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Local Stability Analysis of Delayed Seir Model

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Abstract

In this work, we consider a system of Delay differential equations for SEIR models with logistic and bilinear incidence. This model shows a bifurcation point where a stable disease-free equilibrium (DFE) coexists with a stable endemic equilibrium, according to studies (EE). When the reproduction number determines the local equilibrium stability requirements and the presence of Hopf bifurcations. To obtain stable behavior, we also performed a branch analysis with expected lag times. Numerical simulations were used to demonstrate the relevance and validity of the theoretical results.

Keywords: Delay, Stability analysis, Bifurcation, SEIR Model.

1. Introduction

In recent decades, mathematical modeling has become increasingly important in epidemiological theory. Various epidemic models have been developed and extensively studied, greatly advancing the study of disease control and prevention [1-7]. From equations to statistical analysis, a full understanding of disease dynamics requires a variety of mathematical techniques. Although mathematics has made admirable contributions to the field of epidemiology, there is no denying that certain elements still need proper mathematical models.

Mathematical models help design and engineer, estimate and evaluate, compare, and optimize in combating disease through prevention, treatment, and other control programs. In the SEIR model, the entire population is distributed as susceptible (S), exposed (E), infected (I), and

recovered or cleared (R) individuals. Difficulties in understanding thedetails of infectious disease transmission tend to change depending on the situation. Furthermore, the choice of generalized incidence rate function flexibly determines the function from the incidence rate to be used.

Most of these mathematical models of disease start with the same premise: that the population can be divided into collections of distinct groups based on their experience with the disease. Nonlinear differential equations are used to describe most of them (Delay difference, Stochastic, etc.).

The inclusion of time delay is frequently used to represent the latent period, which is the interval between infection and the host becoming infectious [9]. The majority of authors believe that disease latent periods are insignificant.,i.e.,Each susceptible individual (S) other than exposed (E) becomes infectious (I) almost instantly after being infected, and later recovers (R) with permanent or temporary acquired immunity. These models are commonly referred to as SEIR (susceptible, Exposed, infectious, recovered) models [10-13].

In this study, we add a separate time delay to the model to reflect the time it takes for a suspected individual to become infected. This is called the latent period. The result of this process is a system of delay differential equations. We analyze transcendental characteristic equations of linearized systems in positively infected steady-state, understand the dynamics of delay models, and attempt to determine analytical environments in which the infected steady-state stabilizes. Numerical simulations are performed to illustrate the results obtained.

The following is an overview of the paper's structure. In Section 2, we first propose the SEIR epidemic model with time delay system, after which we verify the existence of equilibrium and the reproduction number for the system. The stability of the disease-free and infected steady states, as well as the existence of Hopf bifurcation around the positive equilibrium, are discussed in Section 3. The length of the delay to maintain stability was described in section 4. In Section 5, we use numerical simulations to demonstrate the paper's primary findings. Finally, we will come to a conclusion and discussion in section 6

2. Characterization of the SEIR model

The following SEIR epidemic model with discrete delay is proposed in this study. As our basic model, we have the following set of equations from [14].

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right)-\beta SI \\
\frac{dE}{dt} &= \beta SI-\left(\frac{\nu I}{1+\zeta I}+\mu\right)E \\
\frac{dI}{dt} &= \beta SI-(\mu+\alpha+\gamma)I-\frac{\nu I}{1+\zeta I} \\
\frac{dR}{dt} &= \gamma I+\frac{\nu I}{1+\zeta I}-\mu R
\end{aligned} \qquad (1)$$

For the purposes of this discussion, we have changed the above model (3) and added a delay to the system, as follows:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right)-\beta SI \\
\frac{dE}{dt} &= \beta SI-\left(\frac{\nu I}{1+\zeta I}+\mu\right)E \\
\frac{dI}{dt} &= \beta S(t-\tau)I(t-\tau)-(\mu+\alpha+\gamma)I-\frac{\nu I}{1+\zeta I} \\
\frac{dR}{dt} &= \gamma I+\frac{\nu I}{1+\zeta I}-\mu R.
\end{aligned} \qquad (2)$$

