Far East Journal of Mathematical Sciences (FJMS)

Published Online: January 2016
http://dx.doi.org/10.17654/MS099040607
Volume 99, Number 4, 2016, Pages 607-614 **ISSN: 0972-0871**

STABILITY AND HOPF BIFURCATION ANALYSIS OF DELAYED SIRS MODEL WITH SATURATED INCIDENCE

Rubono Setiawan

Mathematics Education
Faculty of Teacher Training and Education Sciences
Sebelas Maret University
Ir. Sutami No. 36 A, Kentingan, Jebres
Surakarta, Indonesia
e-mail: rubono.matematika@staff.uns.ac.id

Abstract

In this paper, we analyze the local dynamics of delayed SIRS epidemic model with modified saturated incidence rate. We take into account the incubation time length as time delay into the saturated force of incidence with two saturation factors. Local stability and Hopf bifurcation of disease free and disease equilibrium due to delay time effect have been analyzed.

1. Introduction

In this research, we use SIRS model as basic model. There are three different compartments of this model: susceptible individuals (S), infectious (infective) individuals (I), recovered individuals (R). We assume that a

Received: October 15, 2015; Revised: November 14, 2015; Accepted: December 4, 2015
2010 Mathematics Subject Classification: 92B05, 92-08, 93D20.
Keywords and phrases: Hopf bifurcation, delayed SIRS, saturated incidence.
Communicated by K. K. Azad

recovered individual only has temporary immunity. In 1978, Anderson and May [4] proposed the saturated incidence rate in the form $\frac{\beta SI}{1+\alpha S}$ and then used by some authors (Gao et al. [5], Zhang and Teng [6], Meng et al. [8] and Wei and Chen [1]). The effect of saturated incidence, which is referred to α, stems from epidemical control. In some conditions, the inhibitory effect also comes from infected individuals like self protection by infected individuals or psychological behaviour of infected individuals to stay away from healthy people, so it can be associated with the other saturation factor, which is related to infected individuals. In order to accommodate the natural bahaviour of susceptible and infected individuals and to support the effectivity of disease controlling strategies, we use saturated incidence with two saturation factors α_1 and α_2, which measure the inhibitory effect from susceptible and infected individuals, respectively.

One of representations of time delay is incubation time period of disease and it usually includes in the force of infection as bifurcation parameter. For example, in Zhang et al. [2], Gao et al. [5], Zhang and Teng [6], Jiang and Wei [7] and Meng et al. [8]. Cooke [3] proposed the incubation time period τ in the force of infection $\beta S(t) I(t-\tau)$. Zhang et al. [2] used the incubation time period as time delay and took it into saturated force of infection with single saturated factor $\frac{\beta S(t) I(t-\tau)}{1+\alpha S(t)}$. In this paper, we use saturated incidence with two saturation factors and also take time delay into those saturation factors.

2. Mathematical Model Formulation

We assume that population is not constant and the susceptible host population is given by logistic growth with carrying capacity K and a specific growth rate constant r. In order to have more realistic model, we use saturated incidence rate with two saturation factors $\frac{\beta S(t) I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)}$, which measure the inhibitory effect from susceptible and infected individuals, respectively. In this paper, we assume that it may be due to the self protection by infected individuals or psychological behaviour of infected individuals to stay away from healthy people. Finally, we added the constant time delay $\tau > 0$ as representing incubation time length into saturated incidence form as bifurcation parameter. So, we get generalized SIRS model with time delay as follows:

$$\frac{dS(t)}{dt} = r\left(1 - \frac{S(t)}{K}\right)S(t) - \frac{\beta S(t)I(t-\tau)}{1 + \alpha_1 S(t) + \alpha_2 I(t-\tau)} + \delta R(t),$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1 + \alpha_1 S(t) + \alpha_2 I(t-\tau)} - \mu I(t) - \gamma I(t),$$

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

$$N(t) = S(t) + I(t) + R(t), \tag{2.1}$$

where μ is per capita natural death rate, γ is per capita recovery rate of the infected individuals and δ is per capita loss of immunity rate of recovered individuals.

3. Stability and Bifurcation Occurrence Analysis

Theorem 3.1. (i) *If* $\mathfrak{R}_0 = 1$ *and* $\Phi = 1$, *then there exist two disease free equilibriums*, *that is*, $E_K = (K, 0, 0)$ *and* $E_0 = (0, 0, 0)$.

