

Stability and Bifurcation Analysis of a Delayed SEIR Epidemic Model with Self-Protection

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Abstract: A delayed SEIR epidemic model with self-protection is considered. The local asymptotic stability of the disease free equilibrium is ensured for $R_0 < 1$ and it is unstable otherwise. Whereas, for $R_0 > 1$, the proposed model has the unique endemic equilibrium point. For delay values, the local asymptotic stability of the unique endemic equilibrium is established under some parametric constraints. Further, the occurrence of Hopf bifurcation is also observed when the delay crosses some parameter threshold. The analytical findings are supported by the numerical observations.

Keywords: Disease transmission, Basic reproduction numbers, Delay, Hopf-bifurcation.

1. Introduction

Researchers [1, 2, 3, 4, 5, 6] have designed different epidemiological models such as SI, SIS, SIR, SIER, SIERS etc. Kermack and McKendrick [7] formulated a SIR in 1927, to study the outbreak of the Great Plague in London during 1965-1966, and the outbreak of plague in Mumbai in 1906. The dynamic behavior of the SIR epidemic model is investigated by scholars [8, 9, 10, 11, 12]. The author [13] studied a vaccine induced epidemic SIRS model with natural immunities. They obtained that the system undergoes a backward bifurcation at a certain level of immunization. The SEIR model contains one more compartment additionally to SIR model called exposed compartment (E). These are the people who are infected but the symptoms of the disease are not yet visible. They cannot either communicate the disease and are in latent period. SEIR models can represent many human infectious diseases such as measles, pox, flu and dengue.

Recently, Diaz et al. [14] studied the modified SEIR model for the Ebola disease in Western Africa. They proposed several metrics to figure out the countries which are in most noteworthy need of extra resources to combat the contamination of infection spread. Sun et al. [15] studied the transmission and control dynamics of cholera disease. Mishra et al. [16] discussed the dynamics of bacteriophage infection in cholera disease in the region around a water body. Li [17] discussed the spread of hemorrhagic fever and showed that in China it exhibits monthly periodic outbreak.

For any community, the source of treatment for a disease is sometimes limited, so the constant removal rate which is discussed by Wang and Ruan [18] and further improved by Zhou and Fan [19] as a Holling type II treatment. Dubey et al. [20] studied the SEIR model with two different types of treatment rates. They observed that the existence and stability of equilibria depend on both the basic reproduction number as well as treatment rate. Further, Dubey et al. [21] introduced a model using Crowley-Martin and Holling type III responses to describe the epidemiological situation. Moreover, various epidemiological models have been studied using different treatment rates [22, 23, 24, 25].

In this manuscript, we consider an SEIR model with Crowley–Martin incidence rate and Holling type II and III treatment rates depending upon competency of the community. For any outbreak of epidemic disease, the treatment capacity of Holling type II is initially very slow and after that stage it develops gradually with change of accessibility of treatment assets such as effective medicines and hospital's conditions.

2. The Mathematical Model

Human infectious diseases namely measles, pox, flu and dengue as an important role are formed by the SEIR model. The mathematical structure of the generic SEIR epidemiological model is constructed. The incidence rate as Crowley-Martin (CM) type and treatment rate of infection via Holling type II is considered. The Crowley and Martin [26] functional responses are both prey and predator abundance because of predator interference. It is assumed that predator-feeding rate decreases by higher predator density even when prey density is high. Therefore, the effect of predator interference on the feeding rate remains important all the time whether an individual predator is handling or searching for a prey at a given instant of time. Recently, many researchers have studied the virus dynamics for models with Crowley-Martin infection rate (see [27, 28]).

In this article, consider a nonlinear Crowley-Martin incidence rate $\frac{\alpha SI}{(1+\beta S)(1+\gamma I)}$, which can be used to interpret the case of varicella (chickenpox) dynamic. Here α , β and γ are positive parameters that describe the effects of contact rate, social awareness rate among susceptibles and magnitude of interference among infective population, respectively.