Here,The numbers of susceptible, exposed, infectious, and recovery cells at time t are denoted by $S(t), E(t), I(t)$, and $R(t)$, respectively.r is the intrinsic growth rate of the susceptible population, K is the country's carrying capacity excluding infected and recovered people, β is the transmission rate, μ is the natural death rate, α is the disease-induced death rate, γ is the recovered rate, and ν is the maximum medical resources supplied per unit time andζ is the half-saturation constant, which measures the effect of treatment delay. In this work, it is assumed that ν is a non-negative constant and that all other parameters are positive constants, with τ being the time required for a person to become infectious.

Now that our model (2) has been simplified, we can see that it has two steady states: The stable infection-free state$E_0 = (\bar{S}, \bar{E}, \bar{I}, \bar{R})$ and the infected steady state $E_1 = (S^*, E^*, I^*, R^*)$.

2.1. Positivity and Solution Boundedness

In this section we consider the following system of differential equations

$$\begin{aligned}\frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right)-\beta SI\\ \frac{dE}{dt} &= \beta SI-\left(\frac{\nu I}{1+\zeta I}+\mu\right)E,\\ \frac{dI}{dt} &= \beta SI-(\mu+\alpha+\gamma)I-\frac{\nu I}{1+\zeta I}\end{aligned} \quad (3)$$

(4)

By summing up the above system of equations we have

$$\text{LimSup}(S+E+I) \leq \frac{\beta}{\mu}$$

so the feasible region for model is

$$F = S,E,I: S+E+I \leq \frac{\beta}{\mu}, S>0, E>0, I>0$$

2.2. Equilibrium Points

In this section, we compute the models endemic equilibrium points. The disease free equilibrium is obtained by setting the system of differential equations to zero. At disease free equilibrium, there are no infections and recovery. The disease free equilibrium is given by;

$$(S^*, E^*, I^*, R^*) = \left(\frac{\beta}{\mu}, 0,0,0\right)$$

2.3. The basic reproduction number:

The basic reproduction number, R_0, is the estimated number of secondary cases produced by a typical infected individual in a totally susceptible population, according to [15]. If $R_0 < 1$, an infected person creates less than one new infected person on average throughout the course of their infectious period, and the infection cannot spread.

Conversely, If R_0, each infected person creates more than one new infection on average, and the sickness can spread across the population. R_0 is simply the product of the infection rate and the mean duration of the infection in the case of a single infected compartment. We'll now determine the system's basic reproduction number. (2). Let $X = (S,E,I,R)^T$, then the model (2) can be written as

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X)$$

Where,

$$\mathcal{F}(X) = \begin{bmatrix} \beta IS \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$\mathcal{V}(X) = \begin{bmatrix} (\mu + \alpha + \gamma)I + \dfrac{\nu I}{1 + \zeta I} \\ -\beta SI + \left(\dfrac{\nu I}{1 + \zeta I} + \mu\right)E \\ -rS\left(1 - \dfrac{S}{K}\right)^2 + \beta SI \\ -\gamma I - \dfrac{\nu I}{1 + \zeta I} + \mu R \end{bmatrix}$$

According to Theorem 2 in [16], the reproduction number of model (2) is

$$R_0 = \frac{\beta K}{\mu + \alpha + \gamma + \nu}. \qquad (5)$$

It is obvious that if $R_0 < 1$, then the infection free steady state $E_0(\bar{S}, 0,0,0)$ (where $\bar{S} = K$) is the only steady state, corresponding to the infection-free state's extinction.

In terms of the presence of equilibria, we now obtain the following result. Theorem 2.1. If $R_0 > 1$, then the system (2) has an unique equilibrium $E_1(S^*, E^*, I^*, R^*)$ (i.e., $S^* > 0, E* > 0, I^* > 0, R^* > 0$) where S^*, E^*, I^* and R^* are given in the proof.

