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STABILITY ANALYSIS OF DELAYED SIR MODEL WITH LOGISTIC GROWTH AND BILINEAR INCIDENCE RATE

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Abstract. We look at a system of delay differential equations for an SIR model with logistic and bilinear incidence rates in this study. The model demonstrates bifurcation, where a stable disease-free equilibrium (DFE) coexists with a stable endemic equilibrium, according to the research (EE). When the reproduction number determines the requirements for local equilibrium stability and Hopf bifurcation's existence. In order to preserve the stability behaviour, we also performed a bifurcation analysis with an anticipated duration of delay. We used numerical simulations to demonstrate the theoretical results' relevance and effectiveness.

Keywords: delay; stability analysis; bifurcation; SIR model.

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1. INTRODUCTION

In the last few decades, mathematical modelling has been increasingly important in epidemiological theory. Various epidemic models have been developed and intensively examined, resulting in significant advancements in disease control and prevention research [1–7]. From

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model construction to differential equations to statistical analysis, a full view of disease dynamics necessitates a variety of mathematical methods. Although mathematics has performed admirably in the field of epidemiology, there is no disputing that certain elements still require suitable mathematization..

Mathematical models have their usage in devising and designing, estimating and evaluating, comparing and sub-sequentially implementing the optimum in controlling the diseases by prevention, therapy methods and other controlling programs. In the SIR models, the entire population is trifurcated as Susceptible(S), Infective(I) and Recovered or Removed(R) individuals. It is kept in view that the difficulty to acquire the details of transmission of infection disease tends to change at various condition. Besides, choosing generalized incidence rate function flexibly decides the function from incidence rate in use.

Most such mathematical models of disease begin with the same premise: that the population can be classified into a collection of discrete groups based on their experience with the disease [8]. Nonlinear differential equations are used to describe the majority 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) 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 *SIR* (susceptible, infectious, recovered) models [10–13].

In this study, we add a discrete time delay to the model to reflect the time it takes for a suspected individual to become infectious; this is referred to as the incubation period. A system of delay-differential equations emerges as a result of the process. We analyse the transcendental characteristic equation of the linearized system at the positive infected steady

state and attempt to understand the dynamics of the delay model and determine the analytic circumstances under which the infected steady state is stable. To illustrate the acquired results, numerical simulations are run.

The following is an overview of the paper's structure. In Section 2, we first propose the *SIR* epidemic model with time delay system, after which we verify the existence of equilibria 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 MODEL

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

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

For the purposes of this discussion, we have changed the above model (1) and added a delay to the system, as follows:

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

Here The numbers of susceptible, infectious, and recovery cells at time t are denoted by $S(t), I(t)$, 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, $beta$ is the transmission rate, mu is the natural death rate, $alpha$ is the disease-induced death rate, $gamma$ is the recovered rate, and nu is the maximum medical resources supplied per unit time and

$zeta$ is the half-saturation constant, which measures the effect of treatment delay. In this work, it is assumed that nu is a non-negative constant and that all other parameters are positive constants, with tau 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

$E_0 = (\bar{S}, \bar{I}, \bar{R})$ and the infected steady state $E_1 = (S^*, I^*, R^*)$.

The basic reproduction number:

The basic reproduction number, R_0, is the estimated number of secondary cases produced by a typical infective 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 $X = (S, I, R)^T$, then the model (2) can be written as

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

where

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

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

We are able to receive,

$$\mathscr{F} = \begin{bmatrix} 0 & \beta\bar{S} \\ 0 & 0 \end{bmatrix}, \quad \mathscr{V} = \begin{bmatrix} 0 & (\mu+\alpha+\gamma)+\nu \\ -r+\dfrac{2r\bar{S}}{K}+\beta\bar{I} & \beta\bar{S} \end{bmatrix}.$$

It then follows that the spectral radius of matrix $\mathscr{F}\mathscr{V}^{-1}$ is, $\rho(\mathscr{F}\mathscr{V}^{-1}) = \dfrac{\beta K}{\mu+\alpha+\gamma+\nu}$. According to Theorem 2 in [16], the reproduction number of model (2) is

$$R_0 = \frac{\beta K}{\mu+\alpha+\gamma+\nu}. \tag{3}$$

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 $E_1(S^*,I^*,R^*)$ (i.e., $S^*>0, I^*>0, R^*>0$) where S^*, I^* and R^* are given in the proof.*

