ON THE DYNAMICS OF A DELAYED SIR EPIDEMIC MODEL
WITH A MODIFIED SATURATED INCIDENCE RATE

ABDELILAH KADDAR

ABSTRACT. In this paper, a delayed SIR epidemic model with modified saturated incidence rate is proposed. The local stability and the existence of Hopf bifurcation are established. Also some numerical simulations are given to illustrate the theoretical analysis.

1. Introduction

Epidemic models have been studied by many authors. Most of them are interesting in the formulation of the incidence rate, i.e. the infection rate of susceptible individuals through their contacts with infective (see, for example, [8, 10, 12, 13, 17]). In order to model this disease transmission process several authors employ following incidence functions. The first one is the bilinear incidence rate $\beta SI$, where $S$ and $I$ are respectively the number of susceptible and infective individuals in the population, and $\beta$ is a positive constant [7, 11, 14, 19, 20]. The second one is the saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 S}$, where $\alpha_1$ is a positive constant. The effect of saturation factor (refer to $\alpha_1$) stems from epidemic control (tacking appropriate preventive measures) [1, 3, 15, 18]. The third one is the saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_2 I}$, where $\alpha_2$ is a positive constant. In this incidence rate the number of effective contacts between infective and susceptible individuals may saturate at high infective levels due to crowding of infective individuals or due to the protection measures by the susceptible individuals [11, 2, 16].

We consider a delayed SIR epidemic model with a modified saturated incidence rate as follows:

$$\begin{align*}
\frac{dS}{dt} &= A - \mu S(t) - \frac{\beta S(t-\tau) I(t-\tau)}{1 + \alpha_1 S(t-\tau) + \alpha_2 I(t-\tau)}, \\
\frac{dI}{dt} &= \frac{\beta S(t) I(t)}{1 + \alpha_1 S(t) + \alpha_2 I(t)} - (\mu + \alpha + \gamma) I(t), \\
\frac{dR}{dt} &= \gamma I(t) - \mu R(t).
\end{align*}$$

(1.1)
where $S$ is the number of susceptible individuals, $I$ is the number of infective individuals, $R$ is the number of recovered individuals, $A$ is the recruitment rate of the population, $\mu$ is the natural death of the population, $\alpha$ is the death rate due to disease, $\beta$ is the transmission rate, $\alpha_1$ and $\alpha_2$ are the parameter that measure the inhibitory effect, $\gamma$ is the recovery rate of the infective individuals, and $\tau$ is the incubation period \([4, 16, 18]\).

The fundamental characteristics of this model are:

(C1) The modified saturated incidence rate $\frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I}$, which includes the three forms, $\beta SI$ (if $\alpha_1 = \alpha_2 = 0$), $\frac{\beta SI}{1 + \alpha_1 S}$ (if $\alpha_2 = 0$), and $\frac{\beta SI}{1 + \alpha_2 I}$ (if $\alpha_1 = 0$), is saturated with the susceptible and the infective individuals.

(C2) The inclusion of time delay into susceptible and infective individuals in incidence rate, only on the first equation, because susceptible individuals infected at time $t - \tau$ is able to spread the disease at time $t$.

The first two equations in system (1.1) do not depend on the third equation, and therefore this equation can be omitted without loss of generality. Hence, system (1.1) can be rewritten as

\[
\begin{align*}
\frac{dS}{dt} &= A - \mu S(t) - \frac{\beta S(t - \tau)I(t - \tau)}{1 + \alpha_1 S(t - \tau) + \alpha_2 I(t - \tau)}, \\
\frac{dI}{dt} &= \frac{\beta S(t)I(t)}{1 + \alpha_1 S(t) + \alpha_2 I(t)} - (\mu + \alpha + \gamma)I(t).
\end{align*}
\] (1.2)

The dynamics of the system (1.2) are studied in terms of local stability and of the description of the Hopf bifurcation, that is proven to exist as the delay $\tau$ cross some critical value. A numerical illustrations are given to illustrate the theoretical analysis.