In order to construct the model, our assumptions are stated as follows:

- The entire population N is divided into four groups as: S-susceptible, E-exposed, I-Infective and R-recovered population. So, $N = S + E + I + R$.
- Each population of SEIR well mixed and interact homogeneously with each other [29, 30].

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- The susceptible population is recruited at any time t at the constant rate A of new born and decreases due to natural death rate d .
- Disease is transmitted from infected to susceptible population by Crowley-Martin incidence rate

$$\frac{\alpha SI}{(1+\beta S)(1+\gamma I)} \quad (13)$$

The individuals from class S move to class E and only after latency period it becomes infective and move to class I and μ is the time delay due to latent period of the disease.

- The infected population is recovered by saturated treatment function $\frac{\alpha ul}{1+\delta ul}$ where u is the treatment control and a and δ are positive quantities, respectively [31].
- m is information induced self-protection from susceptible to removed class.
- The recovery is not permanent and m_1R become susceptible.
- d_1 is the disease related extra mortality rate of infective class.

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

$$\frac{ds}{dt} = A - \frac{\alpha SI}{(1 + \beta S)(1 + \gamma I)} - dS - mS + m_1R,$$

$$\frac{dE}{dt} = A - \frac{\alpha SI}{(1 + \beta S)(1 + \gamma I)} - dE - \mu E (t - \tau),$$

$$\frac{dl}{dt} = \mu E (t - \tau) - (d + d_1)I - \frac{\alpha ul}{(1 + \delta ul)},$$

$$\frac{dR}{dt} = \frac{\alpha ul}{(1 + \delta ul)} + mS - (d + m_1)R$$

(1)

With

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0 \quad (2)$$

3. Qualitative analysis of the system

3.1 Boundedness of the System

Theorem 1: All the solutions of the system (1) are bounded.

Proof: Consider the function $U(t) = S(t) + E(t) + I(t) + R(t)$.

Now using the equations (1), we have

$$\frac{dU}{dt} = \frac{ds}{dt} + \frac{dE}{dt} + \frac{dl}{dt} + \frac{dR}{dt}$$

$$= A - dS - dE - (d + d_1)I - dR$$

If we take $\eta = \min \{d, d + d_1\}$, then for each $\eta \geq 0$ the above inequality becomes

$$\frac{dU}{dt} + \eta U \leq A$$

Now by the theory of differential inequality [32] we have,

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

As $t \rightarrow \infty$, then $0 \leq U(t) \leq \frac{A}{\eta}$. Hence $U(t)$ is a bounded quantity.

Thus all the solutions of the system (1) are bounded.

3.2 Basic reproduction number

We observe that the system (1) has a disease free equilibrium (DFE) $E_1(S_1, 0, 0, R_1)$, where

$$S_1 = \frac{A(d + m_1)}{(d + m)(d + m_1) - mm_1}, R_1 = \frac{A}{(d + m)(d + m_1) - mm_1}$$

and it exist if $(d + m)(d + m_1) > mm_1$.

Introducing the basic reproduction number by R_0 and is defined as the number secondary infected individuals caused by a single infected individuals during the whole time of period. To find the expression for R_0 , using the technique which is introduced by Driessche and Watmough [33].

The system (1) can be written as

$$\frac{dX}{dt} = F(X) - G(X),$$

where $X = (S, E, I, R)^T$, $F(X) = [\alpha SI \ 0]$,

$$G(X) = \left[(d + \mu)E(d + d_1) + \frac{\alpha ul}{1 + \delta ul} - \mu I \right]$$

F_1 = Jacobian of F at (DFE)=

$$\begin{bmatrix} 0 & \frac{\alpha S_0}{1 + \beta S_0} & 0 & 0 \end{bmatrix}$$

$$G_1^{-1} = \begin{bmatrix} \frac{1}{d + \mu} & 0 & \frac{\mu}{(d + \mu)(d + d_1 + au)} & \frac{1}{d + d_1 + au} \end{bmatrix}$$