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

$$\begin{aligned} rS^*\left(1-\frac{S^*}{K}\right) - \beta S^*I^* &= 0, \\ \beta S^*I^* - (\mu+\alpha+\gamma)I^* - \frac{\nu I^*}{1+\zeta I^*} &= 0, \\ \gamma I^* + \frac{\nu I^*}{1+\zeta I^*} - \mu R^* &= 0. \end{aligned} \tag{4}$$

From the above equation (4), we easily find that

$$\begin{aligned} S^* &= \frac{-B \pm \sqrt{B^2 - 4AC}}{2A}, \\ I^* &= \frac{r}{\beta}\left(1 - \frac{S^*}{K}\right) \\ R^* &= \frac{1}{\mu}\left(\gamma I^* + \frac{\nu I^*}{1+\zeta I^*}\right). \end{aligned}$$

where

$$\begin{aligned} A &= \frac{\zeta r}{K} \\ B &= -\left(\beta + \zeta r + \frac{\zeta r}{\beta K}(\mu+\alpha+\gamma)\right) \\ C &= \mu+\alpha+\gamma+\nu+\frac{\zeta r}{\beta}(\mu+\alpha+\gamma) \end{aligned}$$

Hence, the system (2) has a unique equilibrium $E_1(S^*, I^*, R^*)$ 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 (3). 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 of DDE Model

We adopt the following notation: $\mathbb{R}^3$ is a four-dimensional real Euclidean space with norm $|.|$. For $\tau > 0$, we denote by $C = C([-\tau, 0], \mathbb{R}^3_+)$, the Banach space of continuous function mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$ with the topology of uniform convergence. By the standard theory of functional differential equation [17–19], we know that for any $\phi \in C([-\tau, 0], \mathbb{R}^3_+)$, there exists a unique solution

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

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

$$S(\xi) = \phi_1(\xi), \quad I(\xi) = \phi_2(\xi), \quad R(\xi) = \phi_3(\xi). \tag{5}$$

Theorem 3.1. *Let $Z(t,\phi)$ be the solution of the delayed system (2) with the initial conditions (5). Then $S(t), I(t)$ 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) has a infected steady state $E_1 = (S^*, I^*, R^*)$. Then the linearized system (2) at E_1 yields

$$\begin{vmatrix} \left(r - \frac{2rS^*}{K} - \beta I^*\right) - \lambda & -\beta S^* & 0 \\ \beta I^* e^{-\lambda\tau} & \left(\beta S^* e^{-\lambda\tau} - (\mu+\alpha+\gamma) - \frac{\nu}{(1+\zeta I^*)^2}\right) - \lambda & 0 \\ 0 & \gamma + \frac{\nu}{(1+\zeta I^*)^2} & -\mu - \lambda \end{vmatrix} = 0,$$

Thus the characteristic polynomial of the above determinant as follows,

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 + e^{-\lambda\tau}(B_1\lambda^2 + B_2\lambda + B_3) = 0. \tag{6}$$

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 (6) an be rewritten as

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

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

Also,

$$\begin{aligned}
A_1 &= \alpha+\gamma+\frac{\nu}{(1+\zeta I^*)^2}-a,\\
A_2 &= a\mu+\left(\frac{\nu}{(1+\zeta I^*)^2}+(\mu+\alpha+\gamma)\right)(\mu-a),\\
A_3 &= -a\mu\left(\frac{\nu}{(1+\zeta I^*)^2}+\mu+\alpha+\gamma\right),\\
B_1 &= -\beta S^*,\\
B_2 &= \beta^2 S^* I^*+a\beta S^*-\mu\beta S^*,\\
B_3 &= a\mu\beta S^*+\beta^2 S^* I^*\mu,\\
a &= r-\frac{2rS^*}{K}-\beta I^*
\end{aligned}$$

