Stability Analysis of SEIR Model with Saturated Incidence and Time Delay

Sirachat Tipsri and Wirawan Chinviriyasit

Abstract—In this paper, the effect of time delay on an SEIR epidemic model with saturated incidence rate is investigated. The model has two equilibria, namely, a disease-free equilibrium and an endemic equilibrium. The threshold value of the model, which is called the basic reproduction number, is derived. The results show that for any time delay if the basic reproduction number is less than unity, the disease-free equilibrium is locally asymptotically stable whereas if it is greater than unity, the endemic equilibrium is locally asymptotically stable. Numerical simulations are presented to illustrate the analytical results.

Index Terms—SEIR model, saturated incidence, time delay, stability.

I. INTRODUCTION

Various biological reasons lead to the introduction of time delays in models of disease transmission. In the last decades, many authors have explored the dynamics of systems with time delay and many interesting results have been obtained [1]–[6]. Delay models are used in an attempt to better understanding of more and more complicated phenomena for describing several aspects of infectious disease dynamics.

Incidence rate plays an important role in the modelling of epidemic dynamics. It has been suggested by several authors that the disease transmission process may have a nonlinear incidence rate. A nonlinear incidence rate can also arise from saturation effects: if the proportion of the infectives in a population is very high, so that exposure to the disease agent is virtually certain, then the transmission rate may respond more slowly than linear to the increase in the number of infectives. This effect was observed by Capasso and Serio [7] who studied the cholera epidemic spread in Bari in 1973. They introduced a saturated incidence rate

$$g(I) = \frac{\beta I}{1 + \alpha I} \tag{1}$$

into epidemic models, where βI measures the infection force of the disease and $1/(1+\alpha I)$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. Xu and Ma [8] studied an *SIR* epidemic model with time delay and this

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incidence rate. They proved that if the basic reproduction number $R_0 \le 1$, the disease-free equilibrium was globally asymptotically stable. Using the means of an iteration technique and Lyapunov functional technique, they found sufficient conditions for the global asymptotic stability of the endemic equilibrium whenever $R_0 > 1$.

Although the *SIR* model can give some useful indications about disease, it is difficult to apply to real situations as most diseases have a latent period before the infected person becomes infectious to others. This latency can be modeled either by incorporating it as a delay effect, or by introducing a new class *E* called the exposed class in which the susceptible remains for a given length of time before moving into the infective class. The resulting model is called *SEIR* model.

As above reasons, the aim of this paper is to modify and to incorporate a discrete delay to the model proposed by Xu and Ma [8] in order to understand the effect of exposed individual on the dynamical behaviors of the model depending on the past information. Thus, a *SEIR* model with saturated incidence rate in (1) and time delay is given by

$$\frac{dS}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t)}{1 + \alpha I(t)},$$

$$\frac{dE}{dt} = \frac{\beta S(t - \tau)I(t - \tau)}{1 + \alpha I(t - \tau)} - (\mu + \sigma)E(t),$$

$$\frac{dI}{dt} = \sigma E(t) - (\mu + \gamma)I(t),$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t),$$
(2)

where *S*, *E*, *I*, *R* are the numbers of susceptible, exposed, infectious and recovered individuals, respectively. The parameters, *A* is the recruitment rate of the population, μ is the natural death of the population, β is the transmission rate, α is the parameter that measures the inhibitory effect, σ is the rate at which exposed individuals become infectious, γ is the recovery rate of the infectious individuals, and τ is a time delay representing the latent period of the disease. The incidence rate $\frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$ appearing in second equation represents the rate at time $t-\tau$ at which susceptible

individual leave the susceptible class and enter the infectious class at time t.

