Research Article

Global Stability of a Delayed SIRS Epidemic Model with Nonlinear Incidence

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In this paper we propose the global dynamics of an SIRS epidemic model with latency and a general nonlinear incidence function. The model is based on the susceptible-infective-recovered (SIR) compartmental structure with relapse (SIRS). Sufficient conditions for the global stability of equilibria (the disease-free equilibrium and the endemic equilibrium) are obtained by means of Lyapunov-LaSalle theorem. Also some numerical simulations are given to illustrate this result.

1. Introduction

Epidemic models have long been an important tool for understanding and controlling the spread of infectious diseases. Most of them are described by delayed differential equations. The introduction of time delay is often used to model the latent period, that is, the time from the acquisition of infection to the time when the host becomes infectious [1, 2].

Recently, considerable attention has been paid to model the relapse phenomenon, that is, the return of signs and symptoms of a disease after a remission. Hence, the recovered individual can return to the infectious class (see [3–6]). For the biological explanations of the relapse phenomenon, we cite two examples.

- (i) For malaria, Bignami [7] proposed that relapses derived from persistence of small numbers of parasite in the blood. Also, it has been observed that the proportion of patients who have successive relapses is relatively constant (see [8]).
- (ii) For tuberculosis, relapse can be caused by incomplete treatment or by latent infection, being observed that HIV-positive patients are significantly more likely to relapse than HIV-negative patients, although it is often difficult to differentiate relapse from reinfection (see [9]).

In this paper, we propose the following epidemic model with time delay and relapse (delayed SIRS epidemic model) as follows (see [10, 11]):

$$\begin{aligned}\frac{dS}{dt} &= A - \mu S - f(S, I), \\ \frac{dI}{dt} &= e^{-\mu\tau} f(S_\tau, I_\tau) - (\mu + \gamma + \alpha) I + \delta R, \\ \frac{dR}{dt} &= \gamma I - (\mu + \delta) R.\end{aligned}\quad (1)$$

The initial condition for the above system is

$$\begin{aligned}S(\theta) &= \varphi_1(\theta), \\ I(\theta) &= \varphi_2(\theta), \\ R(\theta) &= \varphi_3(\theta), \\ \theta &\in [-\tau, 0],\end{aligned}\quad (2)$$

with $\varphi = (\varphi_1, \varphi_2, \varphi_3) \in C^+ \times C^+ \times C^+$, such that $\varphi_i(\theta) \geq 0$ ($-\tau \leq \theta \leq 0$, $i = 1, 2, 3$). Here C denotes the Banach space $C([-\tau, 0], R)$ of continuous functions mapping the interval $[-\tau, 0]$ into R , equipped with the supremum norm. The nonnegative cone of C is defined as $C^+ = C([-\tau, 0], R^+)$.

Here $\psi_\tau = \psi(t - \tau)$ for any given function ψ , $A = \mu N$, where $N = S + I + R$ is the total number of population, 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, f is the nonlinear incidence function, γ is the recovery rate of the infective individuals, δ is the rate that recovered individuals relapse and regained infectious class, and τ is the latent period.

In model (1) the incidence function $f(S, I)$ is a locally Lipschitz continuous function on $R^+ \times R^+$ satisfying $f(0, I) = f(S, 0) = 0$ for $S \geq 0, I \geq 0$, and the following hold:

- (H_1) f is a strictly monotone increasing function of $S \geq 0$, for any fixed $I > 0$, and f is a strictly monotone increasing function of $I \geq 0$, for any fixed $S \geq 0$.
- (H_2) $\phi(S, I) = f(S, I)/I$ is a bounded and monotone decreasing function of $I > 0$, for any fixed $S \geq 0$, and $K(S) = \lim_{I \rightarrow 0^+} \phi(S, I)$ is a continuous and monotone increasing function on $S \geq 0$.

This incidence function includes different forms presented in literature (see, e.g., [12–23]).