And

$$F_1 G_1^{-1} = \begin{bmatrix} \frac{\alpha S_0}{1 + \beta S_0} & \frac{\mu}{(d + \mu)(d + d_1 + au)} & \frac{\alpha S_0}{1 + \beta S_0} & \frac{1}{d + d_1 + au} & 0 & 0 \end{bmatrix}$$

Again, the spectral R_0 of the matrix $F_1 G_1^{-1}$ is the basic reproduction number of the model

$$\text{i.e., } R_0 = \rho(F_1 G_1^{-1}) = \frac{a\mu A}{(m + d + m\beta)((d + \mu)(d + d_1 + au))}$$

3.2 Equilibria and their stability analysis

The system (1) has a disease free equilibrium (DFE) $E_1(S_1, 0, 0, R_1)$, which is already discussed.

The system (1) has another one positive interior equilibrium point $E^*(S^*, E^*, I^*, R^*)$, where

$$S^* = \frac{(d+\mu)(1+\gamma I^*)\{(d+d_1)(1+\delta u I^*)+au\}}{a\mu(1+\delta u I^*)-\beta(d+\mu)(1+\gamma I^*)\{(d+d_1)(1+\delta u I^*)+au\}},$$

$$E^* = \frac{1}{\mu}\left(d + d_1 + \frac{au I^*}{1+\delta u I^*}\right), \quad R^* = \frac{1}{d+m} \left(mS^* + \frac{au I^*}{1+\delta u I^*}\right)$$

And I^* is the positive root of the equation

$$M_0 x^4 + M_1 x^3 + M_2 x^2 + M_3 x + M_4 = 0$$

and the coefficient are given by

$$M_0 = k_2^2 k_3^2 k_4^2 \delta^2 u^2 \beta \gamma$$

$$M_1 = k_2 k_3 k_4 \left\{ \beta \gamma \delta u k_2 k_3 + \beta k_2 k_3 \delta^2 u^2 + \right.$$

$$\left. \beta k_2 (k_3 + au) \gamma - a \mu \delta^2 u^2 - A \mu \beta \gamma \delta^2 u^2 \right\}$$

$$- k_2^2 k_3^2 a \mu \beta \gamma \delta u - \mu (k_1 k_4 - m m_1)$$

$$k_2 k_3 \gamma \delta^2 u^2 - m_1 \mu a \beta \delta \gamma k_2 k_3 u^2,$$

$$M_2 = -k_4 A \mu \left\{ \beta \gamma k_2 k_3 \delta^2 u^2 - a \mu \delta^2 u^2 + k_2 k_3 \beta \delta^2 u^2 + \beta k_2 (k_3 + au) \gamma \delta u \right\}$$

$$- \mu (k_1 k_4 - m m_1) \left\{ k_2 k_3 \gamma \delta u + k_2 k_3 \gamma \delta^2 u^2 + k_2 (k_3 + au) \beta \delta u \gamma \right\} - k_2 k_3 k_4 \left\{ 2 a \mu \delta u \right.$$

$$\left. - k_2 k_3 \beta \delta u - \beta k_2 (k_3 + au) \gamma - \beta k_2 (k_3 + au) \delta u \right\} - k$$

$$M_3 = k_4 A \mu \left\{ 2 a \mu \delta u - k_2 k_3 \beta \delta u - \beta k_2 (k_3 + au) \gamma - \beta k_2 (k_3 + au) \delta u \right\} - k_2 k_4 a \mu \left\{ a \mu - \right.$$

$$\left. \beta k_2 (k_3 + au) \right\} - \mu (k_1 k_4 - k_2 k_3 k_4 \left\{ a \mu - \beta k_2 (k_3 + au) \right\}),$$

$$M_4 = k_4 \mu \left\{ a \mu - \beta k_2 (k_3 + au) \right\} - m u k_2 (k_1 k_4 - m m_1) (k_3 + au),$$

where $k_1 = d + m$, $k_2 = d + \mu$, $k_3 = d + d_1$, $k_4 = d + m$.

