], we get,

$$W_{20}(\varphi) = \frac{ig_{20}}{\omega_0}b(o)e^{i\omega_0\varphi} + \frac{ig_{02}}{3\omega_0}\bar{b}q(o)e^{-i\omega_0\varphi} + E_1^{2i\omega_0\varphi}$$

$$W_{11}(\varphi) = -\frac{ig_{11}}{\omega_0}b(o)e^{i\omega_0\varphi} + \frac{ig_{11}}{\omega_0}\bar{b}(o)e^{-i\omega_0\varphi} + E_2$$

where E_1 and E_2 are the vectors such that,

$$(2i\omega_0 I_2 - \int_{-t_1^*}^0 e^{2i\omega_0\varphi} d\eta(\varphi))E_1 = (S_{11}, S_{21})^T,$$

$$(\int_{-t_1^*}^0 d\eta(\varphi))E_2 = -(S_{12}, S_{22})^T$$

$$J = \begin{pmatrix} \kappa_1 & \kappa_2 \\ \kappa_6 & \kappa_7 + \kappa_8 \end{pmatrix} \begin{pmatrix} E_2^{(1)} \\ E_2^{(2)} \end{pmatrix} = \begin{pmatrix} S_{12} \\ S_{22} \end{pmatrix}$$

and

$$J = \begin{pmatrix} 2i\omega_0 - \kappa_1 & -\kappa_2 \\ -\kappa_6 & 2i\omega_0 - \kappa_7 - \kappa_8 e^{2i\omega_0 t_1^*} \end{pmatrix} \begin{pmatrix} E_1^{(1)} \\ E_2^{(2)} \end{pmatrix} = - \begin{pmatrix} S_{11} \\ S_{21} \end{pmatrix}$$

where $E_1 = (E_1^{(1)}, E_1^{(2)})$, $E_2 = (E_2^{(1)}, E_2^{(2)})$ and I_n is the identity matrix. Therefore, it follows that,

Based on above, we can express g_{ij} in terms of the parameters in the system. Therefore we get the following terms,

$$\tilde{C}_1(0) = \frac{l}{2\omega_0}(g_{20}g_{11} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}) + \frac{g_{21}}{2},$$

$$\tilde{\mu}_2 = -\frac{Re\{\tilde{C}_1(0)\}}{Re\{\frac{d\lambda(t_1')}{dt_1}\}},$$

$$\tilde{\beta}_2 = 2Re\{\tilde{C}_1(0)\},$$

$$\tilde{T}_2 = -\frac{Im\{\tilde{C}_1(0)\} + \tilde{M}_2 Im\{\frac{d\lambda(t_1')}{dt_1}\}}{\omega_0},$$

where,

- (1) $\tilde{\mu}_2$ determines the direction of Hopf bifurcation, for if $\tilde{\mu}_2 > 0$, the Hopf bifurcation will be supercritical, and if $\tilde{\mu}_2 < 0$, the Hopf bifurcation will be subcritical, and the bifurcating periodic solutions exist for $t_1 > t_1'$ or $t_1 < t_1'$.
- (2) $\tilde{\beta}_2$ determines the stability of the bifurcating periodic solutions, for if $\tilde{\beta}_2 < 0$, the bifurcating periodic solutions will be stable, and if $\tilde{\beta}_2 > 0$, the bifurcating periodic solutions will be unstable.
- (3) \tilde{T}_2 determines the period of the bifurcating periodic solutions, for if $\tilde{T}_2 > 0$, the period increases, and if $\tilde{T}_2 < 0$, the period decreases.

7. NUMERICAL SIMULATION

To analyze the dynamical behaviour of the model, we have considered hypothetical and biologically feasible parametric values as given in Table 2. Using MATLAB, numerical simulations have been performed. We have assumed that $X(0) = 10$, $Y(0) = 10$, $Z(0) = 10$, $a = 0.1$ and $b = 0.007$.