(ii) *If* $\mathfrak{R}_0 > 1$ *and* $\Phi > 1$, *then there exists a disease equilibrium* $E_1 = (S^*, I^*, R^*)$, *where*

$$S^* = \frac{(1 + \alpha_2 I^*)P_2}{(\beta - \alpha_1 P_2)},$$

$$I^* = \frac{\left(\frac{1}{2}\mathfrak{R}_0 + \frac{1}{2}\Phi\mathfrak{R}_0 - 1\right)}{\alpha_2} + \frac{\sqrt{\left(\frac{1}{2}\mathfrak{R}_0 + \frac{1}{2}\Phi\mathfrak{R}_0 - 1\right)^2 + \mathfrak{R}_0 - 1}}{\alpha_2},$$

$$R^* = \frac{P_1}{\gamma} I^*,$$

where $\mathfrak{R}_0 = \dfrac{K(\beta - \alpha_1 P_2)}{P_2}$ *and* $\Phi = \dfrac{\left(\dfrac{\delta\eta_2}{\gamma} - P_2\right)(\beta - \alpha_1 P_2)}{P_2 \alpha_2 r}$, *where* $P_2 = (\gamma + \mu)$ *and* $P_1 = (\delta + \mu)$.

By linearized process at any equilibrium $\hat{E} = (\hat{S}, \hat{E}, \hat{I}, \hat{R})$ of system (2.1), then E_k becomes locally asymptotically stable if and only if $\mathfrak{R}_0 > 1$ and $\tau = 0$. Otherwise, E_k is not stable.

If we take $\tau > 0$ into linearized equation and evaluate at $E_k = (K, 0, 0)$, then we get two equilibriums with negative real part, $\lambda_1 = -r$ and $\lambda_3 = -(\delta + \mu)$. The other roots satisfy the following equation:

$$F(\lambda) = \lambda - (\gamma + \mu) - \frac{\beta K e^{-\lambda\tau}}{(1 + \alpha_1 K)} = 0. \tag{3.1}$$

Let $\lambda = a + i\omega$, where $a, \omega \in \mathbb{R}$ is the root of equation (3.1). By separating the real and imaginary parts, and then squaring and adding those both equations, we get

$$\omega^2 = -(\gamma + \mu)^2 + 2a(\gamma + \mu) - a^2 + \left(\frac{\beta K}{(1 + \alpha_1 K)}\right)^2 e^{-2a\tau}. \tag{3.2}$$

When $\mathfrak{R}_0 > 1$ and $\omega_0^2 = \left(\dfrac{\beta K}{1 + \alpha_1 K}\right)^2 - (\gamma + \mu)^2$, then $a = 0$. That is, (3.2) has a unique pair of purely imaginary roots $\lambda = \pm i\omega_0$. From (3.2), we get τ_n corresponding to ω_0

$$\tau_0 = \frac{\arctan\left(-\dfrac{\omega_0}{(\gamma + \mu)}\right)}{\omega_0} + \frac{2n\pi}{\omega_0}, \quad n = 0, 1, 2, \ldots \tag{3.3}$$

with $\frac{2\pi}{\omega_0} < \tau_0 < \frac{3\pi}{2\omega_0}$. For $n = 0$, we get a critical value of delay

$$\tau_0 = \frac{\arctan\left(-\frac{\omega_0}{(\gamma + \mu)}\right)}{\omega_0}. \tag{3.4}$$

Theorem 3.2. *For system* (2.1), *if* $\Re_0 > 1$ *and* $\omega^2 = \left(\frac{\beta K}{1 + \alpha_1 K}\right)^2 - (\gamma + \mu)^2$, *then there is also a critical value of delay time* τ_0 *such that* $E_k = (K, 0, 0)$ *is still asymptotically stable when* $\tau_0 \in (0, \tau_0)$ *and becomes unstable*. *Furthermore*, *system* (2.1) *undergoes Hopf bifurcation at* $E_k = (K, 0, 0)$, *when* $\tau = \tau_n$, $n = 0, 1, 2, 3, \ldots$.

Then it is easy to prove that E_0 is always an unstable saddle point. Finally, we will study the stability of $E_1 = (S^*, I^*, R^*)$ by two possible cases of time delay τ, that is, when $\tau = 0$ and $\tau > 0$. If $\tau = 0$, then we have the following characteristic equation at E_1:

$$\begin{aligned}
&\lambda^3 + (P_1 + P_2 + M + P - Q)\lambda^2 \\
&+ [P_1(P_2 - Q + M + P) + (M + P)(P_2 - Q) + PQ]\lambda \\
&+ P_1(M + P)(P_2 - Q) + PQP_1 - P\delta\gamma = 0,
\end{aligned} \tag{3.5}$$

where

$$P = \frac{\beta I^*(1 + \alpha_2 I^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2}, \quad Q = \frac{\beta I^*(1 + \alpha_1 S^*)}{(1 + \alpha_1 S^* + \alpha_2 I^*)^2},$$

$$M = -r + \frac{2r}{K} S^* P_1 = (\delta + \mu), \quad P_2 = (\gamma + \mu).$$

Using Routh-Hurwitz criterion, we get the following conditions:

(i) $\frac{I^*}{S^*} > \frac{\alpha_1}{\alpha_2}$,

(ii) $\frac{S^*}{K} > 0.5,$

(iii) $\frac{\delta\gamma}{P_1} < Q < P_2.$ (3.6)