Theorem 2. The disease-free equilibrium E_1 of model (2) is

- 1) Absolutely stable if $R_0 < 1$,
- 2) Linearly neutrally stable if $R_0 = 1$, and
- 3) Unstable if $R_0 > 1$.

Proof: The variational matrix of the system (1) around the disease-free equilibrium point E_1 is

$$V(E_1) = \begin{bmatrix} -(d+m) & 0 & -\frac{a S_1}{1+\beta S_1} & m_1 & 0 & -(d+\mu e^{-\lambda \tau}) & \frac{a S_1}{1+\beta S_1} & 0 & 0 & m e^{-\lambda \tau} & 0 & -(d+d_1+au) & au & 0 & -(d+m_1) \end{bmatrix}$$

Eigen values of the variational matrix are $\lambda_1 = -\mu$, $\lambda_1 = -(d+m+m_1)$ and the roots of the equation

$$\lambda^2 + (2d + d_1 + au)\lambda + (d + d_1 + au) +$$

$$e^{-\lambda \tau} \left\{ \mu \lambda - (d + d_1 + au)(d + \mu) \left(R_0 - \frac{\mu}{d + \mu} \right) \right\} = 0, \quad (3)$$

It is clear that if $R_0 < 1$, the roots of the above equation have negative real parts. So the disease free equilibrium E_1 is locally asymptotically stable when $\tau = 0$.

Case II: when $\tau > 0$, let $\lambda = i\omega$ be the roots of the equation (4), then

$$-\omega^2 + i\omega(2d + d_1 + au) + d(2d + d_1 + au)$$

$$+ e^{-i\omega \tau} \left\{ i\mu \omega - (d + d_1 + au)(d + \mu) \left(R_0 - \frac{\mu}{d + \mu} \right) \right\} = 0 \quad (5)$$

Separating real and imaginary parts, we get

$$d(d + d_1 + au) - \omega^2 = \mu\omega \sin \sin \omega\tau - (d + d_1 + au)(d + \mu)\left(R_0 - \frac{\mu}{d+\mu}\right)\cos\omega\tau,$$

$$\omega(2d + d_1 + au) = \mu\omega \cos \cos \omega\tau + (d + d_1 + au)(d + \mu)\left(R_0 - \frac{\mu}{d+\mu}\right)\sin\omega\tau$$

Squaring and adding the above two equations, we have

$$\omega^4 + Q_1\omega^2 + Q_2 = 0, \tag{6}$$

Where $Q_1 = (d + d_1 + \mu + au)(d - \mu + au) + d^2,$

$Q_2 = (d + d_1 + au)^2(d + \mu)(1 - R_0^2) > 0,$ if $R_0 < 1.$

It is clear that ω^2 is negative when $R_0 < 1.$ Which implies that the equation (3) has no purely imaginary root for $\tau > 0.$ Thus by Definition (3) and lemma 3.5(i) [34], the disease free equilibrium E_I is absolutely stable for $\tau \geq 0.$

3.3 Stability analysis of the positive interior equilibrium

The variational matrix of the system (1) at $E^* (S^*, E^*, I^*, R^*)$ is

$$V^* = \begin{bmatrix} m_{11} & 0 & m_{12} & m_{14} & m_{21} & m_{22} & m_{23} & 0 & 0 & m_{41} & m_{32} & 0 & m_{33} & m_{43} & 0 & m_{44} \end{bmatrix}$$

$$m_{11} = - \left\{ d + m + \frac{\alpha I^*}{(1+\beta S^*)^2(1+\gamma I^*)} \right\}, m_{13} = - \frac{\alpha S^*}{(1+\beta S^*)(1+\gamma I^*)^2}, m_{13} = m_1;$$

