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Research Article

Qualitative Analysis of Delayed SIR Epidemic Model with a Saturated Incidence Rate

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We consider a delayed SIR epidemic model in which the susceptibles are assumed to satisfy the logistic equation and the incidence term is of saturated form with the susceptible. We investigate the qualitative behaviour of the model and find the conditions that guarantee the asymptotic stability of corresponding steady states. We present the conditions in the time lag τ in which the DDE model is stable. Hopf bifurcation analysis is also addressed. Numerical simulations are provided in order to illustrate the theoretical results and gain further insight into the behaviour of this system.

1. Introduction

Epidemics have ever been a great concern of human kind, because the impact of infectious diseases on human and animal is enormous, both in terms of suffering and social and economic consequences. Mathematical modeling is an essential tool in studying a diverse range of such diseases to gain a better understanding of transmission mechanisms, and make predictions; determine and evaluate control strategies. Many authors have proposed various kinds of epidemic models to understand the mechanism of disease transmission (see [1–10] and references therein). The basic elements for the description of infectious diseases have been considered by three epidemiological classes: $S(t)$ that measures the susceptible portion of population, $I(t)$ the infected, and $R(t)$ the removed ones. Kermack and McKendrick [11] described the simplest SIR model which computes the theoretical number of people infected with a contagious illness in a closed population over time. Transmission of a disease is a dynamical process driven by the interaction between susceptible and infective. The behaviour of the SIR models are greatly affected by the way in which transmission between infected and susceptible individuals are modelled. The simplest model in which recovery does not give immunity is the SIS model, since individuals move from the susceptible class to

the infective class and then back to the susceptible class upon recovery. If individuals recover with permanent immunity, then the simplest model is an SIR model. If individuals recover with temporary immunity so that they eventually become susceptible again, then the simplest model is an SIRS model. If individuals do not recover, then the simplest model is an SI model. In general, SIR (epidemic and endemic) models are appropriate for viral agent diseases such as measles, mumps, and smallpox, while SIS models are appropriate for some bacterial agent diseases such as meningitis, plague, and sexually transmitted diseases, and for protozoan agent diseases such as malaria and sleeping sickness. Modelling and analysis of such infectious diseases have been done by many scientists; see, for example, [8, 12–22] and the references therein.

Epidemiological models with latent or incubation period have been studied by many authors, because many diseases, such as influenza and tuberculosis, have a latent or incubation period, during which the individual is said to be infected but not infectious. Delay differential equations (DDEs) have been successfully used to model varying infectious period in a range of SIR, SIS, and SIRS epidemic models. Hethcote and van den Driessche [23] have considered an SIS epidemic model with constant time delay, which accounts for duration of infectiousness. Beretta et al. [24] have studied global stability in an SIR epidemic model with distributed delay that describes the time which it takes for an individual to lose infectiousness. Song and Cheng [25] have studied the effect of time delay on the stability of the endemic equilibrium. They gave some conditions for which the endemic equilibrium is asymptotically stable for all delays and also discussed the existence of orbitally asymptotically stable periodic solutions. The mathematical analysis of epidemiological modelling is often used for the assessment of the global asymptotic stability of both the disease free and endemic equilibrium. However, most of the obtained epidemic/endemic delay models are stiff (one definition of the stiffness is that the global accuracy of the numerical solution is determined by stability rather than local error and implicit methods are more appropriate for it; see [26]) and need a special care in their analysis and numerical treatment. The state variables are also very sensitive to small perturbations (or changes) in the initial conditions and parameters which occur in the model.

The contact rate is often a function of population density, reflecting the fact that contacts take time and saturation occurs. In this paper, we consider a delayed SIR epidemic model with time-delay and incidence rate of saturated form with the susceptibles. Qualitative analysis of the model with constant infectious period is carried out. We present the conditions in the time lag τ in which the DDE model is stable. Hopf bifurcation analysis is also addressed and results of simulation scenarios are presented.

