Nonlinear Analysis: Modelling and Control, 2010, Vol. 15, No. 3, 299–306

Stability analysis in a delayed SIR epidemic model with a saturated incidence rate

A. Kaddar

Faculté des Sciences Juridiques, Economiques et Sociales – Salé
Université Mohammed V – Souissi
Route Outa Hssain, BP 5295, Sala Al Jadida, Morocco
a.kaddar@yahoo.fr

Received: 2009-12-16 **Revised:** 2010-04-03 **Published online:** 2010-09-17

Abstract. We formulate a delayed SIR epidemic model by introducing a latent period into susceptible, and infectious individuals in incidence rate. This new reformulation provides a reasonable role of incubation period on the dynamics of SIR epidemic model. We show that if the basic reproduction number, denoted, R_0, is less than unity, the disease-free equilibrium is locally asymptotically stable. Moreover, we prove that if $R_0 > 1$, the endemic equilibrium is locally asymptotically stable. In the end some numerical simulations are given to compare our model with existing model.

Keywords: SIR epidemic model, delayed differential equations, Hopf bifurcation, periodic solutions.

1 Introduction and mathematical models

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 infectious (see, for example, [1–5]). In order to model this disease transmission process several authors employ the following incidence functions: The first one is the bilinear incidence rate βSI, where S and I are respectively the number of susceptible and infective individuals in the population, and β is a positive constant [6–10]. The second one is the saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 S}$, where α_1 is a positive constant. The effect of saturation factor (refer to α_1) stems from epidemic control (tacking appropriate preventive measures) [11–14]. The third one is the saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_2 I}$, where α_2 is a positive constant. In the last one, 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 [7, 15, 16].

In a recent paper [17], we considered a delayed SIR epidemic model with a saturated incidence rate as follows:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \mu S(t) - \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha_1 S(t-\tau)+\alpha_2 I(t-\tau)},\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t)I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)} - (\mu+\alpha+\gamma)I(t),\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I(t) - \mu R(t),
\end{aligned} \tag{1}$$

where S is the number of susceptible individuals, I is the number of infectious individuals, R is the number of recovered individuals, A is the recruitment rate of the population, μ is the natural death of the population, α is the death rate due to disease, β is the transmission rate, α_1 and α_2 are the parameter that measure the inhibitory effect, γ is the recovery rate of the infective individuals, and τ is the incubation period [14, 16–18].

In the SIR model (1), the number of the new infective cases produced in the period $(t-\tau, t]$ is neglected in the evolution of the susceptible class, and is taken into consideration in the evolution of the infectious class. However, it may be more realistic to tack this period into consideration in the evolution of susceptible class, and not in the evolution of infectious class, because susceptible individuals infected at time $t-\tau$ is able to spread the disease at time t. In this paper, incubation period is introduced into the SIR epidemic model (1) to formulate a new delayed SIR model as follow:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \mu S(t) - \frac{\beta S(t)I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)},\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha_1 S(t-\tau)+\alpha_2 I(t-\tau)} - (\mu+\alpha+\gamma)I(t),\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I(t) - \mu R(t).
\end{aligned} \tag{2}$$

The first two equations in system (2) do not depend on the third equation, and therefore this equation can be omitted without loss of generality. Hence, system (2) can be rewritten as

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \mu S(t) - \frac{\beta S(t)I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)},\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha_1 S(t-\tau)+\alpha_2 I(t-\tau)} - (\mu+\alpha+\gamma)I(t).
\end{aligned} \tag{3}$$

This model provides a reasonable role of incubation period on the dynamics of SIR epidemic model. We show that if the basic reproduction number, denoted, R_0, is less than unity, the disease-free equilibrium is locally asymptotically stable, and disease always dies out. Moreover, we prove that if $R_0 > 1$, the endemic equilibrium is locally asymptotically stable, so that the disease, if initially present, will persist at the unique endemic equilibrium. In the end some numerical simulations are given to compare our model with existing model.