$$m_{21} = \frac{\alpha I^*}{(1+\beta S^*)^2(1+\gamma I^*)}, m_{22} = - (d + \mu e^{-\lambda\tau}), m_{23} = \frac{\alpha S^*}{(1+\beta S^*)(1+\gamma I^*)^2};$$

$$m_{32} = \mu e^{-\lambda\tau}, m_{33} = - \left\{ d + d_1 + \frac{\alpha u I^*}{(1+\delta u I^*)^2} \right\};$$

$$m_{41} = m, m_{43} = \frac{\alpha u I^*}{(1+\delta u I^*)^2}, m_{44} = - (d + m)$$

The eigen values of V^* at E^* are the roots of the equation

$$\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 + e^{-\lambda\tau}(B_3\lambda^3 + B_2\lambda^2 + B_1\lambda + B_0) = 0 \tag{7}$$

Where

$$A_3 = - (m_{11} - d + m_{33} + m_{44}),$$

$$A_2 = m_{11}m_{44} - dm_{33} + (m_{11} + m_{44})(-d + m_{33}) - m_{14}m_{41}$$

$$A_1 = dm_{33}(m_{11} + m_{44}) - m_{11}m_{44}(-d + m_{33}) + m_{14}m_{41}(-d + m_{33})$$

$$A_0 = - dm_{11}m_{44}m_{33} + dm_{14}m_{41}m_{33}$$

$$B_3 = - \mu; B_2 = - \mu(m_{11} + m_{33} + m_{11} + m_{23});$$

$$B_1 = \mu m_{33}(m_{11} + m_{33}) + \mu m_{11}m_{44} - \mu m_{14}m_{41} - \mu m_{23}m_{21}$$

$$B_0 = - \mu m_{11}m_{33}m_{44} - \mu m_{11}m_{23}m_{44} + \mu m_{21}m_{23}m_{44} - \mu m_{14}m_{21}m_{43} + \mu m_{14}m_{41}m_{33} + \mu m_{14}m_{41}m_{23}$$

Case-I: When $\tau = 0,$ equation (7) becomes

$$\lambda^4 + A_{13}\lambda^3 + A_{12}\lambda^2 + A_{11}\lambda + A_{10} = 0,$$

Where $A_{13} = A_3 + B_3, A_{12} = A_2 + B_2, A_{11} = A_1 + B_1, A_{10} = A_0 + B_0.$

Obviously, $A_{13} = - (m_{11} + m_{22} + m_{33} + m_{44}).$ Let $Det_1 = A_{13} > 0.$

Thus, by the Routh–Hurwitz theorem, if the condition (H1) Equations (8)–(10) holds, then the positive equilibrium E^* (S^* , E^* , I^* , R^*) of system (1) without time delay is locally asymptotically stable.

$$Det_2 = \begin{vmatrix} A_{11} & 1 & A_{11} & A_{11} \end{vmatrix} > 0, \quad (8)$$

$$Det_2 = \begin{vmatrix} A_{11} & 1 & 0 & A_{11} & A_{12} & A_{13} & 0 & A_{10} & A_{11} \end{vmatrix} > 0, \quad (9)$$

$$Det_3 = \begin{vmatrix} A_{11} & 1 & 0 & 0 & A_{11} & A_{12} & A_{13} & 1 & 0 & 0 & A_{10} & 0 & A_{11} & 0 & A_{12} & A_{10} \end{vmatrix} > 0, \quad (10)$$

Case-II: When $\tau \neq 0$,

Let $\lambda = i\omega$ be the roots of the equation (7), then

$$\omega^4 - A_3 i \omega^3 - A_2 \omega^2 + i A_1 \omega + A_0 + e^{-i\omega\tau} (-i B_3 \omega^3 - B_2 \omega^2 + i B_1 \omega + B_0) = 0, \quad (11)$$