2. Classic SIR Epidemic Models

Let the SIR model be based on the following assumptions: (i) susceptible individuals are born at a rate $\mu(S; I; R)$ which is assumed to be a function of the densities of the susceptible, infected and recovered hosts; (ii) susceptibles are infected at a rate given by the product of the densities of susceptible and infected hosts which times a proportionality constant β describing the infectivity rate per contact between the two types of host. The assumption to model the infection rate proportional to SI is justified if both hosts types are well mixed and the encounter between the two host types is random. This assumption is often called mass-action kinetics and derives from chemical kinetics; (iii) infected hosts recover at a rate α; (iv) susceptible and recovered hosts die at a rate δ which describes the natural death rate due to

causes unrelated to infection. (v) Infected hosts die at a rate a which includes both the natural death rate plus the disease induced death rate. One arrives at the model of the form

$$\begin{aligned} S'(t) &= \mu - \delta S(t) - \beta S(t) I(t), \\ I'(t) &= \beta S(t) I(t) - aI(t) - \alpha I(t), \\ R'(t) &= \alpha I(t) - \delta R(t). \end{aligned} \tag{2.1}$$

It is worth mentioning that in the above model many possibly relevant biological aspects, such as age structure of the population, were ignored. The infectivity, death rate, and rate of recovery may all depend on the age of the infected individual. Moreover, spatial structure is also ignored. Often transmission to cohabiting individuals (i.e., members of the family) occurs with increased probability. Often the total population size remains roughly constant over the period of interest (such as the time for an epidemic to occur). To study the qualitative behaviors of the above model at the equilibrium points, we assume that $N = S + I + R$ is constant and therefore $dN/dt = dS/dt + dI/dt + dR/dt = 0$. Summing up equations of model (2.1), we thus obtain for the birth rate

$$\mu = \delta(S + R) + aI. \tag{2.2}$$

If the total population size is assumed to be constant, we can drop the variable R (since $R = N - S - I$). We thus obtain

$$\begin{aligned} S'(t) &= \delta(N(t) - S(t) - I(t)) + aI(t) - \beta S(t) I(t), \\ I'(t) &= \beta S(t) I(t) - aI(t) - \alpha I(t). \end{aligned} \tag{2.3}$$

The above model has two equilibria:

$$E_0 = (N, 0), \qquad E_+ = (S^*, I^*) = \left(\frac{a + \alpha}{\beta}, \frac{\delta S^*}{\delta + \alpha} (\mathcal{R}_0^* - 1) \right), \tag{2.4}$$

where $\mathcal{R}_0^* = \beta N/(a + \alpha)$ is the reproduction number (reproduction number denotes the number of individuals infected by a single infected individual placed in a totally susceptible population). The first equilibrium represents the case where none of the individuals are infected (free-infection). The second equilibrium represents the case where a fraction of the individuals are infected (infected equilibrium, or endemic equilibrium), when $\mathcal{R}_0^* > 1$.

If a disease is not of short duration, then several changes need to be made to the SIR model. Saturating contact rate of individual contacts is very important in an epidemiology model. For more convenience and a practical point of view, model (2.1) is modified so that the susceptible host population is assumed to have the logistic growth $rS(1 - S/K)$ with carrying capacity K (in a closed community) and a specific growth rate constant r. The

bilinear transmission incidence rate βSI is also replaced by Holling type functional response term $\beta SI/(1+\sigma S)$, which is saturated with the susceptible. Model (2.1) takes the form

$$\begin{aligned} S'(t) &= rS(t)\left(1-\frac{S(t)}{K}\right)-\frac{\beta S(t)I(t)}{1+\sigma S(t)}, \\ I'(t) &= \frac{\beta S(t)I(t)}{1+\sigma S}-aI(t)-\alpha I(t), \\ R'(t) &= \alpha I(t)-\delta R(t), \end{aligned} \tag{2.5}$$

where σ is the saturation factor that measures the inhibitory effect. The resulting model can show oscillatory behaviors that are called epidemic waves. Stability and oscillatory behavior of system (2.5) have been studied in [27].