2. Steady state and local stability analysis

In this section, we discuss the local stability of an endemic equilibrium and a disease-free equilibrium of system (1.2) by analyzing the corresponding characteristic equations, respectively [9]. System (1.2) always has a disease-free equilibrium $E_1 = (\frac{A}{\mu}, 0)$. Further, if

\[
R_0 := \frac{A(\beta - \alpha_1(\mu + \alpha + \gamma))}{\mu(\mu + \alpha + \gamma)} > 1,
\]

system (1.2) admits a unique endemic equilibrium $E^* = (S^*, I^*)$, where

\[
S^* = \frac{A(\mu + \alpha + \gamma) + \alpha_2 A}{\mu[(\mu + \alpha + \gamma)R_0 + \alpha_2 A]}, \quad I^* = \frac{A(R_0 - 1)}{(\mu + \alpha + \gamma)R_0 + \alpha_2 A}.
\]

Remark 2.1. The basic reproduction number (also called the threshold value), $R_0$ representing how many secondary infectious result from the introduction of one infected individual into a population of susceptible [9].

Now let us start to discuss the local behavior of the system (1.2) of the equilibrium points $E_1 = (\frac{A}{\mu}, 0)$, and $E^* = (S^*, I^*)$. At the equilibrium $E_1$, characteristic equation is

\[
(\lambda + \mu)[\lambda - \frac{\mu(\mu + \alpha + \gamma)(R_0 - 1)}{\mu + \alpha_1 A}] = 0.
\] (2.1)
Obviously, (2.1) has two roots \( \lambda_1 = -\mu < 0 \), and \( \lambda_2 = \frac{\mu(\mu + \alpha + \gamma)(R_0 - 1)}{\mu + \alpha + \gamma} \). Hence, we have the following result.

**Proposition 2.2.** If \( R_0 > 1 \), then The equilibrium point \( E_1 \) is unstable.

Let \( x = S - S^* \) and \( y = I - I^* \). Then by linearizing system (1.2) around \( E^* \), we have

\[
\begin{align*}
\frac{dx}{dt} &= -\mu x(t) - \frac{\beta I^*(1 + \alpha_2 I^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2}x(t - \tau) - \frac{\beta S^*(1 + \alpha_1 S^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2}y(t - \tau), \\
\frac{dy}{dt} &= \frac{\beta I^*(1 + \alpha_2 I^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2}x(t) + \left[ \frac{\beta S^*(1 + \alpha_1 S^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2} - (\mu + \alpha + \gamma) \right]y(t).
\end{align*}
\]

The characteristic equation associated to system (2.2) is

\[
\lambda^2 + p\lambda + r + s\lambda \exp(-\lambda \tau) + q \exp(-\lambda \tau) = 0, \tag{2.3}
\]

where

\[
\begin{align*}
p &= \mu + \alpha_2 \mu (\mu + \alpha + \gamma)^2 (R_0 - 1), \\
r &= \frac{\alpha_2 \mu^2 (\mu + \alpha + \gamma)^2 (R_0 - 1)}{\beta(\mu + \alpha + \gamma) + \alpha_2 A}, \\
s &= \frac{\mu^2 (\mu + \alpha + \gamma)^2 R_0 (R_0 - 1)}{\beta A(\mu + \alpha + \gamma) + \alpha_2 A}, \\
q &= \frac{\mu^2 (\mu + \alpha + \gamma)^3 R_0 (R_0 - 1)}{\beta A(\mu + \alpha + \gamma) + \alpha_2 A}.
\end{align*}
\]

**Theorem 2.3.** Let us assume

(H1) \( 1 < R_0 \),
(H2) \( \alpha_2 \mu < \beta - \alpha_1 (\mu + \alpha + \gamma) \).

Then there exists \( \tau_0 > 0 \) such that, when \( \tau \in [0, \tau_0) \) the steady state \( E^* \) is locally asymptotically stable, when \( \tau > \tau_0 \), \( E^* \) is unstable and when \( \tau = \tau_0 \), equation (2.3) has a pair of purely imaginary roots \( \pm i\omega_0 \), with

\[
\omega_0^2 = \frac{1}{2} (s^2 + 2r - p^2) + \frac{1}{2} \left[ (s^2 + 2r - p^2)^2 - 4(r^2 - q^2) \right]^{1/2}, \tag{2.4}
\]

and

\[
\tau_0 = \frac{1}{\omega_0} \arccos \left[ \frac{ps\omega_0^2 + (r - \omega_0^2)q}{s^2\omega_0^2 + q^2} \right], \tag{2.5}
\]

where \( p, r, s, q \) are defined in (2.3).

For the proof of the above theorem, we need the following lemma.