If the equation (6) has a purely imaginary root $\lambda = i\omega$, with $\omega > 0$, separating the real and imaginary parts, we obtain the system of transcendental equations as

$$A_1\omega^2 - A_3 = (B_3 - B_1\omega^2)\cos(\omega\tau) + B_2\omega\sin(\omega\tau), \tag{7}$$

$$\omega^3 - A_2\omega = B_2\omega\cos(\omega\tau) - (B_3 - B_1\omega^2)\sin(\omega\tau). \tag{8}$$

Squaring and adding (7) and (8), we obtain

$$\omega^6 + C_1\omega^4 + C_2\omega^2 + C_3 = 0. \tag{9}$$

where

$$\begin{aligned}
C_1 &= A_1^2 - 2A_2 - B_1^2,\\
C_2 &= A_2^2 - B_2^2 - 2A_1A_3 + 2B_1B_3,\\
C_3 &= A_3^2 - B_3^2.
\end{aligned}$$

Put $\omega^2 = \rho$ in (9), we get

$$h(\rho) = \rho^3 + C_1\rho^2 + C_2\rho + C_3 = 0. \tag{10}$$

From the hypothesis of $R_0 > 1$, we deduce that $C_3 = A_3^2 - B_3^2 > 0$, since for $R_0 > 1$, C_1 and C_2 are also positive. Thus the equation (10) has no viable solution for $R_0 > 1$.

□

We will now assume that $C_3 < 0$, utilising Descartes' rule of signs, equation (10) has positive root ρ and thus equation (9) has a pair of purely imaginary roots $i\omega^*$. From equation (7) and (8), we obtain

$$\begin{aligned}\tau^* &= \frac{1}{\omega^*}\arccos\left(\frac{(A_1\omega^{*2}-A_3)(B_3-B_1\omega^{*2})+B_2\omega^{*2}(A_2-\omega^{*2})}{(B_3-B_1\omega^{*2})^2+B_2^2\omega^{*2}}\right)\\ &\quad +\frac{2j\pi}{\omega^*}, \quad \text{where } j=0,1,2,...,\end{aligned}$$

Now we find sign $\left(\frac{dRe(\lambda)}{d\tau}\right)\Big|_{\tau=\tau^*}$, where sign is the signum function and $Re(\lambda)$ is the real part of λ. Using mathematical calculations, we may conclude that the model's (2) infected steady state remains stable for $\tau < \tau^*$ and Hopf bifurcation occurs when $\tau = \tau^*$.

We first consider for purely imaginary roots of $\lambda = i\omega^*$ of equation (6) implies

$$|P(i\omega^*)| = |Q(i\omega^*)|$$

Differentiating (6) with respect to τ, we have

$$\left\{(3\lambda^2+2A_1\lambda+A_2)+e^{-\lambda\tau}(2B_1\lambda+B_2)-\tau e^{-\lambda\tau}(B_1\lambda^2+B_2\lambda+B_3)\right\}\frac{d\lambda}{d\tau} = \lambda e^{-\lambda\tau}(B_1\lambda^2+B_2\lambda+B_3)$$

which implies,

$$\begin{aligned}\left(\frac{d\lambda}{d\tau}\right)^{-1} &= \frac{3\lambda^2+2A_1\lambda+A_2}{\lambda e^{-\lambda\tau}(B_1\lambda^2+B_2\lambda+B_3)}+\frac{2B_1\lambda+B_2}{\lambda(B_1\lambda^2+B_2\lambda+B_3)}-\frac{\tau}{\lambda},\\ &= \frac{3\lambda^2+2A_1\lambda+A_2}{-\lambda(\lambda^3+A_1\lambda^2+A_2\lambda+A_3)}+\frac{2B_1\lambda+B_2}{\lambda(B_1\lambda^2+B_2\lambda+B_3)}-\frac{\tau}{\lambda},\\ &= \frac{2\lambda^3+A_1\lambda^2-A_3}{-\lambda^2(\lambda^3+A_1\lambda^2+A_2\lambda+A_3)}+\frac{B_1\lambda^2-B_3}{\lambda^2(B_1\lambda^2+B_2\lambda+B_3)}-\frac{\tau}{\lambda}\end{aligned}$$