3. Delayed SIR Epidemic Model

Assuming that the incubation period $\tau > 0$ is a time, during which the infectious agents develop in the vector, and only after that time the infected vector becomes itself infectious, model (2.5) is then generalized into a delayed SIR epidemiological model of the form

$$\begin{aligned} S'(t) &= rS(t)\left(1-\frac{S(t)}{K}\right)-\frac{\beta S(t)I(t-\tau)}{1+\sigma S(t)}, \\ I'(t) &= \frac{\beta S(t)I(t-\tau)}{1+\sigma S(t)}-aI(t)-\alpha I(t), \\ R'(t) &= \alpha I(t)-\delta R(t). \end{aligned} \tag{3.1}$$

All feasible solutions of (3.1) are bounded (see [28]) and the dynamics of model are mainly determined by the first two equations:

$$\begin{aligned} S'(t) &= rS(t)\left(1-\frac{S(t)}{K}\right)-\frac{\beta S(t)I(t-\tau)}{1+\sigma S(t)}, \\ I'(t) &= \frac{\beta S(t)I(t-\tau)}{1+\sigma S(t)}-aI(t)-\alpha I(t). \end{aligned} \tag{3.2}$$

Proposition 3.1. *For the model system* (3.2)*, there always exist infection-free equilibria* $E_0=(0,0)$, $E_1=(K,0)$. *If*

$$\mathcal{R}_0=\frac{K\left[\beta-\sigma(a+\alpha)\right]}{(a+\alpha)}>1, \tag{3.3}$$

there also exists an endemic equilibrium $E_+=(S^*,I^*)$, *where*

$$E_+=(S^*,I^*)=\left(\frac{a+\alpha}{\beta-\sigma(a+\alpha)},\frac{rS^{*2}}{K(a+\alpha)}(\mathcal{R}_0-1)\right). \tag{3.4}$$

3.1. Infection-Free Equilibria and Their Stabilities

The Jacobian matrix of the linearized system of model (3.2) is

$$J = \begin{pmatrix} r - \dfrac{2rS^*}{K} - \dfrac{\beta I^*}{(1+\sigma S^*)^2} & -\dfrac{\beta S^*}{1+\sigma S^*} e^{-\lambda\tau} \\ \dfrac{\beta I^*}{(1+\sigma S^*)^2} & \dfrac{\beta S^*}{1+\sigma S^*} e^{-\lambda\tau} - (a+\alpha) \end{pmatrix}. \tag{3.5}$$

Using (3.5), the characteristic equation at the trivial equilibrium point $E_0 = (0,0)$ reduces to

$$(\lambda - r)(\lambda + (a+\alpha)) = 0. \tag{3.6}$$

Obviously, (3.6) has a positive root $\lambda = r$. Then the trivial equilibrium E_0 of system (3.2) is always unstable (saddle point). However, at the infection-free equilibrium $E_1 = (K,0)$, the Jacobian matrix (3.5) reduces to

$$J_{\text{infection-free}} = \begin{pmatrix} -r & -\dfrac{\beta K}{1+\sigma K} e^{-\lambda\tau} \\ 0 & \dfrac{\beta K}{1+\sigma K} e^{-\lambda\tau} - (a+\alpha) \end{pmatrix} \tag{3.7}$$

with characteristic equation

$$(\lambda + r)\left(\lambda + (a+\alpha)\left[1 - \frac{\mathcal{R}_0 + \sigma K}{1+\sigma K} e^{-\lambda\tau}\right]\right) = 0. \tag{3.8}$$

It is obvious from (3.8) that the two roots are real and negative if $\mathcal{R}_0 < 1$ (when $\tau = 0$) and the equilibrium E_1 is then asymptotically stable. In case of $\tau > 0$, we assume that the root of (3.8) $\lambda = \xi i$ must satisfy

$$\xi^2 = (a+\alpha)^2\left[\frac{\mathcal{R}_0 + \sigma K}{1+\sigma K} - 1\right]. \tag{3.9}$$

Then, when $\mathcal{R}_0 < 1$, then there are no positive real roots ξ. Therefore, we arrive at the following theorem to indicate the stability of E_1.

Theorem 3.2. *If $\mathcal{R}_0$ is defined by* (3.3)*, then infection-free equilibrium $E_1 = (K,0)$ of system* (3.2) *is asymptotically stable when $\mathcal{R}_0 < 1$, and unstable when $\mathcal{R}_0 > 1$, and linearly neutrally stable if $\mathcal{R}_0 = 1$.*