It is observed that the first three equations in system (2) do not depend on the forth equation, and therefore this equation can be omitted without loss of generality. Hence, system (2) can be reduced as

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$$\frac{dS}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t)}{1 + \alpha I(t)},$$

$$\frac{dE}{dt} = \frac{\beta S(t - \tau)I(t - \tau)}{1 + \alpha I(t - \tau)} - (\mu + \sigma)E(t),$$

$$\frac{dI}{dt} = \sigma E(t) - (\mu + \gamma)I(t).$$
(3)

The initial condition of (3) is given by

$$S(\theta) = \phi_1(\theta) > 0, E(0) = E_0 \ge 0, I(\theta) = \phi_3(\theta) \ge 0,$$
 (4)

for all $\theta \in [-\tau, 0]$ where $\phi = (\phi_1, E_0, \phi_3)^T \in \mathbb{C}$. Here \mathbb{C} denotes the Banach space $\mathbb{C}([-\tau, 0], \mathbb{R}^3_+)$ of continuous functions mapping the interval $[-\tau, 0]$ into \mathbb{R}^3_+ ,

$$\mathbb{R}^{3}_{+} = \{ (S, E, I) : S \ge 0, E \ge 0, I \ge 0 \}.$$
(5)

It is well known by the fundamental theory of functional differential equations [9], system (3) has an unique solution (S(t), E(t), I(t)) satisfying the initial condition (4). It can be shown that all solutions of system (3) with initial condition (4) are defined on $[0, \infty)$ and remain positive for all $t \ge 0$.

This paper is organized as follows: Taking time delay as a bifurcation parameter, the equilibriums and their stability are discussed in Section II. Numerical simulations are reported in Section III. Finally, the conclusion of this work is given in Section IV.

II. EQUILIBRIUMS AND STABILITY ANALYSES

A. Equilibria

Equilibria of (3) are obtained by setting all derivatives on the right-hand sides to be zero. Let $\tilde{X} = (\tilde{S}, \tilde{E}, \tilde{I})$ be any equilibrium point of (3). Naturally a stable equilibrium point \tilde{X} is the one for which all the nearby trajectories approach it asymptotically as $t \to \infty$. Hence, the equilibrium solutions of a system with time delays are the same as those of the corresponding system with zero delay. This can be verified that the system (3) has two biologically relevant equilibria, namely,

- 1) a disease-free equilibrium $E_1 = (\frac{A}{\mu}, 0, 0),$
- 2) unique endemic equilibrium $E_2 = (S^*, E^*, I^*)$ where

$$S^* = \frac{A(\beta + \mu \alpha R_0)}{(\mu R_0)(\mu \alpha + \beta)}, \ E^* = \frac{\mu(\mu + \gamma)(R_0 - 1)}{\sigma(\mu \alpha + \beta)}$$
$$\mu(R_0 - 1) \qquad \qquad \sigma \beta A$$

$$I^* = \frac{\mu(R_0 - \gamma)}{\mu\alpha + \beta}$$
 and $R_0 = \frac{0\beta A}{\mu(\mu + \sigma)(\mu + \gamma)}$.

The value R_0 is called the basic reproduction number.

B. Stability Analysis of Disease-Free Equilibrium

Consider a small perturbation about the equilibrium point

 \tilde{X} , i.e. $x = S - \tilde{S}$, $y = E - \tilde{E}$ and $z = I - \tilde{I}$. Substituting these into the differential equations (3) and neglecting the products of small quantities, the jacobian matrix is given by

$$J(\tilde{X}) = \begin{bmatrix} -\mu - \frac{\beta \tilde{I}}{1 + \alpha \tilde{I}} & 0 & \frac{-\beta \tilde{S}}{(1 + \alpha \tilde{I})^2} \\ \frac{\beta \tilde{I} e^{-\lambda \tau}}{1 + \alpha \tilde{I}} & -(\mu + \sigma) & \frac{\beta \tilde{S} e^{-\lambda \tau}}{(1 + \alpha \tilde{I})^2} \\ 0 & \sigma & -(\mu + \gamma) \end{bmatrix}$$
(6)