Therefore,

$$\begin{aligned}
\Xi &= sign\left\{Re\left(\frac{2\lambda^3+A_1\lambda^2-A_3}{-\lambda^2(\lambda^3+A_1\lambda^2+A_2\lambda+A_3)}+\frac{B_1\lambda^2-B_3}{\lambda^2(B_1\lambda^2+B_2\lambda+B_3)}-\frac{\tau}{\lambda}\right)\right\}_{\lambda=i\omega^*} \\
&= \frac{1}{\omega^{*2}}sign\left\{Re\left(\frac{(A_3+A_1\omega^{*2})+i2\omega^{*3}}{(A_1\omega^{*2}-A_3)+i(\omega^{*3}-A_2\omega^*)}+\frac{B_1\omega^{*2}+B_3}{(B_3-B_1\omega^{*2})+i(B_2\omega^*)}\right)\right\} \\
&= \frac{1}{\omega^{*2}}sign\left\{\left(\frac{(A_3+A_1\omega^{*2})(A_1\omega^{*2}-A_3)+2\omega^{*3}(\omega^{*3}-A_2\omega^*)}{(A_1\omega^{*2}-A_3)^2+(\omega^{*3}-A_2\omega^*)^2}+\frac{(B_1\omega^{*2}+B_3)(B_3-B_1\omega^{*2})}{(B_3-B_1\omega^{*2})^2+(B_2\omega^*)^2}\right)\right\} \\
&= \frac{1}{\omega^{*2}}sign\left\{\left(\frac{(A_3+A_1\omega^{*2})(A_1\omega^{*2}-A_3)+2\omega^{*3}(\omega^{*3}-A_2\omega^*)+(B_1\omega^{*2}+B_3)(B_3-B_1\omega^{*2})}{(B_3-B_1\omega^{*2})^2+(B_2\omega^*)^2}\right)\right\} \\
&= \frac{1}{\omega^{*2}}sign\left\{\left(\frac{2\omega^{*6}+(A_1^2-2A_2-B_1^2)\omega^{*4}+(A_3^2-B_3^2)}{(B_3-B_1\omega^{*2})^2+(B_2\omega^*)^2}\right)\right\}
\end{aligned}$$

and this yields a list of probable eigenvalues of ω^*. Our goal is to figure out which way λ moves while τ is changed. i.e., we make a decision,

$$\Xi = sign\left\{\left(\frac{d(Re(\lambda))}{d\tau}\right)\right\}_{\lambda=i\omega^*} = sign\left\{Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\right\}_{\lambda=i\omega^*}$$

As $A_1^2-2A_2-B_1^2$ and $A_3^2-B_3^2$ are both positive by virtue of equation (9), we have $\left(\frac{dRe(\lambda)}{d\tau}\right)\Big|_{\omega=\omega^*,\tau=\tau^*} > 0$. Thus, the solution curve of the characteristic equation (9) crosses the imaginary axis. This demonstrates that at $0<\tau=\tau^*$., a Hopf bifurcation occurs. When $\tau<\tau^*$. The infected steady state is locally asymptotically stable by continuity. We arrive to the following theorem by summarizing the above analysis.

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 $E_1(S^*,I^*,R^*)$ are given by,

$$ImH(i\omega_0)>0, \tag{11}$$

$$ReH(i\omega_0)=0, \tag{12}$$

where $H(\rho) = \rho^3 + A_1\rho^2 + A_2\rho + A_3 + e^{-\rho\tau}(B_1\rho^2 + B_2\rho + B_3)$ and ω_0 is the smallest positive root of (12).

Inequality (11) and (12) can alternatively be written as

$$A_2\omega_0 - \omega_0^3 > -B_2\omega_0\cos(\omega_0\tau) + B_3\sin(\omega_0\tau) - B_1\omega_0^2\sin(\omega_0\tau), \tag{13}$$

$$A_3 - A_1\omega_0^2 = B_1\omega_0^2\cos(\omega_0\tau) - B_3\cos(\omega_0\tau) - B_2\omega_0\sin(\omega_0\tau). \tag{14}$$

Now if (13) and (14) 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 ω_+ to ω_0 from (14) that is independent of τ, and then to estimate τ so that (13) holds true for all values of ω such that $0 \le \omega \le \omega_+$, and hence, in particular at $\omega = \omega_0$.