3.2. Endemic Equilibrium and Its Stability

Here we investigate the linear stability of (3.2) at the endemic equilibrium $E_+ = (S^*, I^*)$ defined in (3.4). $S^* = (a+\alpha)/(\beta - \sigma(a+\alpha)) \Rightarrow \beta S^*/(1+\sigma S^*) = (a+\alpha)$, and $I^* = (rS^{*2}/K(a+\alpha))(\mathcal{R}_0 - 1) \Rightarrow \beta I^*/(1+\sigma S^*) = r(1 - 1/\mathcal{R}_0)$. We also have $\mathcal{R}_0 = K/S^*$. Therefore,

the corresponding Jacobian matrix at the endemic equilibrium E_+ can be easily expressed in terms of the reproduction number $\mathcal{R}_0$ as follows:

$$J_{\text{endemic}} = \begin{pmatrix} r\left(1-\frac{2}{\mathcal{R}_0}\right) - \frac{r}{1+\sigma S^*}\left(1-\frac{1}{\mathcal{R}_0}\right) & -(a+\alpha)e^{-\lambda\tau} \\ \frac{r}{1+\sigma S^*}\left(1-\frac{1}{\mathcal{R}_0}\right) & (a+\alpha)e^{-\lambda\tau} - (a+\alpha) \end{pmatrix}. \tag{3.10}$$

The characteristic equation of (3.10) for the endemic equilibrium is

$$\begin{aligned} &\lambda^2 + \lambda\left[-r\left(1-\frac{2}{\mathcal{R}_0}\right) + (a+\alpha)\left(1-e^{-\lambda\tau}\right) + \frac{r}{1+\sigma S^*}\left(1-\frac{1}{\mathcal{R}_0}\right)\right] \\ &\quad + (a+\alpha)\left[-r\left(1-\frac{2}{\mathcal{R}_0}\right)\left(1-e^{-\lambda\tau}\right) + \frac{r}{1+\sigma S^*}\left(1-\frac{1}{\mathcal{R}_0}\right)\right] = 0. \end{aligned} \tag{3.11}$$

We need to find the necessary and sufficient condition for every root of the characteristic equation (3.11) having negative real part. Introducing

$$\varrho_1 = r\left(1-\frac{2}{\mathcal{R}_0}\right), \qquad \varrho_2 = \frac{r}{1+\sigma S^*}\left(1-\frac{1}{\mathcal{R}_0}\right), \qquad \varrho_3 = (a+\alpha) \tag{3.12}$$

then the characteristic equation (3.11) can be rewritten in the form

$$\lambda^2 + \lambda\left(-\varrho_1 + \varrho_2 + \varrho_3\right) + \varrho_3\left(-\varrho_1 + \varrho_2\right) + e^{-\lambda\tau}\left(-\varrho_3\lambda + \varrho_1\varrho_3\right) = 0. \tag{3.13}$$

For simplicity assume also that

$$A_1 = \left(-\varrho_1 + \varrho_2 + \varrho_3\right), \qquad A_2 = \varrho_3\left(-\varrho_1 + \varrho_2\right), \qquad A_3 = \varrho_3, \qquad A_4 = \varrho_1\varrho_3, \tag{3.14}$$

then (3.13) takes the form

$$\lambda^2 + A_1\lambda + A_2 + e^{-\lambda\tau}(-A_3\lambda + A_4) = 0. \tag{3.15}$$

Theorem 3.3. *Assume that* $\mathcal{R}_c = 2 + 1/(1+2\sigma S^*)$. *Then*

(i) *the endemic equilibrium* E_+ *of system* (3.2) *is feasible and locally asymptotically stable for all* $\tau \geq 0$ *if* $1 < \mathcal{R}_0 \leq \mathcal{R}_c$ *holds;*

(ii) *if* $\mathcal{R}_0 > \mathcal{R}_c > 1$, *then there exist* $\tau^* > 0$ *such that* $\tau \in [0, \tau^*)$ *the endemic equilibrium* E_+ *is asymptotically stable, and unstable when* $\tau > \tau^*$. *When* $\tau = \tau^*$, *the characteristic equation* (3.15) *has a pair of purely imaginary roots* $\pm i\xi_0$, *with*

$$\begin{aligned} \xi_0^2 &= \frac{1}{2}\left(2A_2 + A_3^2 - A_1^2\right) + \frac{1}{2}\sqrt{\left(2A_2 + A_3^2 - A_1^2\right)^2 - 4\left(A_2^2 - A_4^2\right)}, \\ \tau^* &= \frac{1}{\xi_0}\arccos\left(\frac{(A_4 + A_1A_3)\xi_0^2 - A_2A_4}{A_3^2\xi_0^2 + A_4^2}\right) + \frac{2j\pi}{\xi_0}, \end{aligned} \tag{3.16}$$

where A_1, A_2, A_3, *and* A_4 *are defined in* (3.14).