Separating real and imaginary parts, we get

$$(B_1 \omega - B_3 \omega^3) \sin \sin \omega\tau + (B_0 - B_2 \omega^2) \cos \omega\tau = \omega^4 - A_2 \omega^2 + A_0 \quad (12)$$

$$(B_2 \omega^2 - B_0) \sin \sin \omega\tau + (B_1 \omega - B_3 \omega^3) \cos \omega\tau = -A_3 \omega^3 - A_1 \omega \quad (13)$$

Squaring and adding the above two equations we have

$$Q^8 + Q_1 \omega^6 + Q_2 \omega^4 + Q_3 \omega^2 + Q_4 = 0, \quad (14)$$

Where

$$\text{where } Q_1 = A_3^2 - (B_3^2 - 2A_2), Q_2 = A_2^2 + 2B_0 + 2B_1 B_3 - (B_2^2 + 2A_1 A_3), \\ Q_3 = B_1^2 + 2B_0 B_2 + A_1^2 - 2A_0 A_2^2, Q_4 = A_0^2 - B_0^2.$$

Let $\omega^2 = v$, we have,

$$v^4 + Q_1 v^3 + Q_2 v^2 + Q_3 v + Q_4 = 0, \quad (15)$$

Thus, in order to obtain the main result in this paper, we make the following assumptions.

H_{21} Equation (14) has at least one positive root. Then there exists a positive root of (15) v_1 such that equation (7) has a pair of purely imaginary roots $\pm i\omega_1 = \pm \sqrt{v_1}$. Then from equations (12) and (13), we can obtain the corresponding critical value of the delay for ω_1

$$\tau_1 = \frac{1}{\omega_1} \arccos \frac{p_1 \omega_1^6 + p_2 \omega_1^4 + p_3 \omega_1^2 + p_4}{q_1 \omega_1^6 + q_2 \omega_1^4 + q_3 \omega_1^2 + q_4}$$

Where

$$p_1 = A_3 B_3 - B_1, p_2 = B_0 + A_2 B_2 + A_0 B_0 - A_3 B_1 - A_1 B_3,$$

$$p_3 = A_1 B_1 - A_2 B_0 - A_0 B_2, p_4 = A_0 B_0, q_1 = B_3^2, q_2 = B_2^2 - 2B_3 B_1,$$

$$q_3 = B_1^2 - 2B_0 B_2, q_4 = A_0^2 - B_0^2$$

Substituting the value of λ (τ) in (3) and differentiating w.r.t. τ we have

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = -\frac{4\lambda^3 + 3A_3 \lambda^2 + 2A_2 \lambda + A_1}{\lambda(\lambda^4 + A_3 \lambda^3 + A_2 \lambda^2 + A_1 \lambda + A_0)} + \frac{3B_3 \lambda^2 + 2B_2 \lambda + B_1}{B_3 \lambda^3 + B_2 \lambda^2 + B_1 \lambda + B_0} - \frac{\tau}{\lambda}$$

Which leads to

$$Re\left(\frac{d\lambda}{d\tau}\right)^{-1}_{\tau=\tau_1} = -\frac{f'_1(v_1)}{(B_1 \omega_1 - B_3 \omega_1^3)^2 + (B_0 - B_2 \omega_1^2)^2}$$

Where

$$f'_1(v_1) = v^4 + Q_1 v^3 + Q_2 v^2 + Q_3 v + Q_4 = 0 \text{ and } \omega_1^2 = v_1$$

Thus if the conditions

$$H_{22}, f'_1(v_1) \neq 0 \text{ then } Re\left(\frac{d\lambda}{d\tau}\right)^{-1}_{\tau=\tau_1} \neq 0$$

According to the Hopf bifurcation theorem in [35], we have the following for the system (1),

Theorem3: If the conditions $H_{21} - H_{22}$ holds, then

- 1) the positive equilibrium of (1) is asymptotically stable for $\tau \in [0, \tau_1]$,
- 2) System (1) undergoes a Hopf bifurcation at the positive equilibrium E^* when $\tau = \tau_1$