Equation (14) can be rewritten as

$$A_1\omega_0^2 = A_3 - B_1\omega_0^2\cos(\omega_0\tau) + B_3\cos(\omega_0\tau) + B_2\omega_0\sin(\omega_0\tau). \tag{15}$$

Maximizing the right hand side of (15) subject to,

$$|\sin(\omega_0\tau)| \le 1, \qquad |\cos(\omega_0\tau)| \le 1, \tag{16}$$

we obtain

$$|A_1|\omega_0^2 \le |A_3| + |B_3| + |B_1|\omega_0^2 + |B_2|\omega_0. \tag{17}$$

Hence if,

$$\omega_+ = \frac{1}{2(|A_1| - |B_1|)}\left\{|B_2| + \sqrt{B_2^2 + 4(|A_1| - |B_1|)(|A_3| + |B_3|)}\right\}, \tag{18}$$

then clearly from (17) we have $\omega_0 \le \omega_+$.

From (13), we obtain

$$\omega_0^2 < A_2 + B_2\cos(\omega_0\tau) + B_1\omega_0\cos(\omega_0\tau) - \frac{B_3\sin(\omega_0\tau)}{\omega_0}. \tag{19}$$

Since $E_1(S^*,I^*,R^*)$ is locally asymptotically stable for $\tau = 0$, the inequality (19) will continue to hold for sufficiently small $\tau > 0$. Using (15) and (19) can be rearranged as

$$\begin{aligned}\left(B_3 - B_1\omega_0^2 - A_1B_2\right)\left(\cos(\omega_0\tau) - 1\right) + \left((B_2 - A_1B_1)\omega_0 + \frac{A_1B_3}{\omega_0}\right)\sin(\omega_0\tau) \\ < A_1A_2 - A_3 - B_3 + B_1\omega_0^2 + A_1B_2. \end{aligned} \tag{20}$$

Using the bound

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

$$\left((B_2 - A_1B_1)\omega_0 + \frac{A_1B_3}{\omega_0}\right)\sin(\omega_0\tau) \leq \left(|(B_2 - A_1B_1)|\omega_+^2 + |A_1||B_3|\right)\tau, \tag{21}$$

we obtain from (19)

$$L_1\tau^2 + L_2\tau < L_3, \tag{22}$$

where,

$$\begin{aligned} L_1 &= \frac{1}{2}|(-B_3 + B_1\omega_0^2 + A_1B_2)|\omega_+^2, \\ L_2 &= |(B_2 - A_1B_1)|\omega_+^2 + |A_1||B_3|, \\ L_3 &= A_1A_2 - A_3 - B_3 + B_1\omega_+^2 + A_1B_2. \end{aligned} \tag{23}$$

Hence if,

$$\tau_+ = \frac{1}{2L_1}\left(Ł_2 + \sqrt{L_2^2 + 4L_1L_3}\right), \tag{24}$$

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. Application with 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.

$$r = 0.7, 1.3, 1.3, \quad \beta = 0.02, 0.3, 0.02, \quad K = 0.8, 0.05, 0.08, \quad \mu = 0.5, 0.9, 0.2,$$

$$\alpha = 0.1, 0.05, 0.5 \quad \gamma = 0.05, 0.2, 0.1, \quad \zeta = 0.01, 0.3, 0.5, \quad \nu = 0.1, 0.4, 0.1.$$

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 $(S(t), I(t), R(t))$ are the solutions of System (2) with initial conditions