System (1) always has a disease-free equilibrium $P_0 = (A/\mu, 0, 0)$. On the other hand, under the hypothesis (H_2), if

$$R_0 := \frac{K(A/\mu) e^{-\mu\tau}}{\mu + \gamma + \alpha - \gamma\delta/(\mu + \delta)} > 1, \quad (3)$$

then system (1) also admits a unique endemic equilibrium $P^* = (S^*, I^*, R^*)$, where S^*, I^* , and R^* satisfy the following system (see [24]):

$$\begin{aligned} A - \mu S - f(S, I) &= 0, \\ e^{-\mu\tau} f(S, I) - (\mu + \gamma + \alpha) I + \delta R &= 0, \\ \gamma I - (\mu + \delta) R &= 0. \end{aligned} \quad (4)$$

Hereafter, we replace $\mu + \gamma + \alpha - \gamma\delta/(\mu + \delta)$ by η .

In [5] (1990), Tudor developed and analyzed qualitatively one of the first SIRI epidemic models for the spread of a herpes-type infection in either human or animal populations. This model consists of a system of nonlinear ordinary differential equations with a bilinear incidence rate (i.e., $f(S, I) = \beta SI$) and a constant total population (i.e., $N = S + I + R = \text{constant}$).

In [25] (1997), Moreira and Wang extended a Tudor-model to include nonlinear incidence functions. By using an elementary analysis of Liénard's equation and Lyapunov's direct method, they derived sufficient conditions for the global asymptotic stability of the disease-free and endemic equilibria.

In [23] (2000), Castillo-Garsow et al. considered an SIRI model for drug use in a population of adolescents. The authors assumed that $N = \text{constant}$ and $f(S, I) = \beta SI/N$; they estimated the parameters of the model and they determined a rough approximation of the basic reproductive number. Based on these parameters, they performed some

simulations that clearly showed the endemic character of tobacco use among adolescents.

In [26] (2004), Blower developed a compartmental model for genital herpes, assuming standard incidence rate (i.e., $f(S, I) = \beta SI/N$) and constant recruitment rate.

In [24] (2006), Korobeinikov proved that the endemic equilibrium of the model (1) with $\delta = 0$ and $\tau = 0$ is globally asymptotically stable.

In [27] (2007), van den Driessche and Zou proposed an integrodifferential equation to model a general relapse phenomenon in infectious diseases. The resulting model, in particular case, is a delay differential equation with a constant population and standard incidence. The basic reproduction number for this model is identified and some global results are obtained by employing the Lyapunov-Razumikhin technique.

In [10] (2007), Van Den Driessche and co-authors formulated a delay differential SIRI model (System (1) with $f(S, I) = \beta SI$). For this system, the endemic equilibrium is locally asymptotically stable if $R_0 > 1$, and the disease is shown to be uniformly persistent with the infective population size either approaching or oscillating about the endemic level.

In [22] (2011), Liu et al. proposed a mathematical model for a disease with a general exposed distribution, the possibility of relapse and nonlinear incidence rate ($f(S, I) = \beta SI/N$). By the method of Lyapunov functionals, they showed that the disease dies out if $R_0 = 1$ and that the disease becomes endemic if $R_0 > 1$. They also analyzed, as a special case of this model, the system (1) with $f(S, I) = g(S) \cdot I$; the result confirms that the endemic equilibrium is globally asymptotically stable.

In [28] (2012), Abta et al. considered a global asymptotic stability of a delayed SIR model (system (1) with $f(S, I) = \beta SI/(1 + \alpha_1 S + \alpha_2 I)$ and $\delta = 0$).

In [29] (2013), Vargas-De-Leon presented the global stability conditions of an ordinary SIRI model with bilinear and standard incidence rates, respectively, that includes recruitment rate of susceptible individuals into the community and that the disease produces nonnegligible death in the infectious class. The author presented the construction of Lyapunov functions using suitable combinations of known functions, common quadratic and Volterra-type, and a composite Volterra-type function.

In [21] (2013), Georgescu and Zhang analyzed the dynamics of an ordinary SIRI model under the assumption that the incidence of infection is given by $f(S, I) = C(S)g(I)$. They obtained by means of Lyapunov's second method sufficient conditions for the local stability of equilibria and they showed that global stability can be attained under suitable monotonicity conditions.

In [30] (2013), Shuai and van den Driessche presented two systematic methods for the construction of Lyapunov functions for general infectious disease models (Ordinary SEIRI, SIS, etc.). Specifically, a matrix-theoretic method using the Perron eigenvector is applied to prove the global stability of the disease-free equilibrium, while a graph-theoretic method based on Kirchhoff's matrix tree theorem and two new combinatorial identities are used to prove the global stability of the endemic equilibrium.