If all the conditions (3.6) hold, then all of the eigenvalues of (3.5) lie in the left half plane. Therefore, E_1 is locally asymptotically stable equilibrium when $\tau = 0$, otherwise, if one of the conditions (3.6) is not satisfied, then E_1 is not stable equilibrium for $\tau = 0$. When $\tau > 0$, we consider the following characteristic equation (3.5) at E_1:

$$\lambda^3 + (P_1 + P_2 + M + P - Qe^{-\lambda\tau})\lambda^2$$
$$+ [P_1(P_2 - Qe^{-\lambda\tau} + M + P) + (M + P)(P_2 - Qe^{-\lambda\tau}) + PQe^{-\lambda\tau}]\lambda$$
$$+ P_1(M + P)(P_2 - Qe^{-\lambda\tau}) + PQe^{-\lambda\tau}P_1 - P\delta\gamma = 0. \tag{3.7}$$

If $\lambda = i\omega\ (\omega > 0)$ is the root of (3.7), then by separating the real and imaginary parts, squaring and adding both equations, then we get

$$\omega^6 + \omega^4[(M + P)^2 + P_1^2 + P_2^2 - Q^2]$$
$$+ \omega^2[P_1^2P_2^2 + (M + P)^2(P_1^2 + P_2^2)$$
$$+ 2P\delta\gamma(P_1 + P_2 + M + P) - Q^2(M^2 + P_1^2)]$$
$$+ [(P_1P_2(M + P) - P\delta\gamma)^2 - (QMP_1)^2] = 0. \tag{3.8}$$

If there exists a positive value of ω_0 that satisfies (3.8), then τ_n corresponding to ω_0 can be obtained as follows:

$$\tau_0 = \frac{1}{\omega_0}\arccos\left[\frac{-M_1\omega_0^4 + \omega_0^2[M_1M_4 + M_1M_2P_2 + M_3P_2(M_1 + P_2 + M_4 - P\delta\gamma)]}{[\omega_0^2M_1^2 - M_3^2]Q}\right], \tag{3.9}$$

where

$$M_1 = M + P_1,\ M_2 = M + P + P_1,\ M_3 = P_1 M - \omega_0^2,\ M_4 = P_1(M + P).$$

Then it is easy to prove that $sign\left\{Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\right\}_{\lambda = i\omega_0} > 0$ if and only if the following conditions hold:

(i) $M > 1$,

(ii) $0 < Q < 1$,

(iii) $V > 3\omega_0^2$,

(iv) $Q\tau(\omega_0^2 + P_1 M) < 1$. (3.10)

Hence, if there exists critical time delay τ_0 in the form (3.9) with certain specific conditions and also satisfies transversality condition in the form (3.10), then system (2.1) undergoes locally Hopf bifurcation at endemic equilibrium E_1.

4. Conclusion

In this paper, the local stability and bifurcation analysis of disease and disease free equilibriums have been analyzed. We have proved that the stability of disease and disease free equilibriums and existence of a critical value of time delay. When the time delay passes a critical value, then a stable equilibrium point will turn into an unstable equilibrium and we say that the equilibrium point is experiencing locally Hopf bifurcation. For further research, there are several possible extensions of this work, like global stability and bifurcation analysis.

Acknowledgement

The author thanks the Editor in Chief and anonymous referees for their valuable comments and suggestions.

References

[1] C. Wei and L. Chen, A delayed epidemic model with pulse vaccination, Discrete Dyn. Nat. Soc. (2008).

[2] J. Z. Zhang, Z. Jin, Q. X. Liu and Z. Y. Zhang, Analysis of a delayed SIR model with nonlinear incidence, Discrete Dyn. Nat. Soc. (2008).

[3] K. L. Cooke, Stability analysis for a vector disease model, Rocky Mountain J. Math. 9(1) (1979), 31-42.

[4] R. M. Anderson and R. M. May, Regulation and stability of host-parasite population interactions: I. Regulatory processes, Journal of Animal Ecology 47(1) (1978), 219-267.

[5] S. Gao, L. Chen, J. J. Nieto and A. Torres, Analysis of a delayed epidemic model with pulse vaccination and saturation incidence, Vaccine 24 (2006), 6037-6045.

[6] T. Zhang and Z. Teng, Pulse vaccination delayed SEIRS epidemic model with saturation incidence, Applied Mathematical Modelling 32 (2008), 1403-1416.

[7] Z. Jiang and J. Wei, Stability and bifurcation analysis in a delayed SIR model, Chaos Solitons Fractals 35 (2008), 609-619.

[8] X. Meng, L. Chen and H. Cheng, Two profitless delays for the SEIRS epidemic disease model with nonlinear incidence and pulse vaccination, Appl. Math. Comput. 186(1) (2007), 516-529.