The eigenvalues of (6) evaluated at E_1 is $\lambda_1 = -\mu$ and the roots of equation

$$\lambda^{2} + (2\mu + \sigma + \gamma)\lambda + (\mu + \sigma)(\mu + \gamma) - \frac{\sigma\beta A e^{-\lambda \tau}}{\mu} = 0.$$
 (7)

For $\tau = 0$, equation (7) becomes

$$\lambda^{2} + (2\mu + \sigma + \gamma)\lambda + (\mu + \sigma)(\mu + \gamma)(1 - R_{0}).$$
(8)

It is easy to see that if $R_0 < 1$, then the roots of (8) have negative real parts. Therefore, the following proposition is obtained.

Proposition 1. If $R_0 < 1$, E_1 is locally asymptotically stable if and only if $\tau = 0$.

For $\tau > 0$, by corollary 2.4 in Ruan and Wei [10], it follows that if instability occurs for a particular value of the delay τ , a characteristic root of (7) must intersect the imaginary axis. Assume that $\lambda = i\omega$, $\omega > 0$ is the roots of (7). Substituting $\lambda = i\omega$ into (7) gives

$$-\omega^{2} + i(2\mu + \sigma + \gamma)\omega + (\mu + \sigma)(\mu + \gamma) - \frac{\sigma\beta A(\cos\omega\tau - i\sin\omega\tau)}{\mu} = 0.$$
⁽⁹⁾

Separating the real and imaginary parts, gives

$$\frac{\sigma\beta A}{\mu}\cos\omega\tau = (\mu+\sigma)(\mu+\gamma) - \omega^{2},$$

$$\frac{\sigma\beta A}{\mu}\sin\omega\tau = -\omega(2\mu+\sigma+\gamma).$$
(10)

Squaring and adding the two equations of (10) yields

$$\omega^{4} + [(\mu + \sigma)^{2} + (\mu + \gamma)^{2}]\omega^{2} + (\mu + \sigma)^{2}(\mu + \gamma)^{2}(1 - R_{0}^{2}) = 0.$$
(11)

It is clear that if $R_0 < 1$, then the roots of (11) have negative real parts. Then, equation (7) cannot have purely imaginary solutions. As above result and Proposition 1, the stability of disease-free equilibrium is obtained in the following theorem. Theorem 1. The disease-free equilibrium E_1 is locally asymptotically stable if and only if $\tau \ge 0$ whenever $R_0 < 1$.

C. Stability Analysis of Endemic Equilibrium

The local stability of E_2 is explored as follows. The characteristic equation of (6) evaluated at endemic equilibrium E_2 is a third degree transcendental polynomial

$$\lambda^{3} + p_{1}\lambda^{2} + p_{2}\lambda + p_{3} + (q_{2}\lambda + q_{3})e^{-\lambda\tau} = 0,$$
(12)

where

$$p_{1} = 3\mu + \sigma + \gamma + \frac{\beta\mu(R_{0} - 1)}{\beta + \mu\alpha R_{0}},$$

$$p_{2} = (\mu + \sigma)(\mu + \gamma) + \frac{\mu(2\mu + \sigma + \gamma)(\mu\alpha + \beta)R_{0}}{\beta + \mu\alpha R_{0}},$$

$$p_{3} = \frac{\mu(\mu + \sigma)(\mu + \gamma)(\mu\alpha + \beta)R_{0}}{\beta + \mu\alpha R_{0}},$$

$$q_{2} = -\frac{(\mu + \sigma)(\mu + \gamma)(\mu\alpha + \beta)}{\beta + \mu\alpha R_{0}},$$

$$q_{3} = -\frac{\mu(\mu + \sigma)(\mu + \gamma)(\mu\alpha + \beta)}{\beta + \mu\alpha R_{0}}.$$