In [31] (2014), Xu investigated a delayed SIRS model (system (1) with $f(S, I) = \beta SI$). The author established the global stability of a disease-free equilibrium and an endemic equilibrium by means of suitable Lyapunov functionals and LaSalle's invariance principle.

In this paper we extend the global stability results presented in [31] to a delayed SIRS epidemic model (system (1)) with a general nonlinear incidence function. It is shown that global stability can be attained under suitable monotonicity conditions and it is established that the basic reproduction number R_0 is a threshold parameter for the stability of a delayed SIRS model. The rest of the paper is organized as follows. In Section 2, the global stability of disease-free and endemic equilibria are established. In Section 3, numerical simulations and concluding remarks are provided. In the appendix, some results on the global stability are stated.

2. Global Stability Analysis of Delayed SIRS Model

In this section, we discuss the global stability of a disease-free equilibrium P_0 and an endemic equilibrium P^* of system (1). Since $(d/dt)(S + I + R) \leq A - \mu(S + I + R)$, we have $\limsup(S + I + R) \leq A/\mu$. Hence we discuss system (1) in the closed set

$$\Omega = \left\{ (\varphi_1, \varphi_2, \varphi_3) \in C^+ \times C^+ \times C^+ : \|\varphi_1 + \varphi_2 + \varphi_3\| \leq \frac{A}{\mu} \right\}. \quad (5)$$

It is easy to show that Ω is positively invariant with respect to system (1). Next we consider the global asymptotic stability of the disease-free equilibrium P_0 and the endemic equilibrium P^* of (1) by Lyapunov functionals, respectively.

Proposition 1. *If $R_0 \leq 1$, then the disease-free equilibrium P_0 is globally asymptotically stable.*

Proof. Define a Lyapunov functional

$$V_0(t) = e^{-\mu t} \int_{A/\mu}^{S_t} \left(1 - \frac{K(A/\mu)}{K(u)} \right) du + I + \eta \int_{t-\tau}^t I(u) du + \frac{\delta}{\mu + \delta} R. \quad (6)$$

We will show that $dV_0(t)/dt \leq 0$ for all $t \geq 0$. We have

$$\begin{aligned} \frac{dV_0(t)}{dt} &= e^{-\mu t} \left(1 - \frac{K(A/\mu)}{K(S_t)} \right) \dot{S}_t + e^{-\mu t} f(S_t, I_t) - (\mu + \gamma + \alpha) I + \delta R + \eta (I - I_t) \\ &\quad + \frac{\gamma \delta}{\mu + \delta} I - \delta R \end{aligned}$$

$$\begin{aligned} &= e^{-\mu t} \left(1 - \frac{K(A/\mu)}{K(S_t)} \right) (A - \mu S_t) \\ &\quad + \frac{K(A/\mu)}{K(S_t)} e^{-\mu t} f(S_t, I_t) - \eta I_t \\ &= \mu e^{-\mu t} \left(1 - \frac{K(A/\mu)}{K(S_t)} \right) \left(\frac{A}{\mu} - S_t \right) \\ &\quad + \eta I_t \left(\frac{\phi(S_t, I_t)}{\eta} \frac{K(A/\mu)}{K(S_t)} e^{-\mu t} - 1 \right). \end{aligned} \quad (7)$$

By the hypothesis (H_1) , we obtain that

$$\left(1 - \frac{K(A/\mu)}{K(S_t)} \right) \left(\frac{A}{\mu} - S_t \right) \leq 0, \quad (8)$$

where equality holds if and only if $S = A/\mu$.