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

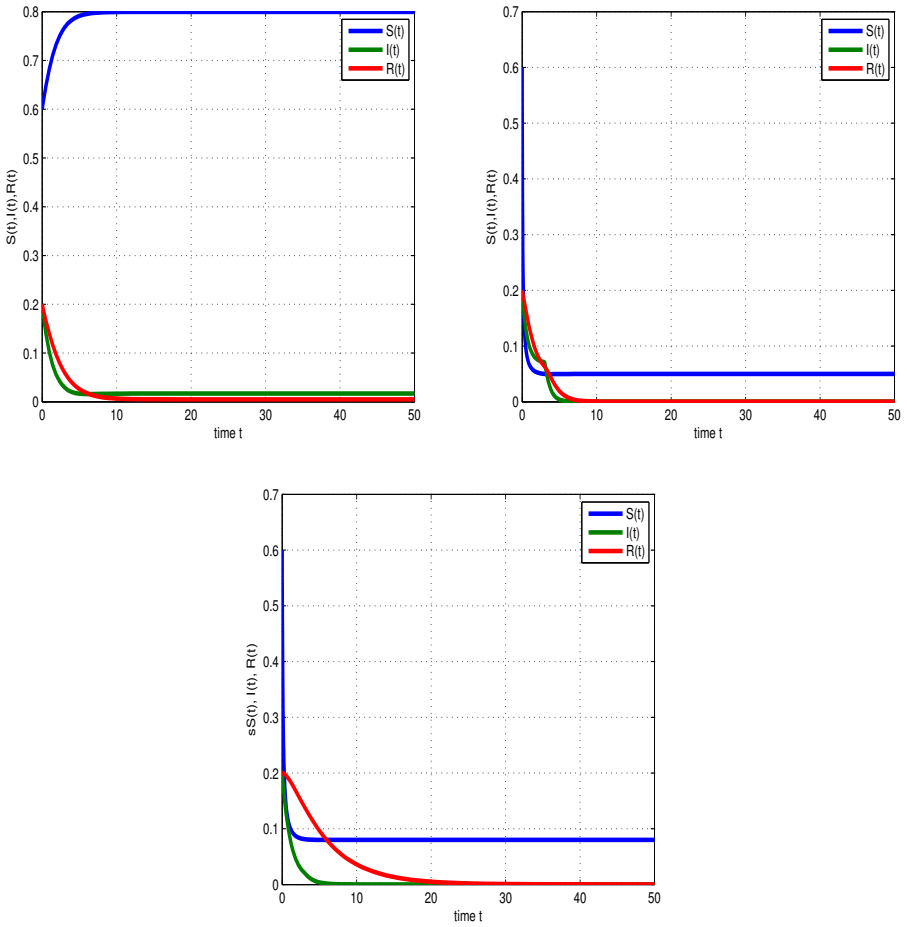


FIGURE 1: Solutions of the system (2) go to the disease-free steady state, where $S(t)$ represents the suspected cells, $I(t)$ represents the infected cells, $R(t)$ represents recovered cells.

(ii) We choose the different values of parameters satisfying the conditions in Theorem 3.2 as follows.

$$r = 0.7, 1.3, 1.3, \ \beta = 0.2, 0.3, 0.2, \ K = 8, 18, 10, \ \mu = 0.5, 0.9, 0.2,$$

$$\alpha = 0.1, 0.05, 0.5 \ \gamma = 0.05, 0.2, 0.1, \ \zeta = 0.01, 0.3, 0.5, \ \nu = 0.1, 0.4, 0.1.$$

Then we have $R_0 = 2.13, 3.48, 2.22 > 1$, and due to Theorem 3.2 the infected equilibrium E_1 of System (2) is asymptotically stable which is shown well in Fig. 2. Here $(S(t), I(t), R(t))$ are the solutions of System (2) with initial conditions