Proof. If $R_0 > 1$, the system (2) then looks like this,

$$\begin{aligned} rS^*\left(1 - \frac{S^*}{K}\right) - \beta S^* I^* &= 0, \\ \beta S^* I^* - \left(\frac{\nu I^*}{1 + \zeta I^*} + \mu\right)E^* &= 0, \\ \beta S^* I^* - (\mu + \alpha + \gamma)I^* - \frac{\nu I^*}{1 + \zeta I^*} &= 0, \\ \gamma I^* + \frac{\nu I^*}{1 + \zeta I^*} - \mu R^* &= 0. \end{aligned} \qquad (6)$$

Hence, the system (2) has a unique equilibrium $E_1(S^*, E^*, I^*, R^*)$ if $R_0 > 1$.

As a result of the foregoing analysis, we arrive at the following conclusion.

Theorem 2.2. Consider the system (2) with R_0 defined in (5). If $R_0 < 1$, then there is unique equilibrium, which is the infection-free steady state E_0; while if $R_0 > 1$, then there is unique equilibrium, which is the infected steady state E_1.

3. Stability analysis

We adopt the following notation: $\mathbb{R}^4$ is a four-dimensional real Euclidean space with norm $|.|$. For $\tau > 0$, we denote by $C = C([-\tau, 0], \mathbb{R}^4_+)$, the Banach space of continuous function mapping the interval $[-\tau, 0]$ into $\mathbb{R}^4_+$ with the topology of uniform convergence. By the standard theory of functional differential equation $[17 - 19]$, we know that for any $\phi \in C([-\tau, 0], \mathbb{R}^4_+)$, there exists a unique solution

$$Z(t, \phi) = (S(t, \phi), E(t, \phi), I(t, \phi), R(t, \phi)),$$

of the delayed system (2), which satisfy $Z_0 = \phi$, where $\phi = (\phi_1, \phi_2, \phi_3, \phi_4) \in \mathbb{R}^4_+$ with $\phi_i(\xi) \geq 0: (\xi \in [-\tau, 0], \quad i = 1,2,3,4)$, and $\phi_1(0), \phi_2(0), \phi_3(0), \phi_4(0) > 0$. And the initial conditions are given by,

$$S(\xi) = \phi_1(\xi), \quad E(\xi) = \phi_2(\xi) \tag{7}$$
$$I(\xi) = \phi_3(\xi), \quad R(\xi) = \phi_4(\xi). \tag{8}$$

Theorem 3.1. Let $Z(t, \phi)$ be the solution of the delayed system (2) with the initial conditions (7). Then $S(t), E(t), I(t)$ and $R(t)$ are all non-negative and ultimately uniformly bounded $(\forall t \geq 0)$ at which the solution exists.

3.1. Local stability analysis

In this section, we look at the model's local stability analysis. (2).

Theorem 3.2. The infection free steady state of model (2) is unstable when $R_0 > 1$ and locally asymptotically stable at E_0 when $R_0 < 1$ in the case of $\tau \geq 0$.

When $R_0 > 1$, the system (2) hasa infected steady state $E_1 = (S^*, E^*, I^*, R^*)$. Then the characteristic polynomial of the the linearized system (2) at E_1 is

$$\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 + e^{-\lambda\tau}(\lambda) = B_1\lambda^3 + B_2\lambda^2 + B_3\lambda + B_4\big) = 0 \qquad (9)$$

Theorem 3.3. The infected steady state of model (2) is locally asymptotically stable when $R_0 > 1$ in the case of $\tau > 0$.

Proof. In the case of $\tau > 0$, the above characteristic equation (9) an be rewritten as

$$H(\lambda, \tau) = P(\lambda) + Q(\lambda)e^{-\lambda\tau} = 0,$$

where $P(\lambda) = \lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4$ and $Q(\lambda) = B_1\lambda^3 + B_2\lambda^2 + B_3\lambda + B_4$.