Furthermore, It follows from the hypothesis (H_2) that

$$\begin{aligned} \frac{\phi(S_t, I_t)}{\eta} \frac{K(A/\mu)}{K(S_t)} e^{-\mu t} &\leq \frac{K(S_t)}{\eta} \frac{K(A/\mu)}{K(S_t)} e^{-\mu t} \\ &\leq \frac{K(A/\mu)}{\eta} e^{-\mu t} \\ &\leq R_0. \end{aligned} \quad (9)$$

Therefore, $R_0 \leq 1$ ensures that $dV_0(t)/dt \leq 0$ for all $t \geq 0$, where $dV_0(t)/dt = 0$ holds if $(S, I, R) = (A/\mu, 0, 0)$. Hence, it follows from system (1) that $\{P_0\}$ is the largest invariant set in $\{(S, I, R) \mid dV_0(t)/dt = 0\}$. From the Lyapunov-LaSalle asymptotic stability, we obtain that P_0 is globally asymptotically stable. This completes the proof. $\square$

Proposition 2. *If $R_0 > 1$, then the endemic equilibrium P_1^* is globally asymptotically stable.*

Proof. To prove global stability of the endemic equilibrium, we define a Lyapunov functional $V(t) = V_1(t) + V_2(t) + V_3(t) + V_+$, with

$$\begin{aligned} V_1(t) &= S - S^* - \int_{S^*}^S \frac{f(S^*, I^*)}{f(u, I^*)} du, \\ V_2(t) &= e^{\mu t} \left(I - I^* - I^* \ln \frac{I}{I^*} \right), \\ V_3(t) &= \frac{\delta e^{\mu t}}{\mu + \delta} \left(R - R^* - R^* \ln \frac{R}{R^*} \right), \\ V_+ &= \int_{t-\tau}^t \left(f(S, I) - f(S^*, I^*) - f(S^*, I^*) \right. \\ &\quad \left. \cdot \ln \frac{f(S(u), I(u))}{f(S^*, I^*)} \right) du. \end{aligned} \quad (10)$$

The time derivative of the function $V(t)$ along the positive solution of system (1) is

$$\begin{aligned} \frac{dV(t)}{dt} = & \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (A - \mu S - f(S, I)) \\ & + e^{\mu\tau} \left(1 - \frac{I^*}{I}\right) \\ & \cdot (e^{-\mu\tau} f(S_\tau, I_\tau + \delta R) - (\mu + \alpha + \gamma) I) \\ & + \frac{\delta e^{\mu\tau}}{\mu + \delta} \left(1 - \frac{R^*}{R}\right) (\gamma I - (\mu + \delta) R) \\ & + f(S, I) - f(S_\tau, I_\tau) + f(S^*, I^*) \\ & \cdot \ln \frac{f(S_\tau, I_\tau)}{f(S, I)}. \end{aligned} \quad (11)$$

Using the relation $A = \mu S^* + f(S^*, I^*)$, simple calculations give

$$\begin{aligned} \frac{dV(t)}{dt} = & \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (-\mu(S - S^*) + f(S^*, I^*)) \\ & + \frac{f(S^*, I^*)}{f(S, I^*)} f(S, I) - e^{\mu\tau} (\mu + \alpha + \gamma) I \\ & - \frac{I^*}{I} f(S_\tau, I_\tau) - \delta e^{\mu\tau} \frac{I^*}{I} R + e^{\mu\tau} (\mu + \alpha + \gamma) I^* \\ & + \frac{\delta e^{\mu\tau}}{\mu + \delta} \left(\gamma I - \gamma I \frac{R^*}{R} + (\mu + \delta) R^*\right) \\ & + f(S^*, I^*) \ln \frac{f(S_\tau, I_\tau)}{f(S, I)}. \end{aligned} \quad (12)$$

Here by using

$$\begin{aligned} e^{\mu\tau} (\mu + \alpha + \gamma) I^* - \frac{\delta e^{\mu\tau}}{\mu + \delta} \gamma I^* &= f(S^*, I^*), \\ (\mu + \delta) R^* &= \gamma I^*, \end{aligned}$$

$$\begin{aligned} \ln \frac{f(S_\tau, I_\tau)}{f(S, I)} &= \ln \frac{f(S^*, I^*)}{f(S, I^*)} + \ln \frac{I^* f(S_\tau, I_\tau)}{I f(S^*, I^*)} + \ln \frac{I f(S, I)}{I^* f(S, I)}, \end{aligned} \quad (13)$$