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 (3) by analyzing the corresponding characteristic equations, respectively [19]. System (3) always has a disease-free equilibrium $E_1 = (\frac{A}{\mu}, 0)$. Further, if

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

system (3) admits a unique endemic equilibrium $E^* = (S^*, I^*)$, where

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

where $R_c := \frac{A[\beta-\alpha_1(\mu+\alpha+\gamma)]}{\mu(\mu+\alpha+\gamma)}$.

Remark 1. (i) The basic reproduction number, R_0 representing how many secondary infectious result from the introduction of one infected individual into a population of susceptible [20].

(ii) $R_0 > 1$ is equivalent to $R_c > 1$.

Now let us start to discuss the local behavior of the system (3) of the equilibrium points $E_1 = (\frac{A}{\mu}, 0)$, and $E^* = (S^*, I^*)$. At the equilibrium E_1, characteristic equation is

$$(\lambda+\mu)\left[\lambda + (\mu+\alpha+\gamma) - \frac{\beta A}{\mu+\alpha_1 A}\exp(-\lambda\tau)\right] = 0. \tag{4}$$

We have the following result

Proposition 1. *If $R_0 < 1$, then the disease free equilibrium E_1 is locally asymptotically stable. And if $R_0 > 1$, then the equilibrium point E_1 is unstable.*

Proof. For $\tau = 0$, the equation (4) reads to

$$(\lambda+\mu)\left[\lambda - \frac{\mu(\mu+\alpha+\gamma)(R_c-1)}{\mu+\alpha_1 A}\right] = 0. \tag{5}$$

Obviously, (5) has two roots $\lambda_1 = -\mu < 0$, and $\lambda_2 = \frac{\mu(\mu+\alpha+\gamma)(R_C-1)}{\mu+\alpha_1 A}$. Hence, if $R_c < 1$, then the disease free equilibrium E_1 is locally asymptotically stable for $\tau = 0$. By Corollary 2.4 in Ruan and Wei [21, p. 867], it follows that if instability occurs for a particular value of the delay τ, a characteristic root of (4) must intersect the imaginary axis. Suppose that (4) has a purely imaginary root $i\omega$, with $\omega > 0$. Then, by separating real and imaginary parts in (4), we have

$$\begin{cases} \mu+\alpha+\gamma = \frac{\beta A}{\mu+\alpha_1 A}\cos(\omega\tau), \\ \omega = -\frac{\beta A}{\mu+\alpha_1 A}\sin(\omega\tau). \end{cases} \tag{6}$$

Hence,

$$\omega^2 = \frac{\mu(\mu+\alpha+\gamma)(R_c-1)}{\mu+\alpha_1 A}\left[(\mu+\alpha+\gamma)+\frac{\beta A}{\mu+\alpha_1 A}\right]. \tag{7}$$

For $R_c < 1$, equation (7) has no positive solution. Thus from Remark 1, if $R_0 < 1$, then the disease free equilibrium E_1 is locally asymptotically stable for all $\tau \geq 0$.

If $R_c > 1$, then the disease free equilibrium E_1 is unstable for $\tau = 0$. By Kuang's theorem [22, p. 77], it follows that E^* is unstable for all $\tau \geq 0$. This concludes the proof.

Let $x = S - S^*$ and $y = I - I^*$. Then by linearizing system (3) around E^*, we have

$$\begin{aligned}
\frac{\mathrm{d}x}{\mathrm{d}t} &= \left[-\mu - \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}\right]x(t) - \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}y(t),\\
\frac{\mathrm{d}y}{\mathrm{d}t} &= \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}x(t-\tau) + \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}y(t-\tau)\\
&\quad - (\mu+\alpha+\gamma)y(t).
\end{aligned} \tag{8}$$

The characteristic equation associated to system (8) is

$$\lambda^2 + p\lambda + s\lambda\exp(-\lambda\tau) + r + q\exp(-\lambda\tau) = 0, \tag{9}$$

where

$$\begin{aligned}
p &= \mu + (\mu+\alpha+\gamma) + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}, \quad s = -\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2},\\
r &= \left[\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}\right](\mu+\alpha+\gamma), \quad q = -\frac{\mu\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}.
\end{aligned}$$