Proof. If $\lambda = \xi i$ is a root of (3.13). After substitution and separation the real and imaginary parts, we have

$$\begin{aligned} -\xi^2 + \varrho_3(-\varrho_1 + \varrho_2) &= \xi\varrho_3 \sin\xi\tau - \varrho_1\varrho_3\cos\xi\tau, \\ \xi(-\varrho_1 + \varrho_2 + \varrho_3) &= \varrho_1\varrho_3\sin\xi\tau + \xi\varrho_3\cos\xi\tau, \end{aligned} \tag{3.17}$$

which are equivalent to

$$\begin{aligned} -\xi^2 + A_2 &= \xi A_3 \sin\xi\tau - A_4\cos\xi\tau, \\ \xi A_1 &= A_4\sin\xi\tau + \xi A_3\cos\xi\tau. \end{aligned} \tag{3.18}$$

Squaring and adding both equations yields

$$\xi^4 + \xi^2(-\varrho_1 + \varrho_2)^2 + \varrho_2\varrho_3^2(-2\varrho_1 + \varrho_2) = 0 \tag{3.19}$$

which is equivalent to

$$\xi^4 - \left(2A_2 + A_3^2 - A_1^2\right)\xi^2 + \left(A_2^2 - A_4^2\right) = 0. \tag{3.20}$$

Equation (3.19) can also be rewritten in the form

$$\xi^4 + \xi^2\left[\varrho_1^2 + \varrho_2(-2\varrho_1 + \varrho_2)\right] + \varrho_2\varrho_3^2(-2\varrho_1 + \varrho_2) = 0. \tag{3.21}$$

Therefore, if $-2\varrho_1 + \varrho_2 \geq 0$ (when $\mathcal{R}_0 > 1$), then there is no positive real ξ satisfying (3.19). According the definitions given in (3.12), the inequality $-2\varrho_1 + \varrho_2 \geq 0$ which is equivalent to $\mathcal{R}_0 \leq 2 + 1/(1 + 2\sigma S^*)$ so that all the roots ($\lambda = \xi_i$) of (3.11) are negative.

However, if $-2\varrho_1 + \varrho_2 < 0$, then (3.21) has one and only one positive root denoted by ξ_0, and the characteristic equation (3.15) has a pair of purely imaginary roots $\pm i\xi_0$.

Let $\lambda(\tau) = \sigma(\tau) + i\xi(\tau)$ be the eigenvalue of (3.15) such that $\sigma(\tau^*) = 0$ and $\xi(\tau^*) = \xi_0$. From (3.18), we have

$$\tau^* = \frac{1}{\xi_0}\arccos\left(\frac{(A_4 + A_1A_3)\xi_0^2 - A_2A_4}{A_3^2\xi_0^2 + A_4^2}\right) + \frac{2j\pi}{\xi_0} \tag{3.22}$$

and from (3.20)

$$\xi_0^2 = \frac{1}{2}\left(2A_2 + A_3^2 - A_1^2\right) + \frac{1}{2}\sqrt{\left(2A_2 + A_3^2 - A_1^2\right)^2 - 4\left(A_2^2 - A_4^2\right)}. \tag{3.23}$$

Hence the proof is complete. □

3.3. Hopf Bifurcation Analysis

A bifurcation analysis of a dynamical system is used to understand how the solutions and their stability change as the parameters in the system vary. In particular, it can be used for the stability, analysis, and continuation of equilibria (steady-state solutions), and periodic and quasi-periodic oscillations; see [15, 21, 29].