For $\tau = 0$, equation (12) becomes

$$\lambda^{3} + a_{1}\lambda^{2} + a_{2}\lambda + a_{3} = 0, \tag{13}$$

where

$$\begin{split} a_1 &= p_1 = 3\mu + \sigma + \gamma + \frac{\beta\mu(R_0 - 1)}{\beta + \mu\alpha R_0}, \\ a_2 &= p_2 + q_2 = \frac{(\mu + \sigma)(\mu + \gamma)\mu\alpha(R_0 - 1)}{\beta + \mu\alpha R_0} \\ &+ \frac{(2\mu + \sigma + \gamma)\mu R_0(\beta + \mu\alpha)}{\beta + \mu\alpha R_0}, \\ a_3 &= p_3 + q_3 = \frac{\mu(\mu + \sigma)(\mu + \gamma)(\mu\alpha + \beta)(R_0 - 1)}{\beta + \mu\alpha R_0}. \end{split}$$

It is clear that, for $R_0 > 1$, $a_i > 0$, i = 1, 2, 3 and

$$a_{1}a_{2} - a_{3} = \frac{(\mu + \sigma)(\mu + \gamma)\mu\alpha(R_{0} - 1)}{\beta + \mu\alpha R_{0}} [(2\mu + \sigma + \gamma)]$$

$$+ \frac{(\mu + \sigma)(\mu + \gamma)\mu\alpha(R_{0} - 1)}{\beta + \mu\alpha R_{0}} \left[\frac{\beta\mu(R_{0} - 1)}{(\beta + \mu\alpha R_{0})}\right]$$

$$+ \frac{\mu^{2}(2\mu + \sigma + \gamma)R_{0}}{\beta + \mu\alpha R_{0}} \left[\frac{\beta(R_{0} - 1)(\beta + \mu\alpha)}{(\beta + \mu\alpha R_{0})}\right]$$

$$+ \frac{\mu\beta(5\mu^{2} + 4\sigma\mu + 4\gamma\mu + \sigma\gamma + \sigma^{2} + \gamma^{2})R_{0}}{\beta + \mu\alpha R_{0}}$$

$$+ \frac{\mu\beta(\mu + \sigma)(\mu + \gamma)}{\beta + \mu\alpha R_{0}} > 0.$$
(14)

Hence, using the Routh-Hurwitz criterion [11], the roots of (13) have negative real parts. Then, in the absence of time delay, the following result is obtained.

Proposition 2. If $R_0 > 1$, the endemic equilibrium E_2 is locally asymptotically stable for $\tau = 0$.

For $\tau > 0$, after substitution $\lambda = i\omega$, $\omega > 0$ into (12) and separation the real and imaginary part, yields

$$q_{3}\cos\omega\tau + q_{2}\omega\sin\omega\tau = p_{1}\omega^{2} - p_{3},$$

$$q_{2}\omega\cos\omega\tau - q_{3}\sin\omega\tau = \omega^{3} - p_{2}\omega.$$
(15)

Squaring and adding both equations gives

$$\omega^{6} + (p_{1}^{2} - 2p_{2})\omega^{4} + (p_{2}^{2} - 2p_{1}p_{3} - q_{2}^{2})\omega^{2} + p_{3}^{2} - q_{3}^{2} = 0.$$
(16)

Letting $z_1 = \omega^2$, equation (16) can be rewritten in the form

$$z_1^3 + p z_1^2 + q z_1 + r = 0, (17)$$

where

$$p = p_1^2 - 2p_2$$

= $\mu^2 + (\mu + \sigma)^2 + (\mu + \gamma)^2 + \left(\frac{\beta\mu(R_0 - 1)}{\beta + \mu\alpha R_0}\right)^2$ (18)
+ $\frac{2\beta\mu^2(R_0 - 1)}{\beta + \mu\alpha R_0}$,

$$q = p_{2}^{2} - 2p_{1}p_{3} - q_{2}^{2}$$

$$= \left(\mu + \frac{\beta\mu(R_{0} - 1)}{\beta + \mu\alpha R_{0}}\right)^{2} [2\mu(\mu + \sigma + \gamma) + \sigma^{2} + \gamma^{2}] \quad (19)$$