The local stability of the steady state E^* is a result of the localization of the roots of the characteristic equation (9). In order to investigate the local stability of the steady state, we begin by considering the case without delay $\tau = 0$. In this case the characteristic equation (9) reads as

$$\lambda^2 + (p+s)\lambda + r + q = 0, \tag{10}$$

where

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

hence, according to the Hurwitz criterion and Remark 1, we have the following proposition. □

Proposition 2. *For $\tau = 0$, the equilibrium E^* is locally asymptotically stable if and only if $R_0 > 1$.*

We now return to the study of equation (9) with $\tau > 0$.

Theorem 1. *If $R_0 > 1$, then the steady state E^* is locally asymptotically stable for all $\tau \geq 0$.*

Proof. From the hypothesis $R_0 > 1$, the characteristic equation (9) has negative real parts for $\tau = 0$ (see Proposition 2). By Corollary 2.4 in Ruan and Wei [21, p. 867], it follows that if instability occurs for a particular value of the delay τ, a characteristic root of (9) must intersect the imaginary axis. Suppose that (9) has a purely imaginary root $i\omega$, with $\omega > 0$. Then, by separating real and imaginary parts in (9), we have

$$\begin{cases} r - \omega^2 - s\omega\sin(\omega\tau) + q\cos(\omega\tau) = 0, \\ p\omega + s\omega\cos(\omega\tau) - q\sin(\omega\tau) = 0. \end{cases} \tag{11}$$

Hence,

$$\omega^4 + (p^2 - s^2 - 2r)\omega^2 + r^2 - q^2 = 0. \tag{12}$$

From the expressions of r and q, we have $r - q > 0$ and from hypothesis $R_c > 1$, we deduce that $r^2 - q^2 > 0$.

Evaluating $p^2 - s^2 - 2r$,

$$p^2 - s^2 - 2r = \frac{\alpha_2\mu(\mu+\alpha+\gamma)^2(R_c-1)}{\beta[(\mu+\alpha+\gamma)+\alpha_2 A]}\left[(\mu+\alpha+\gamma) + \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}\right] + \left[\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}\right]^2.$$

Since for $R_c > 1$, we have $p^2 - s^2 - 2r > 0$.

Thus from Remark 1, equation (12) has no positive solution for $R_0 > 1$. This concludes the proof. □

3 Numerical application

Let's compare the principal results of systems (1) and (2) by a numerical illustration. Consider the following parameters:

$$\alpha_1 = 0.01, \quad \alpha_2 = 0.01, \quad A = 0.94, \quad \beta = 0.1, \quad \mu = 0.05, \quad \alpha = 0.5, \quad \gamma = 0.5.$$

System (1) and (2) has the positive equilibrium $E^* = (11.771, 0.334, 3.347)$. It follows from Theorem 3.1 in [17], that for system (1), as the delay cross some critical value $\tau_0 = 2.8465$, E^* loses its stability and a family of periodic solutions with period $P = 38.0965$ bifurcating from E^* occurs (see Fig. 1). However, for system (2), E^* is asymptotically stable for all $\tau \geq 0$ (see Fig. 2).