Table 2. Parameters and Units

Parameters	Value	Unit
r	0.1	$person(day)^{-1}$
K	10	$person(day)^{-1}$
λ	0.0001	$(person)^{-1}(day)^{-1}$
ε	0.01	$(person)^{-1}(day)^{-1}$
μ	0.02	$(day)^{-1}$
d	0.002	$(day)^{-1}$
σ	0.02	$(day)^{-1}$
α_1	0.2	$(day)^{-1}$
α_2	0.1	$(day)^{-1}$

First, considering the above values of parameters, when $U^* = 0$ the value of disease-free equilibrium observed is $E(10.0000, 0.0000)$ and $R_0 = 0.0056$ (figure 1(a)). Similarly, when $U^* = 0.3$, the steady state value for disease-free equilibrium point is $E(7.0000, 0.0000)$ and $R_0 = 0.0238$ (figure 1(b)). From both the figures 2 and 3 it is noted that system is locally stable for E as stated in theorem (2). Also it can be seen that, if there is an increase in level of pollution

the disease-free equilibrium point is achieved at a lower value.

Next, when $U^* = 0$ and $\lambda = 0.1$ the steady state value for E^* is $(1.3038, 0.8772, 4.7047)$ and $R_0 = 23.8095$ (Figure 2(a)) and when $U^* = 0.3$ and $\lambda = 0.1$ the steady state value for E^* is $(1.5731, 0.5457, 0.8125)$ and $R_0 = 6.8627$ (figure 2(b)). For the above parameters the conditions of theorems (4) and (5) holds true and E^* is locally stable for all $t_1 \geq 0$ which can be verified from figures (2(a)) and (2(b)) respectively. Here, we notice that the susceptible population inclines with the incline in the level of pollution whereas infectives and recovered declines with the incline in the level of pollution. The increase in the susceptibles could be accounted because of the decrease in the infectives and therefore lesser number of infected are available for the transmission of the disease. But it can also be seen that with the increase in pollution level, overall population and the number of recovered also decreases. Hence, the level of pollution should be controlled for the survival and better recovery of the population.

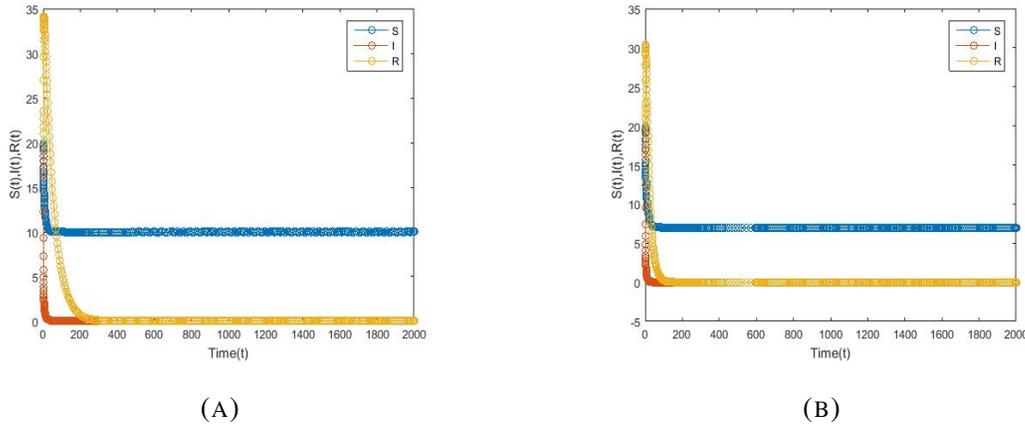


FIGURE 3. The local stability of E . (a) Disease-free equilibrium when $U^* = 0$; (b) Disease-free equilibrium when $U^* = 0.3$

From theorem (6) we know that E^* loses its stability and Hopf Bifurcation occurs at $t_1 = t_0$. It is observed that when $r = 6, \lambda = 0.9365, \alpha_1 = 0.9241, \alpha_2 = 0.6585, \alpha_3 = 0.7832, \mu = 0.0075, d = 0.0010, \sigma = 0.12$ and $U^* = 0$, E^* is stable for $t_1 \leq 0.9$ (Figure 3(a)) and undergoes Hopf bifurcation when $t_1 = 1$ (Figure 3(b)). Here, the steady-state values of E^* is calculated as $(1.6623, 11.4628, 574.8027)$. For $U^* = 0.3$, E^* is stable for $t_1 \leq 0.8$ (Figure 4(a)), Hopf bifurcation occurs at $t_1 = 0.9$ (Figure 4(b)) and E^* is calculated as $(1.8993, 9.5778, 27.7901)$.