Theorem 3.4. *Suppose that (ii) of Theorem 3.3 holds. Then there exists $\varepsilon > 0$ such that for each $0 < \varepsilon < \varepsilon^*$, system (3.2) has a family of periodic solutions $\mathcal{P} = \mathcal{P}(\varepsilon)$ with period $\mathcal{T} = \mathcal{T}(\varepsilon)$, for the parameter values $\tau = \tau(\varepsilon)$ such that $\mathcal{P}(0) = 0$, $\mathcal{T}(0) = 2\pi/\xi_0$.*

Proof. We already showed in Theorem 3.3 that the characteristic equation (3.15) has a pair of purely imaginary roots $\pm i\xi_0$. Now, we apply the Hopf bifurcation theorem introduced in [30]. Let $\lambda(\tau) = \sigma(\tau) + i\xi(\tau)$ be the eigenvalue of (3.15), we need to verify that the transversality condition

$$\left.\frac{d\Re(\lambda)}{d\tau}\right|_{\tau=\tau^*} = \left.\frac{d\sigma(\tau)}{d\tau}\right|_{\tau=\tau^*} > 0 \tag{3.24}$$

holds. Differentiating (3.15) with respect to τ, we get

$$\left[2\lambda + A_1 - A_4\tau e^{-\lambda\tau} - A_3e^{-\lambda\tau} + A_3\tau\lambda e^{-\lambda\tau}\right]\frac{d\lambda}{d\tau} = e^{-\lambda\tau}\left(A_4\lambda - A_3\lambda^2\right). \tag{3.25}$$

This gives

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{2\lambda + A_1 - e^{-\lambda\tau}[A_3 + \tau(-A_3\lambda + A_4)]}{\lambda(-A_3\lambda + A_4)e^{-\lambda\tau}}. \tag{3.26}$$

Using (3.15), and after some algebraic manipulations, we arrive at

$$\begin{aligned}\left(\frac{d\lambda}{d\tau}\right)^{-1} &= \frac{2\lambda + A_1}{(-A_3\lambda + A_4)\lambda e^{-\lambda\tau}} - \frac{A_3}{\lambda(-A_3\lambda + A_4)} - \frac{\tau}{\lambda} \\ &= \frac{2\lambda + A_1}{-\lambda(\lambda^2 + A_1\lambda + A_2)} - \frac{A_3}{A_4\lambda} - \frac{A_3^2}{A_4(-A_3\lambda + A_4)} - \frac{\tau}{\lambda}.\end{aligned} \tag{3.27}$$

Thus,

$$\begin{aligned}\operatorname{sign}\left\{\frac{d}{d\tau}\Re(\lambda)\right\}_{\lambda=i\xi_0} &= \operatorname{sign}\left\{\Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\right\}_{\lambda=i\xi_0} \\ &= \operatorname{sign}\left\{\Re\left[\frac{2\lambda + A_1}{-\lambda(\lambda^2 + A_1\lambda + A_2)}\right]_{\lambda=i\xi_0} + \Re\left[-\frac{A_3^2}{A_4(-A_3\lambda + A_4)}\right]_{\lambda=i\xi_0}\right\} \\ &= \operatorname{sign}\left[\frac{2\xi_0^2 + \left(A_1^2 - 2A_2\right)}{A_1^2\xi_0^2 + \left(\xi_0^2 - A_2\right)^2} - \frac{A_3^2}{A_4^2 + A_3^2\xi_0^2}\right] = \operatorname{sign}\left[\frac{P}{Q}\right],\end{aligned} \tag{3.28}$$

where, by doing some manipulations, the expressions

$$P = \xi_0^4 A_3^2 + \xi_0^2\left(2A_4^2\right) + A_4^2\left(A_1^2 - 2A_2\right) + A_2^2A_3^2, \qquad Q = \left[A_1^2\xi_0^2 + \left(\xi_0^2 - A_2\right)^2\right]\left[A_4^2 + A_3^2\xi_0^2\right] \tag{3.29}$$

which are positive when $\mathcal{R}_0 > \mathcal{R}_c > 1$. Then we have

$$\left\{\frac{d}{d\tau}\Re(\lambda)\right\}_{\tau=\tau^*} > 0. \tag{3.30}$$

Therefore, the transversality condition holds and hence Hopf bifurcation occurs at $\xi = \xi_0$, $\tau = \tau^*$. This completes the proof. □

4. Numerical Simulations

System (3.1) is an example of stiff model, in the sense that it has properties that make it slow and expensive to solve using explicit numerical methods. The efficient use of reliable numerical methods, that is based in general on implicit formulae, for dealing with stiff problems involves a degree of sophistication. In this work, we used the so called mono-implicit Runge-Kutta schemes for solve the underlying DDEs [31]. The schemes are suitable for non-stiff and stiff problems.