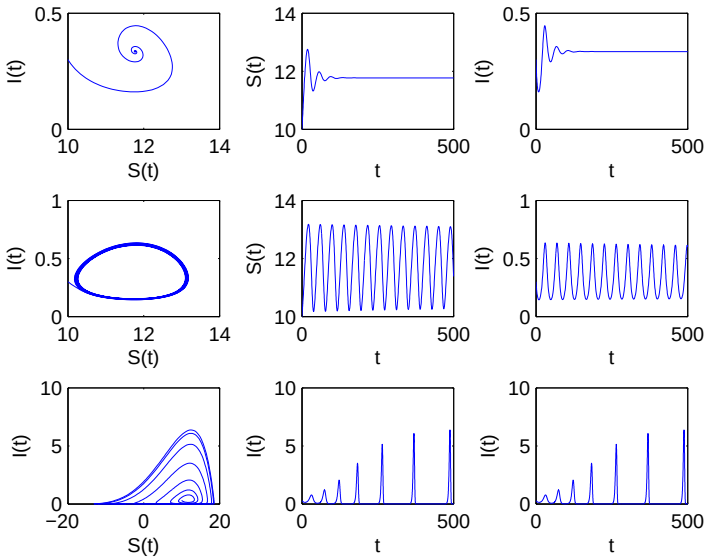

Fig. 1. For $\tau = 0$, the solutions $(S(t), I(t), R(t))$ of system (1) are asymptotically stable and converge to the equilibrium E^* (top). When $\tau = 2.8465$, a Hopf bifurcation occurs and periodic solutions appear, with same period $T(0) = 38.0965$ (middle). For $\tau = 4$, the equilibrium E^*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

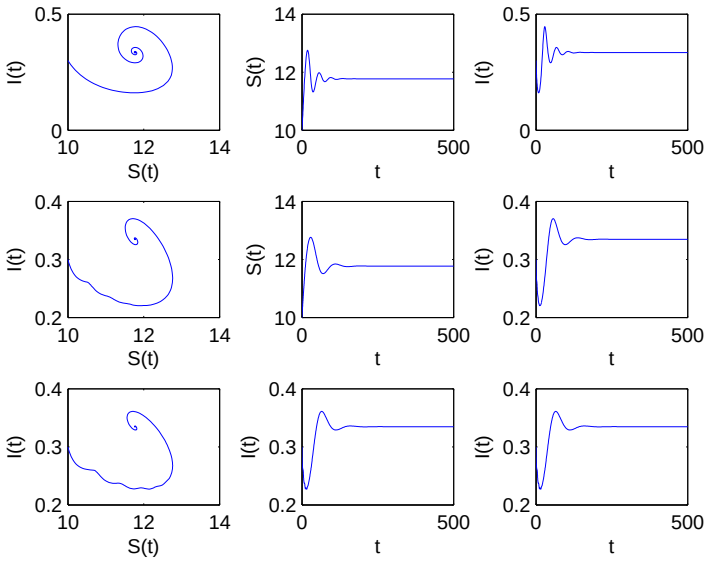

Fig. 2. For $\tau = 0$; $\tau = 2.8465$; $\tau = 4$, the solutions (S, I, R) of system (2) are asymptotically stable and converge to the equilibrium E^*.

4 Concluding remarks and future research

In this paper, we considered a delayed SIR model with a modified saturated incidence rate $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$, and a constant recruitment, A. We showed that the local stability of the endemic equilibrium point, E^*, depend on the basic reproduction number, R_0, and doesn't change with respect to time delay, τ, (the incubation period); If $R_0 < 1$, the disease-free equilibrium, E_1, is locally asymptotically stable so the disease dies out. Moreover, we prove that if $R_0 > 1$, the disease-free equilibrium, E_1, is unstable and the endemic equilibrium, E^*, is locally asymptotically stable for all $\tau \geq 0$ (the disease approaches the endemic value E^*). In the end some numerical simulations are given to illustrate the theoretical analysis and to compare our model with existing model in [17].

For the future research, we consider a delayed SIR model with a saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$ and a logistic growth. In this case we show that the local stability of the endemic equilibrium point, E^*, depend on time delay, τ.

References

1. S. Gao, L. Chen, J.J. Nieto, A. Torres, Analysis of a delayed epidemic model with pulse vaccination and saturation incidence, *Vaccine*, **24**(35–36), pp. 6037–6045, 2006.

2. H.W. Hethcote, H.W. Stech, P. Van den Driessche, Periodicity and stability in epidemic models: a survey, in: S.N. Busenberg, K.L. Cooke (Eds.), *Differential Equations and Applications in Ecology, Epidemics and Population Problems*, Academic Press, New York, pp. 65–82, 1981.