straightforward calculations give

$$\begin{aligned} \frac{dV(t)}{dt} = & -\mu \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (S - S^*) \\ & - f(S^*, I^*) \left(\frac{f(S^*, I^*)}{f(S, I^*)} - 1 - \ln \frac{f(S^*, I^*)}{f(S, I^*)}\right) \\ & - f(S^*, I^*) \left(\frac{I^* f(S_\tau, I_\tau)}{I f(S^*, I^*)} - 1 - \ln \frac{I^* f(S_\tau, I_\tau)}{I f(S^*, I^*)}\right) \\ & + f(S^*, I^*) \left(\frac{f(S, I)}{f(S, I^*)} - \frac{I}{I^*} + \frac{I f(S, I)}{I^* f(S, I)} - 1\right) \\ & - f(S^*, I^*) \left(\frac{I f(S, I)}{I^* f(S, I)} - 1 - \ln \frac{I f(S, I)}{I^* f(S, I)}\right) \\ & + \frac{\delta \gamma I^* e^{\mu\tau}}{\mu + \delta} \left(2 - \frac{I^* R}{I R^*} - \frac{I R^*}{I^* R}\right) \\ = & -\mu \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (S - S^*) \\ & - f(S^*, I^*) \left(\frac{f(S^*, I^*)}{f(S, I^*)} - 1 - \ln \frac{f(S^*, I^*)}{f(S, I^*)}\right) \\ & - f(S^*, I^*) \left(\frac{I^* f(S_\tau, I_\tau)}{I f(S, I^*)} - 1 - \ln \frac{I^* f(S_\tau, I_\tau)}{I f(S, I^*)}\right) \\ & + \frac{I}{I^*} f(S^*, I^*) \left(1 - \frac{f(S, I)}{f(S, I^*)}\right) \left(\frac{\phi(S, I)}{\phi(S, I^*)} - 1\right) \\ & - f(S^*, I^*) \left(\frac{I f(S, I)}{I^* f(S, I)} - 1 - \ln \frac{I f(S, I)}{I^* f(S, I)}\right) \\ & - \frac{\delta \gamma e^{\mu\tau}}{\mu + \delta} \frac{I R^*}{R} \left(\frac{I^* R}{I R^*} - 1\right)^2. \end{aligned} \quad (14)$$

It follows from (H_1) and (H_2) that

$$\begin{aligned} -\mu \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (S - S^*) &\leq 0, \\ \frac{I}{I^*} f(S^*, I^*) \left(1 - \frac{f(S, I)}{f(S, I^*)}\right) \left(\frac{\phi(S, I)}{\phi(S, I^*)} - 1\right) &\leq 0. \end{aligned} \quad (15)$$

Furthermore, since the function $g(x) = 1 - x + \ln(x)$ is always nonpositive for any $x > 0$, and $g(x) = 0$ if and only if $x = 1$, then $dV(t)/dt \leq 0$, for all $t \geq 0$, where the equality holds only at the equilibrium point (S^*, I^*, R^*) . Hence, the functional V satisfies all the conditions of Theorem A.2. This proves that P_1^* is globally asymptotically stable. $\square$

3. Numerical Simulations and Concluding Remarks

In this section, we give a numerical simulation supporting the theoretical analysis given in Section 2. Let

$$f(S, I) = \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I}. \quad (16)$$

We take the parameters of the system (1) as follows:

$$\begin{aligned} A &= 10, \\ \alpha_1 &= 0.9, \\ \alpha_2 &= 0.9, \\ \mu &= 0.01, \\ \gamma &= 0.02, \\ \alpha &= 0.005, \\ \beta &= 0.1, \\ \delta &= 0.001, \\ \tau &= 10. \end{aligned} \quad (17)$$

By Proposition 2, the endemic equilibrium P^* is globally asymptotically stable; see Figure 1.

In this paper, we presented a mathematical analysis and numerical simulations for an SIRI epidemiological model applied to the evolution of the spread of disease with relapse in a given population. We denote R_0 the basic reproduction number. It is defined as the average number of contagious persons infected by a typical infectious in a population of susceptible. We prove in this paper that the basic reproduction number, R_0 , depends on the incubation period and we show that the disease-free equilibrium P_0 is globally asymptotically stable if $R_0 \leq 1$ and that a unique endemic equilibrium P^* is globally asymptotically stable if $R_0 > 1$.