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

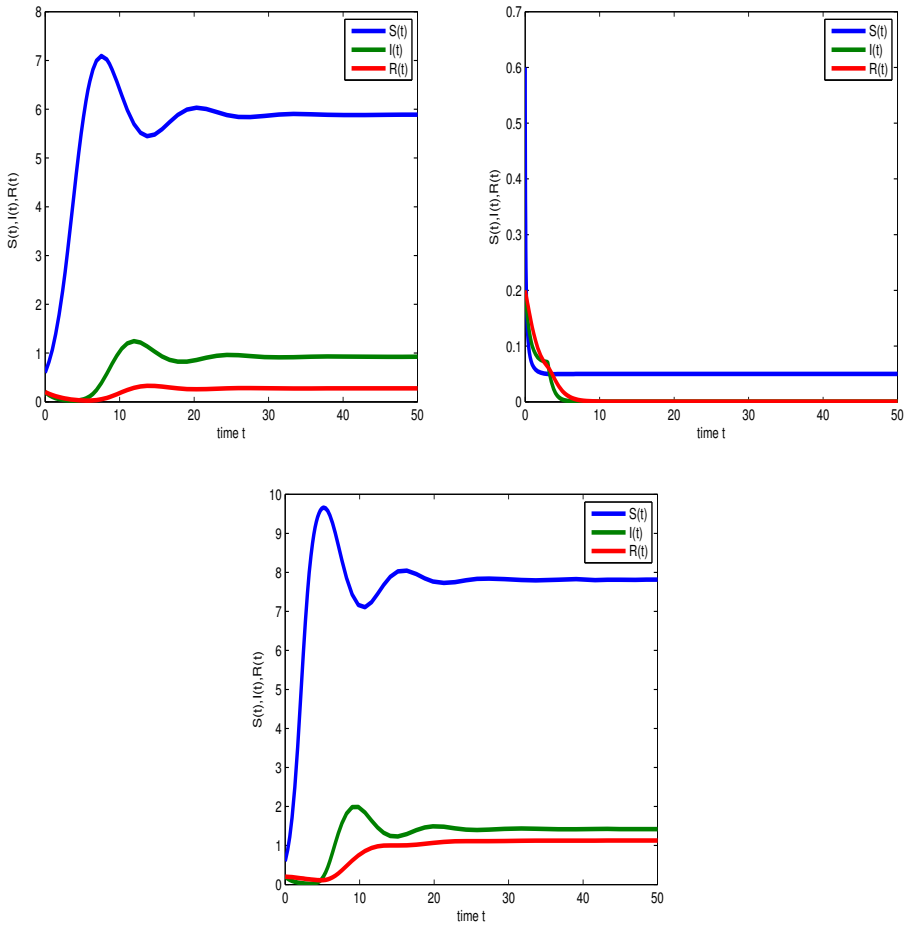


FIGURE 2: Solutions of the system (2) go to the steady state, where $S(t)$ represents the suspected cells, $I(t)$ represents the infected cells, $R(t)$ represents recovered cells.