Theorem 3.4. Suppose $R_0 > 1$, the following result can be obtained.

1 The infected equilibrium E_1 is stable when $\tau \in [0, \tau^*)$ and unstable when $\tau > \tau^*$. τ is the Hopf bifurcation value, which means that periodic solutions will bifurcate from this infected equilibrium as τ passes through the critical value τ^*.

4. Estimation of the Length of Delay to Preserve Stability

Following lines of Erbe et al. [20] and using the Nyquist criterion [21], it can be shown that the sufficient conditions for the local asymptotic stability of $E_1(S^*, E^*, I^*, R^*)$ are given by,

$$\mathrm{Im}H(i\omega_0) > 0, \qquad (10)$$
$$\mathrm{Re}H(i\omega_0) = 0, \qquad (11)$$

where $H(\varrho) = \varrho^4 + A_1\varrho^3 + A_2\varrho^2 + A_3\varrho + A_4 + e^{-\varrho\tau}(B_1\varrho^3 + B_2\varrho^2 + B_3\varrho + B4)$ and ω_0 is the smallest positive root of (11).

Inequality (10) and (11) can alternatively be written as

$$A_2\omega_0 - \omega_0^3 > -B_2\omega_0\cos(\omega_0\tau) + B_3\sin(\omega_0\tau) - B_1\omega_0^2\sin(\omega_0\tau), \qquad (12)$$
$$A_3 - A_1\omega_0^2 = B_1\omega_0^2\cos(\omega_0\tau) - B_3\cos(\omega_0\tau) - B_2\omega_0\sin(\omega_0\tau). \qquad (13)$$

Now if (12) and (13) are both satisfied at the same time, they are sufficient to provide stability. These are now used to estimate how long the time delay will be. The goal is to establish an upper

bound $\omega_+$to ω_0 from (13) that is independent of τ, and then to estimate τ so that (12) holds true for all values of ω such that $0 \leq \omega \leq \omega_+$, and hence, in particular at $\omega = \omega_0$. Equation (13) can be rewritten as

$$A_1\omega_0^2 = A_3 - B_1\omega_0^2\cos(\omega_0\tau) + B_3\cos(\omega_0\tau) + B_2\omega_0\sin(\omega_0\tau). \quad (14)$$

Maximizing the right hand side of (14) subject to,

$$|\sin(\omega_0\tau)| \leq 1, \quad |\cos(\omega_0\tau)| \leq 1, \quad (15)$$

we obtain

$$|A_1|\omega_0^2 \leq |A_3| + |B_3| + |B_1|\omega_0^2 + |B_2|\omega_0. \quad (16)$$

Hence if,

$$\omega_+ = \frac{1}{2(|A_1|-|B_1|)}\left\{|B_2| + \sqrt{B_2^2 + 4(|A_1| - |B_1|)(|A_3| + |B_3|)}\right\}, \quad (17)$$

then clearly from (16) we have $\omega_0 \leq \omega_+$.

From (12), we obtain

$$\omega_0^2 < A_2 + B_2\cos(\omega_0\tau) + B_1\omega_0\cos(\omega_0\tau) - \frac{B_3\sin(\omega_0\tau)}{\omega_0}. \quad (18)$$

Since $E_1(S^*, I^*, R^*)$ is locally asymptotically stable for $\tau = 0$, the inequality (18) will continue to hold for sufficiently small $\tau > 0$. Using (14) and (18) can be rearranged as

$$(B_3 - B_1\omega_0^2 - A_1B_2)(\cos(\omega_0\tau) - 1) + \left((B_2 - A_1B_1)\omega_0 + \frac{A_1B_3}{\omega_0}\right)\sin(\omega_0\tau) < A_1A_2 - A_3 - B_3 + B_1\omega_0^2 + A_1B_2. \quad (19)$$