4. Numerical Simulations

Table 1: A set of parameter values

Parameter	Default Value
A	1
α	0.14
β	0.1
γ	0.2
m_1	0.07
m	0.02
a	0.09
μ	0.025

u	1
d	0.011
d_1	0.1
δ	0.01

In this part, numerically supported the theoretical analysis for the set hypothetical parameters values in table

1. For the set of parameters, from the system (1) we get, $R_0 = 2.76$, endemic equilibrium point $E^* = (16.6570, 15.8870, 29.9727, 19.3451)$. By direct computation, we Have $Det1 > 0, Det2 > 0, Det3 > 0, Det4 > 0$. Clearly, the conditions H_1

holds. So E^* is asymptotically stable (see in Figure 1). If $u = 2.5$ and other parameter fixed, then from the system (1) we get $R_0 = 0.9196$ and disease free equilibrium point $E_1 = (72.9096, 0, 0, 18.0018)$ is asymptotically stable (see in Figure 2).

For $\tau > 0$, using Matlab software, show that the conditions H_2 and H_3 hold. Then, we get

$\omega = 0.0886, \tau \geq 15.2919$. From the theorem 1, we can deduce that E^* is asymptotically stable when $\tau \in [0, 15.2919)$ and a Hopf bifurcation occurs at the critical values $\tau \geq 15.2919$ (describe in Figure4).

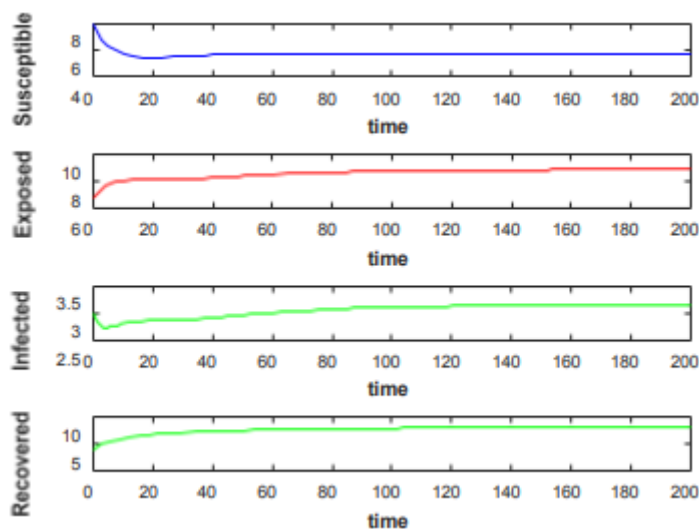


Figure 1: The equilibrium point E^* is locally asymptotically stable for the set of parameter in the Table 1.

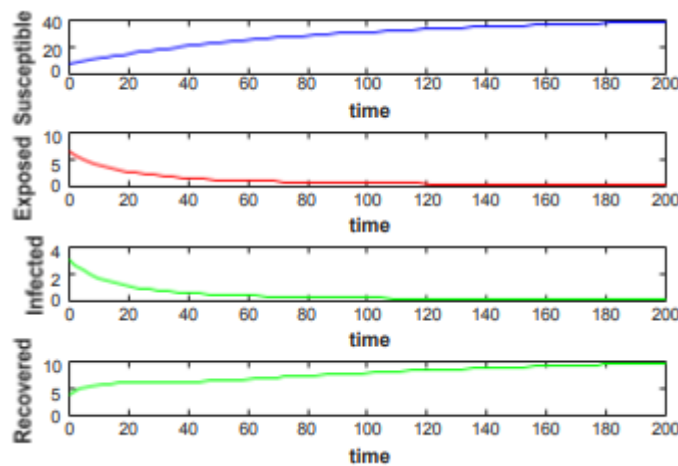


Figure 2: The figures depicts that the disease free equilibrium point E_1 is stable for $\alpha = 0.039, u = 2.5$ and other set of parameter fixed in Table 1.