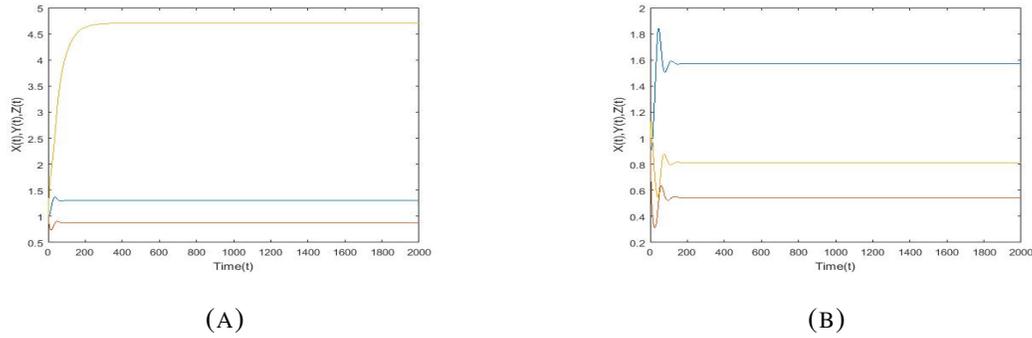


FIGURE 4. The local stability of E^* . (a) Endemic equilibrium when $U^* = 0$; (b) Endemic equilibrium when $U^* = 0.3$

It can readily seen that with the increase in pollution the recovered population decreases, also the logistic delay decreases for which the system is stable. This may be due to the fact that due to presence of pollutants the reproductive capacities of the exposed population is affected. Therefore, it can be said that pollution effects the reproductive function as well as the overall health of the individuals, so awareness should be increased and appropriate measures should be taken accordingly.

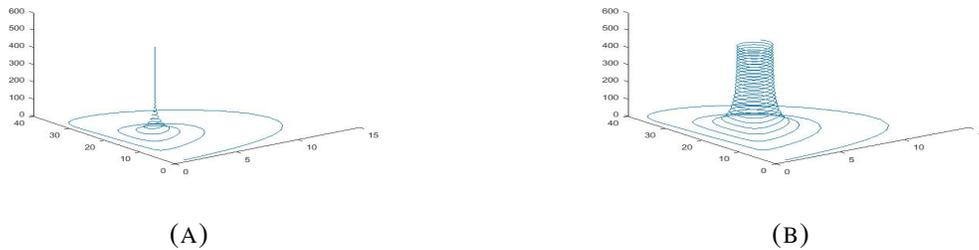


FIGURE 5. Hopf Bifurcation. (a) The system is stable when $U^* = 0$ and $t_1 \leq 0.9$; (b) The system is unstable when $U^* = 0$ and $t_1 = 1$.

Now we study the role of treatment when the system is under pollution by varying the values of cure rate a and following observations were made:

Parameters	a	$X(t)$	$Y(t)$	$Z(t)$
$U^* = 0$	0.1	1.3038	0.8772	4.7047
$U^* = 0.3$	0.1	1.5731	0.5457	0.8125
$U^* = 0.3$	0.5	3.0251	0.3991	1.7504



FIGURE 6. Hopf Bifurcation. (a) The system is stable when $U^* = 0.3$ and $t_1 \leq 0.8$; (b) The system is unstable when $U^* = 0.3$ and $t_1 = 0.9$

From figures (5) and (6) it can be seen that increase in a diminishes the spread of diseases. From the above table it is evident that with the incline in pollution, it results in decline of the recovered populace but if we increase a i.e. the cure rate, the recovered populace increases. Hence, in the dynamics of the model, treatment plays a vital role but this may result in over usage of the resources and is not economically feasible and hence, we need to control the pollution.