Using the bound

$$
\begin{aligned}
(B_3 - B_1\omega_0^2 - A_1B_2)(\cos(\omega_0\tau) - 1) = \quad & (B_3 - B_1\omega_0^2 - A_1B_2)2\sin^2\left(\frac{\omega_0\tau}{2}\right) \\
& \leq \frac{1}{2}|(-B_3 + B_1\omega_0^2 + A_1B_2)|\omega_+^2\tau^2 \\
\left((B_2 - A_1B_1)\omega_0 + \frac{A_1B_3}{\omega_0}\right)\sin(\omega_0\tau) \quad & \leq (|(B_2 - A_1B_1)|\omega_+^2 + |A_1||B_3|)\tau, \qquad (20)
\end{aligned}
$$

we obtain from (18)

$$L_1\tau^2 + L_2\tau < L_3, \qquad (21)$$

Where,

$$
\begin{aligned}
L_1 &= \frac{1}{2}|(-B_3 + B_1\omega_0^2 + A_1B_2)|\omega_+^2 \\
L_2 &= |(B_2 - A_1B_1)|\omega_+^2 + |A_1||B_3| \\
L_3 &= A_1A_2 - A_3 - B_3 + B_1\omega_+^2 + A_1B_2 \qquad (22)
\end{aligned}
$$

Hence if,

$$\tau_+ = \frac{1}{2L_1}\left(\mathrm{E}_2 + \sqrt{L_2^2 + 4L_1L_3}\right), \qquad (23)$$

then for $0 \leq \tau \leq \tau_+$, the Nyquist criterion holds true and τ_+estimates the maximum length of the delay preserving the stability.

5. Numerical simulation

In this part, we provide some numerical simulations to illustrate the theoretical results given in Theorems 2.2 and 3.2. The precise values of the time delays intervals for some parameter based on information, we assumed that τ is ≤ 6 days.

(i) We choose the different values of parameters satisfying the conditions in Theorem 2.2 as follows.

$$
\begin{aligned}
r = 0.7,1.3,1.3, \quad \beta = 0.02,0.3,0.02, \quad K = 0.8,0.05,0.08, \quad \mu = 0.5,0.9,0.2, \\
\alpha = 0.1,0.05,0.5 \quad \gamma = 0.05,0.2,0.1, \quad \zeta = 0.01,0.3,0.5, \quad \nu = 0.1,0.4,0.1.
\end{aligned}
$$

Then we have $R_0 = 0.021,0.099,0.0001 < 1$, and due to Theorem 2.2 the disease-free equilibrium E_0 of System (3) is asymptotically stable which is shown well in Fig. 1. Here $(S(t), E(t), I(t), R(t))$ are the solutions of System (2) with initial conditions

$$S(\xi) = \phi_1(\xi) = 0.6, \quad E(\xi) = \phi_2(\xi) = 0.3, \quad I(\xi) = \phi_3(\xi) = 0.2, \quad R(\xi) = \phi_4(\xi) = 0.2, \quad \xi \in [-\tau, 0].$$