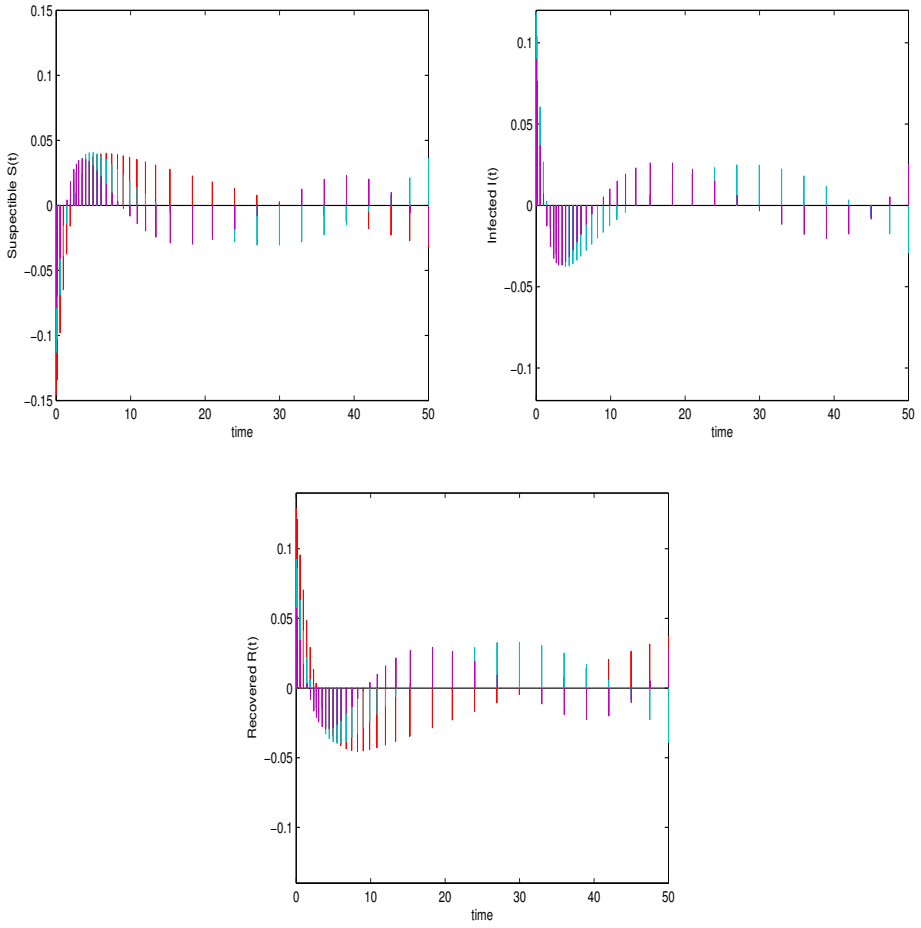
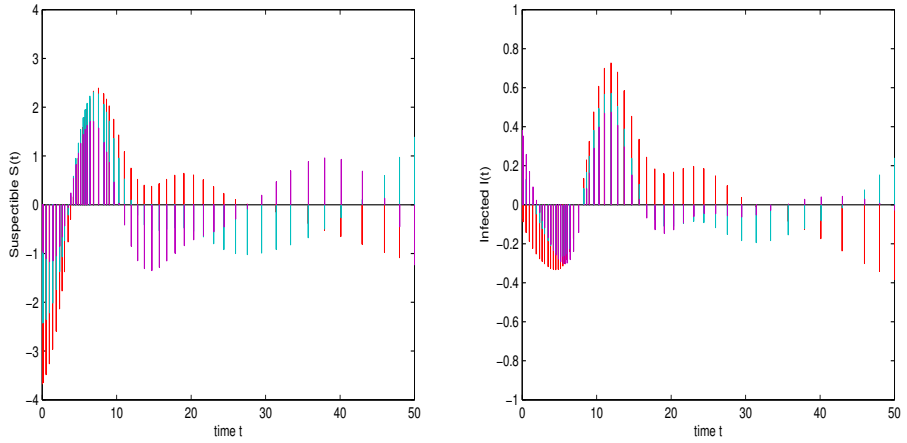


FIGURE 3: The trajectories of solutions of the system (2) and its basic curve fitting with $(S(0), I(0), R(0)) = (0.6, 0.2, 0.2)$ and $R_0 < 1$.



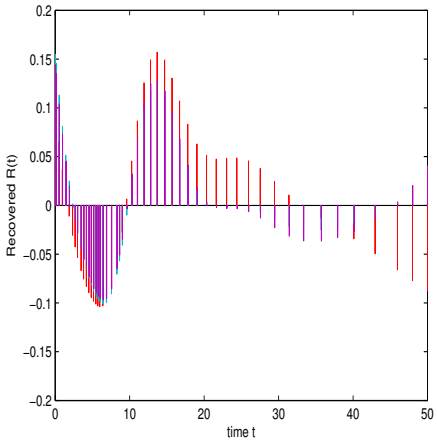


FIGURE 4: The trajectories of solutions of the system (2) and its basic curve fitting with $(S(0), I(0), R(0)) = (0.6, 0.2, 0.2)$ and $R_0 > 1$.

From the preceding figures (1-4), we can see that our delay model (2) is rather reliable. As a result, our numerical results are appropriate for representing our model. Furthermore, slight changes in parameters will result in tiny changes in the matrix entries required to find eigenvalues and determine the stability of two steady-state locations.

Our numerical calculation's stability results are biologically justifiable and represent the best possible outcome for the parameter values.

6. Discussion and Conclusion

We have included time delay in our SIR 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.

Conflict of Interests

The authors declare that there is no conflict of interests.

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