3. Y. Kyrychko, B. Blyuss, Global properties of a delayed SIR model with temporary immunity and nonlinear incidence rate, *Nonlinear Anal., Real World Appl.*, **6**, pp. 495–507, 2005.

4. G. Li, W. Wang, K. Wang, Z. Jin, Dynamic behavior of a parasite-host model with general incidence, *J. Math. Anal. Appl.*, **331**(1), pp. 631–643, 2007.

5. J.A. Yorke, W.P. London, Recurrent outbreak of measles, Chickenpox and mumps II, *Am. J. Epidemiol.*, **98**, pp. 469–482, 1981.

6. M. Gabriela, M. Gomes, L.J. White, G.F. Medley, The reinfection threshold, *J. Theor. Biol.*, **236**, pp. 111–113, 2005.

7. Z. Jiang, J. Wei, Stability and bifurcation analysis in a delayed SIR model, *Chaos Soliton. Fract.*, **35**, pp. 609–619, 2008.

8. W. Wang, S. Ruan, Bifurcation in epidemic model with constant removal rate infectives, *J. Math. Anal. Appl.*, **291**, pp. 775-793, 2004.

9. F. Zhang, Z.Z. Li, F. Zhang, Global stability of an SIR epidemic model with constant infectious period, *Appl. Math. Comput.*, **199**, pp. 285–291, 2008.

10. Y. Zhou, H. Liu, Stability of periodic solutions for an SIS model with pulse vaccination, *Math. Comput. Model.*, **38**, pp. 299-308, 2003.

11. R.M. Anderson, R.M. May, Regulation and stability of host-parasite population interactions I. Regulatory processes, *J. Anim. Ecol.*, **47**(1), pp. 219–267, 1978.

12. L.S. Chen, J. Chen, *Nonlinear biological dynamics system*, Scientific Press, China, 1993.

13. C. Wei, L. Chen, A delayed epidemic model with pulse vaccination, *Discrete Dyn. Nat. Soc.*, **2008**, doi:10.1155/2008/746951, pp. 1–12, 2008.

14. J.-Z. Zhang, Z. Jin, Q.-X. Liu, Z.-Y. Zhang, Analysis of a delayed SIR model with nonlinear incidence rate, *Discrete Dyn. Nat. Soc.*, **2008**, doi:10.1155/2008/636153, pp. 1–16, 2008.

15. V. Capasso, G. Serio, A generalization of Kermack-Mckendrick deterministic epidemic model, *Math. Biosci.*, **42**, pp. 41–61, 1978.

16. R. Xu, Z. Ma, Stability of a delayed SIRS epidemic model with a nonlinear incidence rate, *Chaos, Soliton. Fract.*, **41**(5), pp. 2319–2325, 2009.

17. A. Kaddar, On the dynamics of a delayed SIR epidemic model with a modified saturated incidence rate, *Electronic Journal of Differential Equations*, **2009**(133), pp. 1–7, 2009.

18. K.L. Cooke, Stability analysis for a vector disease model, *Rocky Mt. J. Math.*, **9**(1), pp. 31–42, 1979.

19. J.K. Hale, S.M. Verduyn Lunel, *Introduction to Functional Differential Equations*, Springer-Verlag, New York, 1993.

20. O. Diekmann, J.A.P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases*, John Wiley & Son, Ltd, 2000.

21. S. Ruan, J. Wei, On the zeros of transcendental functions with applications to stability of delay differential equations with two delays, *Dynamics of Continuous, Discrete and Impulsive Systems A: Mathematical Analysis*, **10**, pp. 863–874, 2003.

22. Y. Kuang, *Delay Differential Equations with Applications in Population Dynamics*, Academic Press, Inc., New York, 1993.

23. K.L. Cooke, Z. Grossman, Discrete delay, distributed delay and stability switches, *J. Math. Anal. Appl.*, **86**(2), pp. 592–627, 1982.