**Lemma 2.4 (15).** If the hypotheses

(S1) \( p + s > 0 \),
(S2) \( q + r > 0 \),
(S3) \( r - q < 0 \),

hold, then there exists \( \tau_0 > 0 \) such that, when \( \tau \in [0, \tau_0) \), all roots of the equation (2.3) have negative real parts, when \( \tau = \tau_0 \), equation (2.3) has a pair of purely imaginary roots \( \pm i\omega_0 \), and when \( \tau > \tau_0 \), equation (2.3) has at least one root with positive real part, where \( \tau_0 \) and \( \omega_0 \) are defined in Theorem 2.3.

**Proof of Theorem 2.3.** From hypothesis (H1), the hypotheses (S1) and (S2) of lemma 2.4 are satisfied. From the expression of \( q \) and \( r \), we have

\[
r - q = \frac{\mu(\mu + \alpha + \gamma)(R_0 - 1)}{\beta(\mu + \alpha + \gamma) + \alpha_2 A} (\alpha_2 \mu - \beta + \alpha_1 (\mu + \alpha + \gamma)).
\]
From hypotheses (H1) and (H2), we have \( r - q < 0 \). Therefore, the hypothesis (S3) of lemma 2.4 is satisfied. Thus we have

- For \( \tau \in [0, \tau_0) \), \((E^*, I^*)\) is asymptotically stable.
- For \( \tau > \tau_0 \), \((E^*, I^*)\) is unstable.
- For \( \tau = \tau_0 \), equation (2.3) has a pair of purely imaginary roots \( \pm i\omega_0 \).

\( \square \)

3. Hopf bifurcation

From theorem 2.3, we have the following result.

**Theorem 3.1.** Suppose that (H1)-(H2) hold. Then there exists \( \varepsilon_0 > 0 \) such that for each \( 0 \leq \varepsilon < \varepsilon_0 \), system \((1.2)\) has a family of periodic solutions \( P = P(\varepsilon) \) with period \( T = T(\varepsilon) \), for the parameter values \( \tau = \tau(\varepsilon) \) such that \( P(0) = 0 \), \( T(0) = \frac{2\pi}{\omega_0} \) and \( \tau(0) = \tau_0 \).

**Proof.** We apply the Hopf bifurcation theorem introduced in [9]. We only need to verify that \( \pm i\omega_0 \) are simple, and the transversally condition \( \frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} \neq 0 \).

First, we show that \( i\omega_0 \) is simple: Consider the branch of the characteristic root \( \lambda(\tau) = \mu(\tau) + i\nu(\tau) \), of (2.3), bifurcating from \( i\omega_0 \) at \( \tau = \tau_0 \). By differentiating (2.3) with respect to the delay \( \tau \), we obtain

\[
\{2\lambda + p + s \exp(-\lambda \tau) - s\tau \lambda \exp(-\lambda \tau) - q \tau \exp(-\lambda \tau)\} \frac{d\lambda}{d\tau} = (s\lambda + q)\lambda \exp(-\lambda \tau). \tag{3.1}
\]

If we suppose, by contradiction, that \( i\omega_0 \) is not simple, the right hand side of (3.1) gives

\[
(s + q)i\omega_0 = 0,
\]

and leads a contradiction with the fact that \( s + q > 0 \).

Lastly, we need to verify the transversally condition,

\[
\frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} \neq 0.
\]

From (3.1), we have

\[
(\frac{d\lambda}{d\tau})^{-1} = \frac{(2\lambda + p) \exp(\lambda \tau) + s - \tau}{\lambda(s\lambda + q)}.
\]

As,

\[
\text{sign} \frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} = \text{sign} \left( \text{Re}(\lambda)^{-1} \big|_{\tau=\tau_0} \right).
\]

Then

\[
\text{sign} \frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} = \text{sign} \left( \text{Re} \left( \frac{(2\lambda + p) \exp(\lambda \tau) + s}{\lambda(s\lambda + q)} \right) \right). \tag{3.2}
\]

From (2.3), we have

\[
\exp(\lambda \tau) = -\frac{s\lambda + q}{\lambda^2 + p\lambda + r}. \tag{3.3}
\]

So, by (3.2) and (3.3) we obtain

\[
\text{sign} \frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} = \text{sign}(\{(s^2 + 2r - p^2)^2 - 4(r^2 - q^2)\}^{1/2}).
\]

Consequently, \( \frac{d}{d\tau} \text{Re}(\lambda) \big|_{\tau=\tau_0} > 0. \) \( \square \)
4. Numerical Application