$$+ (\mu + \sigma)^{2}(\mu + \gamma)^{2} \left(\frac{\mu\alpha(R_{0} - 1)[2\beta + \mu\alpha(R_{0} + 1)]}{(\beta + \mu\alpha R_{0})^{2}}\right),$$

$$r = p_{3}^{2} - q_{3}^{2}$$

$$= \frac{\mu^{2}(\mu + \sigma)^{2}(\mu + \gamma)^{2}(\mu\alpha + \beta)^{2}(R_{0}^{2} - 1)}{(\beta + \mu\alpha R_{0})^{2}}.$$
(20)

It is found that if $R_0 > 1$, p, q and r given in (18)-(20), respectively, are positive which satisfy Lemma 3.3.1 in [2]. Then, the following theorem is obtained.

Theorem 2. If $R_0 > 1$, the endemic equilibrium E_2 is locally asymptotically stable for $\tau \ge 0$.

III. NUMERICAL SIMULATIONS

In this section the dynamic behaviors of the delayed model (2) is investigated by integrating numerically the system (2) with the following set of parameters [12]:

$$A = 10, \ \mu = 0.2, \ \sigma = 1.2, \ \gamma = 0.4, \ \alpha = 2 \tag{21}$$

whereas β and delay τ are varied.

Fig. 1 (a)-Fig. 1 (b) depict the numerical results of the system (2) when $R_0 < 1$. It is evident from these figures that all numerical solutions converged to the disease-free equilibrium, E_1 (in line with Theorem 1) for the case $R_0 < 1$. Numerical results, in Fig. 2, shows convergence of the solutions to endemic equilibrium, E_2 (in line with Theorem 2) whenever $R_0 > 1$.

The effect of the time delay is monitored by varying the values of τ . The results are shown in Fig. 3. It is found that, when $\tau = 0$, the number of infectious individuals monotone increasing approach endemic equilibrium. When the value of time delay increased, compared with $\tau = 0$, the number of infectious individuals on days 1-14 decrease, and after that approach the same endemic equilibrium.



Fig. 1. Simulation of the system (2) showing the variation of population with time. The parameter used are A = 10, $\mu = 0.2$, $\sigma = 1.2$, $\gamma = 0.4$, $\alpha = 2$, $\beta = 0.01$ and $\tau = 1$.



Fig. 2. Simulation of the system (2) showing the variation of population with time. The parameter used are A = 10, $\mu = 0.2$, $\sigma = 1.2$,

 $\gamma = 0.4$, $\alpha = 2$, $\beta = 1$ and $\tau = 1$.



Fig. 3. The profile solution of the system (2) for $\tau = 0$, $\tau = 1$, $\tau = 2$ and $\tau = 3$. The parameter used are A = 10, $\mu = 0.2$, $\sigma = 1.2$, $\gamma = 0.4$, $\alpha = 2$ and $\beta = 1$.

IV. CONCLUSIONS

In this paper, a *SEIR* model with saturated incidence rate of the form $\frac{\beta S(t)I(t)}{1+\alpha I(t)}$ and time delay is formulated. The model has two positive equilibria, namely, the disease-free equilibrium and the endemic equilibrium. Detailed local stability analysis of the model reveals that the equilibrium state E_1 , corresponding to disappearance of disease is locally asymptotically stable if $R_0 < 1$ for all $\tau \ge 0$. When $R_0 > 1$, a unique endemic equilibrium E_2 exists and is locally asymptotically stable for all $\tau \ge 0$. This implies that the infection is maintained in the population.

The effect of time delay on the model will delay the transmission of disease in the sense that the number of infectious individuals decrease in the short period. Moreover, even in the early days the longer of time delay seems to give the lower of the infectious individuals but later the number of infectious individuals reach endemic equilibrium point. So this result indicates that, the time delay representing the fixed latency period of the disease has no effect on the stability of feasible equilibria.

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