Figures 1–3 show the numerical simulations of model (3.2), with different values of the model parameters given in the corresponding captions, with $S_0 = 28$, $I_0 = 3$, and $R_0 = 6$. According to the obtained analysis, Figure 1 shows that the numerical simulations of the model, for particular values of the parameters, admit limit cycles, while Figure 2 shows that

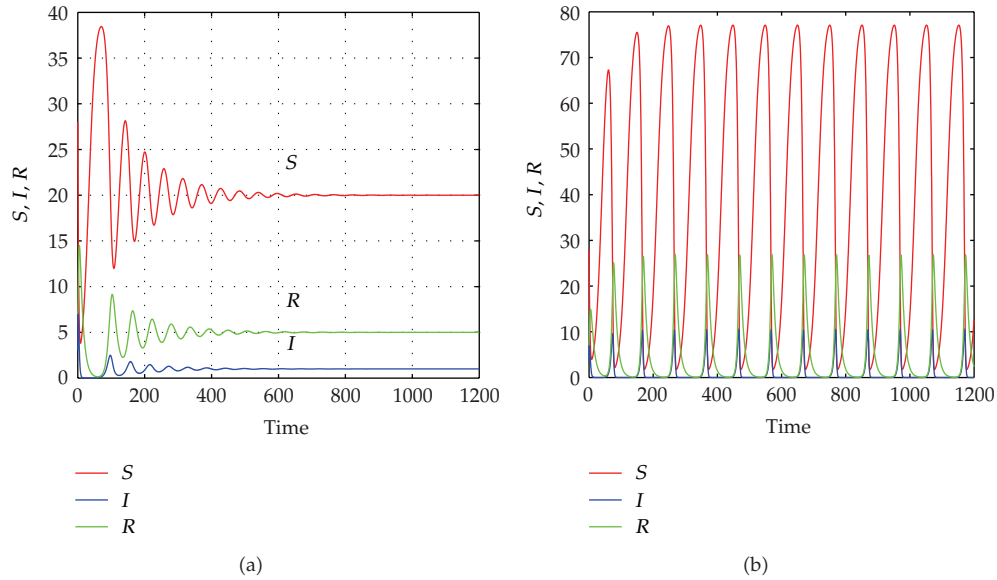

Figure 1: Solution of delayed SIR model (3.2), when $r = 0.1$, $\beta = 0.1$, $\sigma = 0.05$, $a = 0.5$, $\alpha = 0.5$, $\delta = 0.1$, and time-lag $\tau = 1$ when $S^* = 20$, $K = 40$, $\mathcal{R}_0 = 2$, $\mathcal{R}_c = 2.33$ (a) and $K = 80$, $\mathcal{R}_0 = 4$, $\mathcal{R}_c = 2.33$ (b) that display periodic outbreak of the disease. We get a stable solution when $1 < \mathcal{R}_0 \leq \mathcal{R}_c$ and a stable periodic solution when $\mathcal{R}_0 > \mathcal{R}_c$.

periodic solutions arise due to Hopf bifurcation. When the reproduction number $\mathcal{R}_0 < 1$, the disease-free equilibrium is stable (see Figure 3) and when $\mathcal{R}_0 > 1$, the disease free-equilibrium is unstable, and the endemic equilibrium exists. The endemic equilibrium is stable if $1 < \mathcal{R}_0 \leq \mathcal{R}_c$ and a sustained periodic solution is obtained when $\mathcal{R}_0 > \mathcal{R}_c$.

5. Concluding Remarks

This paper investigates the role of time delays in the stability of an SIR model with a nonlinear incidence function. The dynamical behavior of the model is studied and the basic reproductive number $\mathcal{R}_0$ is defined. For particular values of time-lag τ, oscillations occur which can destabilize the system, and periodic solutions can arise due to Hopf bifurcation. We studied the conditions, in terms of the threshold parameter $\mathcal{R}_0$, that guaranteed the asymptotic stability of the infection-free and endemic equilibria. It has been noted that when $\mathcal{R}_0 < 1$, the infection dies out and when $\mathcal{R}_0 > 1$ the disease becomes endemic and infection is maintained in the population. The effect of the time-lag parameter τ on the behavior of the infection has been investigated. Although the underlying DDE model is simple, it displays very rich dynamics, such as quasiperiodic and chaotic patterns, and is suitable for large population densities.