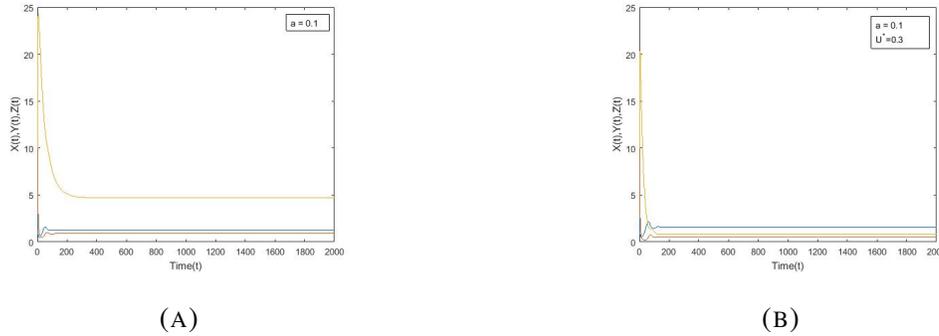


FIGURE 7. Effect of treatment. (a) $a = 0.1$ and $U^* = 0$; (b) $a = 0.1$ and $U^* = 0.3$.

8. CONCLUSION

In our study, an epidemic SIR model has been developed. The model includes time delay in the growth component, saturated type incidence rate along with Holling type III treatment rate. We have also included the effect of pollution in our model. Using R_0 , it was noted that E is locally stable when $R_0 < 1$ which indicates that the infection is no longer present in the environment at $R_0 < 1$. It was also noted that E is unstable when $R_0 > 1$, which clearly indicates

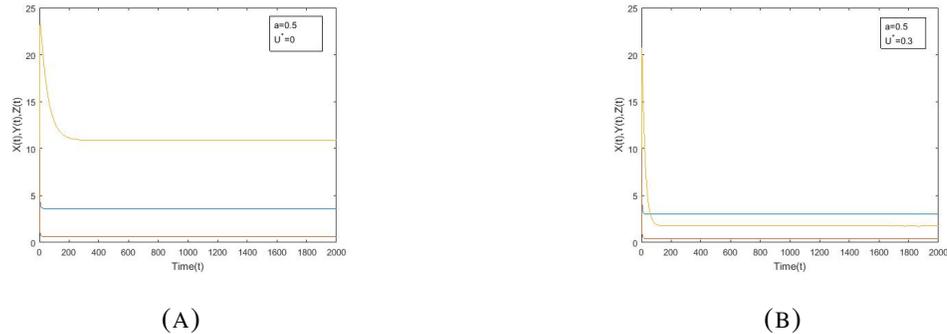


FIGURE 8. Effect of treatment. (a) $a = 0.5$ and $U^* = 0$; (b) $a = 0.5$ and $U^* = 0.3$.

that infection exists in the environment at R_0 greater than one. Using center manifold theory, it was shown that at $R_0 = 1$, system (2) undergo forward bifurcation. We have investigated that for time lag $t_1 \geq 0$, E^* i.e. the endemic equilibrium point is locally stable if conditions stated in theorem 4 and 5 holds true. Furthermore, hopf bifurcation at E^* has also been discussed. Through numerical simulations, we have observed that with the increase in the level of pollution we can see a decline in overall populace and in the recovered individuals. Also, it can be seen that the system is stable at lower value of logistic delay when the pollution increases, which is due to decrease in population size and perhaps may be due to the reason that reproduction capabilities of the population decreases. Thus, to remove the disease completely we need to find ways to reduce the level of pollution and choose favourable techniques to expand the capacity of treatment.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

REFERENCES

- [1] A. Kumar, Nilam, Dynamical model of epidemic along with time delay; Holling type II incidence rate and Monod–Haldane type treatment rate, *Differ. Equ. Dyn. Syst.* 27 (2019), 299-312.
- [2] A. Kumar, Nilam, Mathematical analysis of a delayed epidemic model with nonlinear incidence and treatment rates, *J. Eng. Math.* 115 (2019), 1-20.