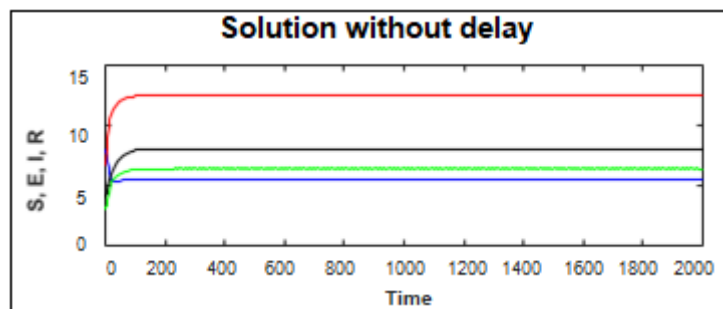


Figure 3 (a)

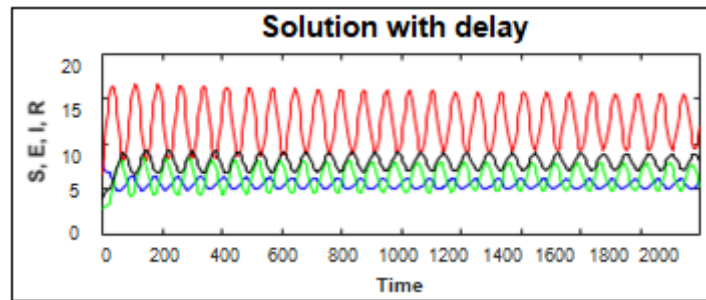
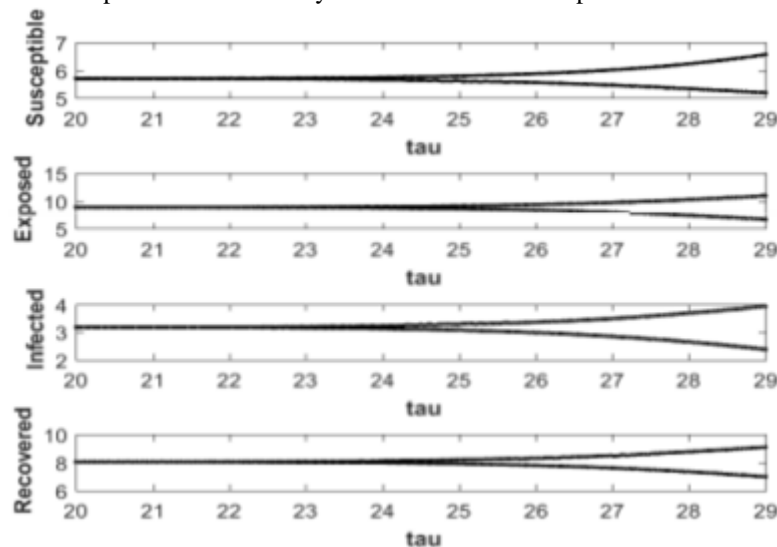


Figure 3 (b)

Figure 3: The figure 3(a) show that E^* is locally asymptotically stable for the set of parameter in the Table 1 and $\tau = 0$; the figure 3(b) show that all species are oscillatory behavior for the set of parameter in the Table 1 and $\tau = 22$.

Figure 4: The bifurcation diagram of all the populations with tau (τ) as the bifurcation parameter.

5. Conclusion

This paper is concerned with a delayed SEIR epidemic model with saturated incidence and saturated treatment function. The effect of the delays on the model is investigated and the main results are given in terms of local stability and local Hopf bifurcation. It has been shown that the model is stable when the value of the bifurcation parameter is below the critical value, which means that the disease can be controlled easily. However, when the value of the bifurcation parameter is above the critical value, a Hopf bifurcation will occur. In this condition, the disease is out of control. Accordingly, we should shorten the delay in the model as much as possible so that we can predict and control the disease propagation. Finally, some numerical simulations are carried out to support our theoretical results.

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