4.1. Effect of incubation period. In this section, we give a numerical simulation supporting the theoretical analysis given in section 2 and 3. Consider the following parameters:

\[ \alpha_1 = 0.01, \quad \alpha_2 = 0.01, \quad A = 0.94, \quad \beta = 0.1, \quad d = 0.05, \quad \alpha = 0.5, \quad \gamma = 0.5. \]

System (1.2) has the unique positive equilibrium \( E^\ast = (11.7711, 0.3347). \) It follows from Theorem 2.3 that the critical positive time delay \( \tau_0 = 2.8465. \) Thus we know that when \( 0 \leq \tau < \tau_0, \) \( E^\ast \) is asymptotically stable. And from Theorem 3.1, when \( \tau \) passes through the critical value \( \tau_0, \) \( E^\ast \) loses its stability and a family of periodic solutions with period \( P = 38.0965 \) bifurcating from \( E^\ast \) occurs (see Figure 1).

![Figure 1](image)

Figure 1. For \( \tau = 1, \) the solutions \((S(t), I(t))\) of system (1.2) are asymptotically stable and converge to the equilibrium \( E^\ast \) (top). When \( \tau = 2.8465, \) a Hopf bifurcation occurs and periodic solutions appear, with same period \( T(0) = 38.0965 \) (middle). For \( \tau = 4, \) the equilibrium \( E^\ast \) of system (1.2) is unstable (bottom).

4.2. Effect of changing the inhibitory effect. Now, we show how the critical delay \( \tau_0, \) changes as the parameters \( \alpha_1, \) and \( \alpha_2 \) move. In table 1 we assume that the parameters

\[ A = 0.94, \quad \beta = 0.4, \quad d = 0.01, \quad \alpha = 0.02, \quad \gamma = 0.3 \]

are fixed. The delayed SIR epidemic model with a bilinear incidence rate \( (\alpha_1 = \alpha_2 = 0) \) and saturated incidence rate \( ((\alpha_1, \alpha_2) \neq (0, 0)) \) generate the same local asymptotic properties if \( (\alpha_1, \alpha_2) \) is close enough to \( (0, 0). \) However with large values of \( \alpha_1 \) and/or \( \alpha_2 \) this equivalence was not true anymore (see, table 1).
Table 1. Dependence of the critical value of delay \( \tau_0 \) on the inhibitory effect \( \alpha_1 \), and \( \alpha_2 \).

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5. Concluding remarks and Future research

In this article, we introduced a delayed SIR model with a modified saturated incidence rate of the form \( \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I} \), which includes the three forms, \( \beta SI \) (if \( \alpha_1 = \alpha_2 = 0 \)), \( \frac{\beta SI}{1+\alpha_1 S} \) (if \( \alpha_2 = 0 \)), and \( \frac{\beta SI}{1+\alpha_2 I} \) (if \( \alpha_1 = 0 \)). In this formulation it includes

- The mixing process (related to \( \frac{1}{1+\alpha_1 S+\alpha_2 I} \)), i.e., the individuals in the population will be totally mixed and the probability of contact with an infective will decrease as population size increases.
- The saturation effects due to crowding of infective individuals and to the protection measures by the susceptible individuals.

We showed that the local stability of the endemic equilibrium point, \( E^* \), depend on time delay, \( \tau \), (the incubation period). The system changes its behavior from stable to unstable nature around \( E^* \) when \( \tau \) crosses the critical value \( \tau_0 \) via a Hopf bifurcation and periodic solutions bifurcating from \( E^* \). The numerical simulations are given to illustrate the theoretical analysis and to show that for large values of the inhibitory effect \( \alpha_1 \) and/or \( \alpha_2 \) the dynamics generated by the modified saturated incidence rate is not equivalent to the following three forms, \( \beta SI, \frac{\beta SI}{1+\alpha_1 S} \), and \( \frac{\beta SI}{1+\alpha_2 I} \).

For the future research, we consider a delayed SIR model with a generalized saturated incidence rate of the form \( \frac{\beta SI^{p_1 I^{q_1}}}{1+\alpha_1 S^{p_2}+\alpha_2 I^{q_2}} \) which must be much more complicated to explore.

References


Abdelilah Kaddar  
Department of Mathematics, Faculty of Sciences, Chouaib Doukkali University, PO Box 20, El Jadida, Morocco  
*E-mail address*: a.kaddar@yahoo.fr