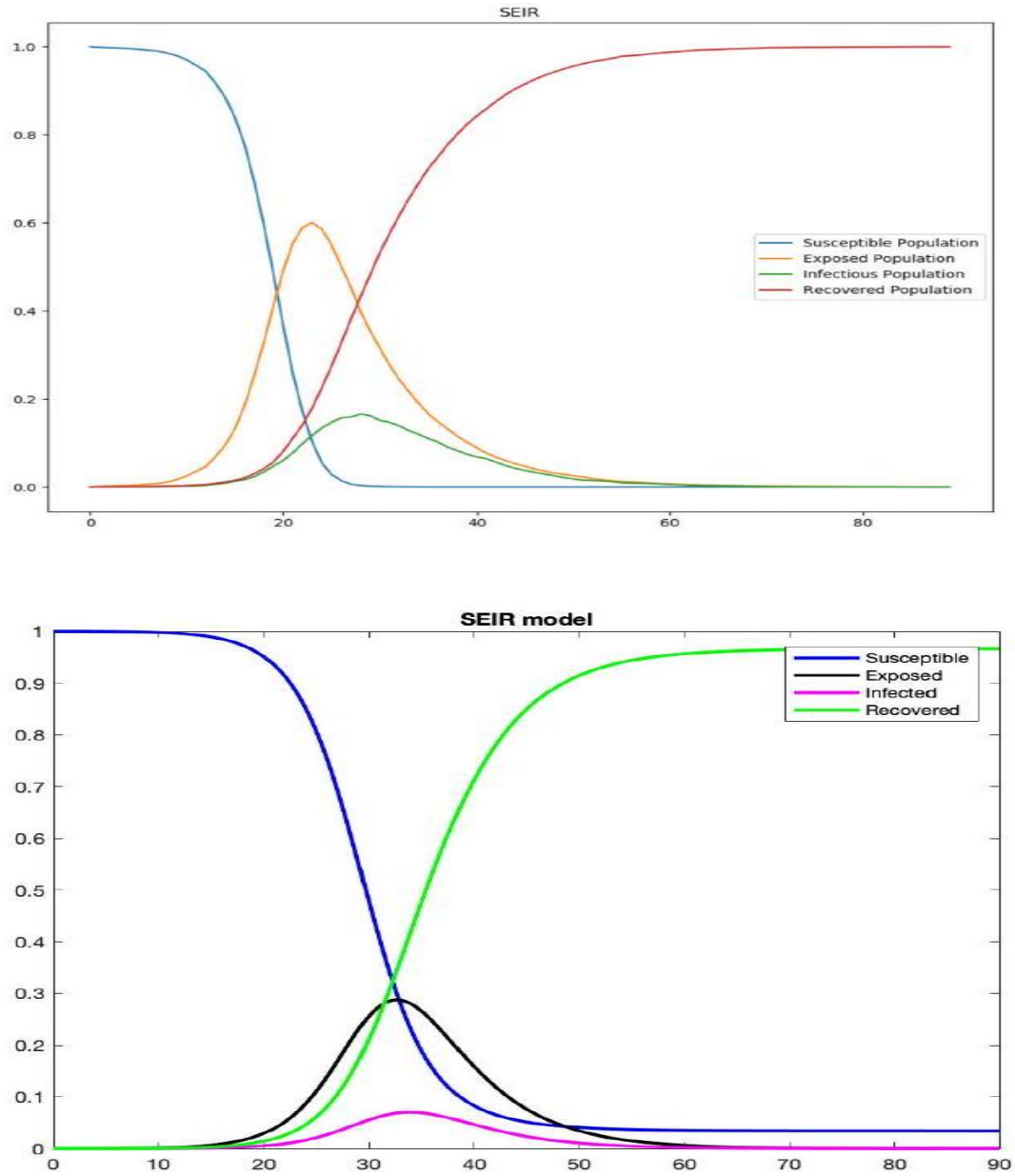

FIGURE 1: Solutions of the system (2) go to the disease-free steady state, where $S(t)$ represents the suspected cells, $E(t)$ represents the exposed cells, $I(t)$ represents the infected cells, and $R(t)$ represents recovered cells.

We can see from the preceding figures that our delay model (2) is fairly reliable. As a result, our numerical results are suitable for describing our model. Furthermore, minor changes in parameters will result in minute changes in the matrix entries required to calculate eigenvalues and determine the stability of two steady-state locations.

6. Discussion and Conclusion

We have included time delay in our SEIR models in this work. We demonstrated that the threshold value R_0 is crucial in determining the stability of the model dynamics' steady states. We have demonstrated that the infected steady state is locally asymptotically stable if the threshold value R_0 is bigger than unity. The permissible time delay for activation of infected cells, as well as the prediction of the length of delay required to maintain stability, could be a crucial parameter beta in determining the disease's method of management.

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