- [3] A. Kumar, Nilam, Nonlinear dynamics of a time-delayed epidemic model with two explicit aware classes, saturated incidences, and treatment, *Nonlinear Dyn.* 101 (2020), 1693-1715.
- [4] A. Kumar, Nilam, Stability of a time delayed SIR epidemic model along with nonlinear incidence rate and Holling type-II treatment rate, *Int. J. Comput. Meth.* 15 (2018), 1850055.
- [5] B. Pimpunchat, W.L. Sweatman, G.C. Wake, W. Triampo, A. Parshotam, A mathematical model for pollution in a river and its remediation by aeration, *Appl. Math. Lett.* 22 (2009), 304-308.
- [6] C. Castillo Chavez, B. Song, Dynamical models of tuberculosis and their applications, *Math. Biosci. Eng.* 1 (2004), 361-404.
- [7] G.E. Hutchinson, Circular causal systems in ecology, *Ann. NY Acad. Sci.* 50 (1948), 221-246.
- [8] H.R. Thieme, Uniform Persistence and permanence of nonautonomous semiflows in population biology, *Math. Biosci.* 166 (2000), 173-201.
- [9] J. Li, Z. Teng, G. Wang, L. Zhang, C. Hu, Nonlinear science, and nonequilibrium and complex phenomena, *Chaos Solitons Fractals*, 99(2017), 63–71.
- [10] K. Ghosh, S. Biswas, S. Samanta, P.K. Tiwari, A.S. Alshomrani, J. Chattopadhyay, Effect of multiple delays in an eco-epidemiological model with strong Allee effect, *Int. J. Bifurcation Chaos.* 27 (2017), 1750167.
- [11] K.P. Singh, A.A. Warsono, B.S. Bae, Mathematical modelling of environment data, *Math. Computer Model.* 33 (2001), 793-800.
- [12] M.F. Nia, F. Rezaei, Hartman-Grobman theorem for iterated function systems, *Rocky Mountain J. Math.* 49 (2019), 307-333.
- [13] P. Dubey, B. Dubey, U.S. Dubey, An SIR model with nonlinear incidence rate and Holling type III treatment rate, in: J.M. Cushing, M. Saleem, H.M. Srivastava, M.A. Khan, M. Merajuddin (Eds.), *Applied Analysis in Biological and Physical Sciences*, Springer India, New Delhi, 2016: pp. 63–81.
- [14] P.V.D. Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartment models of disease transmission, *Math. Biosci.* 180 (2002), 29-48.
- [15] S. Chauhan, S.K. Bhatia, S. Gupta, Effect of pollution on dynamics of SIR model with treatment, *Int. J. Biomath.* 08 (2015), 1550083.
- [16] S. Guo, H. Zheng, Q. Liu, Hopf bifurcation analysis for congestion control with heterogeneous delays, *Nonlinear Anal.: Real World Appl.* 11 (2010), 3077-3090.
- [17] S. Sastry, *Analysis, Stability and Control*, Springer-Verlag, New-York, (1999).
- [18] S. Tipsri, W. Chinviriyasit, Stability analysis of SEIR model with saturated incidence and time delay, *Int. J. Appl. Phys. Math.* 4 (2014), 42-45.
- [19] S.N. Singh, R. Mehta, Effect of environmental pollution on the growth and existence of biological populations involving H-function, *IOSR J. Math.* 1 (2012), 01-02.
- [20] W. Wang, Backward bifurcation of an epidemic model with treatment, *Math. Biosci.* 201(2006), 58-71.

- [21] W. Wang, S. Ruan, Bifurcation in an epidemic model with constant removal rate of infectives, *J. Math. Anal. Appl.* 291 (2004), 775-793.
- [22] X. Wang, A simple proof of descartes's rule of signs, *Amer. Math. Mon.* 111 (2004), 525-526.
- [23] X. Yang, L. Chen, J. Chen, Permanence and positive periodic solution for the single-species nonautonomous delay diffusive models, *Comput. Math. Appl.* 32 (1996), 09-116.
- [24] X. Zhang, X. Liu, Backward bifurcation of an epidemic model with saturated treatment, *J. Math. Anal. Appl.* 348 (2008), 433-443.