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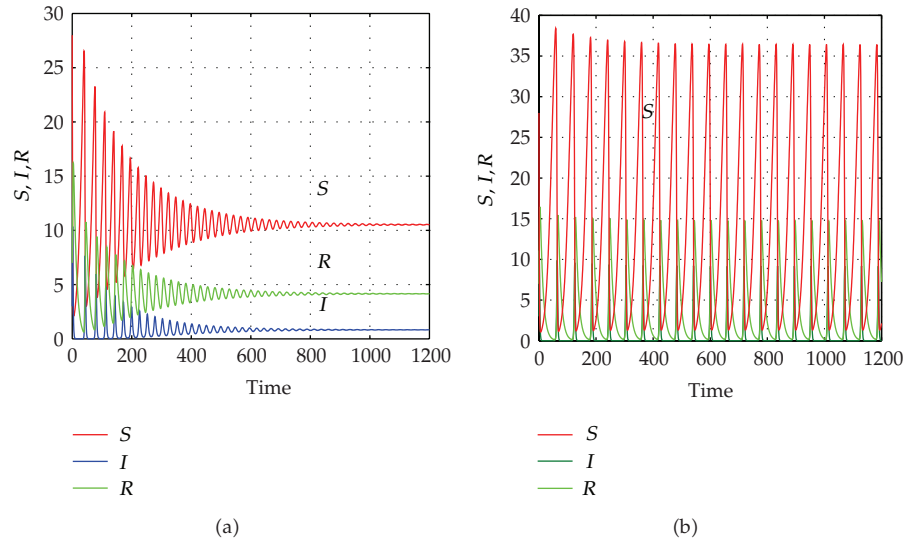

Figure 2: Solution of delayed SIR model (3.2), when $r = 0.1$, $K = 50$, $\beta = 0.1$, $\sigma = 0.005$, $a = 0.5$, $\alpha = 0.5$, $\delta = 0.1$, $S^* = 10.5$, $\mathcal{R}_0 = 4.7 > \mathcal{R}_c = 3$ with time-lag $\tau = 0.01 < \tau^*$ (a) and $\tau = \tau^* = 0.58$ (b) that display periodic outbreak of the disease due to a Hopf bifurcation when $\tau = \tau^*$.

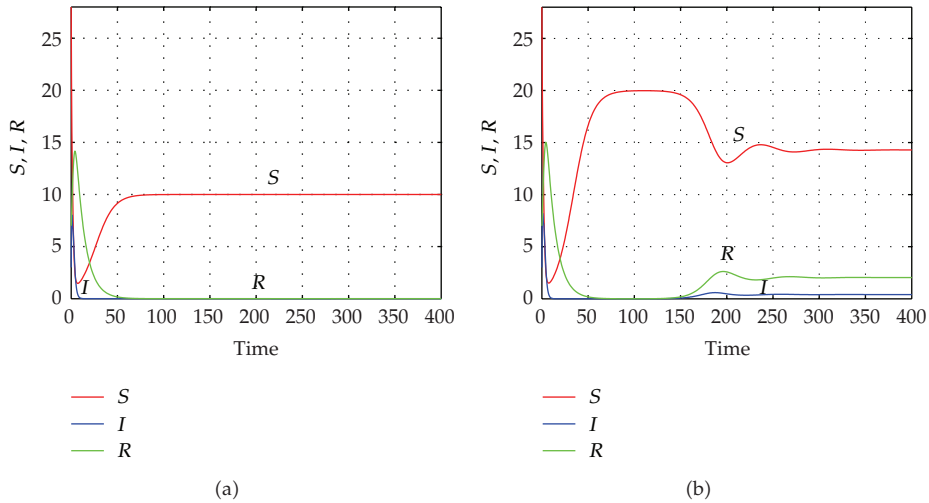

Figure 3: Solution of delayed SIR model (3.2). We have asymptotically stable infection-free equilibrium when $\mathcal{R}_0 < 1$ (a), and small portion endemic equilibrium when $1 < \mathcal{R}_0 < \mathcal{R}_c$ (b), with $\tau = 10$.

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