Appendix

The Lyapunov-LaSalle Theorem

In the following, we present the method of Lyapunov functionals in the context of a delay differential equations:

$$\frac{dx}{dt} = f(x_t), \quad (A.1)$$

where $f : C \rightarrow R^n$ is completely continuous and solutions of (A.1) are unique and continuously dependent on the initial data. We denote by $x(\phi)$ the solution of (A.1) through $(0, \phi)$. For a continuous functional $V : C \rightarrow R$, we define

$$\dot{V} = \limsup_{h \rightarrow 0^+} \frac{1}{h} [(Vx_h(\phi)) - V(\phi)], \quad (A.2)$$

the derivative of V along a solution of (A.1). To state the Lyapunov-LaSalle type theorem for (A.1), we need the following definition.

FIGURE 1: Solutions (S, I, R) of the SIRI epidemic model (1) are globally asymptotically stable and converge to the endemic equilibrium P^* .

Definition A.1 (see [32, page 30]). We say $V : C \rightarrow R$ is a Lyapunov functional on a set G in C for (A.1) if it is continuous on $\bar{G}$ (the closure of G) and $\dot{V} \leq 0$ on G . We also

define $E = \{\phi \in \bar{G} : \dot{V}(\phi) = 0\}$, and M is the largest set in E which is invariant with respect to (A.1).

The following result is the Lyapunov-LaSalle type theorem for (A.1).

Theorem A.2 (see [32, page 30]). *If V is a Lyapunov functional on G and $x_t(\phi)$ is a bounded solution of (A.1) that stays in G , then ω -limit set $\omega(\phi) \subset M$; that is, $x_t(\phi) \rightarrow M$ as $t \rightarrow +\infty$.*

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

- [1] K. L. Cooke, "Stability analysis for a vector disease model," *Rocky Mountain Journal of Mathematics*, vol. 9, no. 1, pp. 31–42, 1979.
- [2] H. W. Hethcote and P. van den Driessche, "An SIS epidemic model with variable population size and a delay," *Journal of Mathematical Biology*, vol. 34, no. 2, pp. 177–194, 1995.
- [3] J. Chin, *Control of Communicable Diseases Manual*, American Public Health Association, Washington, DC, USA, 1999.
- [4] S. W. Martin, *Livestock Disease Eradication: Evaluation of the Cooperative State-Federal Bovine Tuberculosis Eradication Program*, National Academy Press, Washington, DC, USA, 1994.
- [5] D. Tudor, "A deterministic model for herpes infections in human and animal populations," *SIAM Review*, vol. 32, no. 1, pp. 136–139, 1990.
- [6] K. E. VanLandingham, H. B. Marsteller, G. W. Ross, and F. G. Hayden, "Relapse of herpes simplex encephalitis after conventional acyclovir therapy," *Journal of the American Medical Association*, vol. 259, no. 7, pp. 1051–1053, 1988.
- [7] A. Bignami, "Concerning the pathogenesis of relapses in malarial fevers," *Southern Medical Journal*, vol. 6, pp. 79–89, 1913.
- [8] N. J. White, "Determinants of relapse periodicity in *Plasmodium vivax* malaria," *Malaria Journal*, vol. 10, article 297, 2011.
- [9] H. Cox, Y. Kebede, S. Allamuratova et al., "Tuberculosis recurrence and mortality after successful treatment: impact of drug resistance," *PLoS Medicine*, vol. 3, no. 10, pp. 1836–1843, 2006.
- [10] P. Van Den Driessche, L. I. N. Wang, and X. Zou, "Modeling diseases with latency and relapse," *Mathematical Biosciences and Engineering*, vol. 4, no. 2, pp. 205–219, 2007.
- [11] A. Kaddar, "On the dynamics of a delayed SIR epidemic model with a modified saturated incidence rate," *Electronic Journal of Differential Equations*, no. 133, pp. 1–7, 2009.
- [12] S. Gao, L. Chen, J. J. Nieto, and A. Torres, "Analysis of a delayed epidemic model with pulse vaccination and saturation incidence," *Vaccine*, vol. 24, no. 35–36, pp. 6037–6045, 2006.
- [13] J. A. Yorke and W. P. London, "Recurrent outbreaks of measles, chickenpox and mumps: II," *American Journal of Epidemiology*, vol. 98, no. 6, pp. 469–482, 1973.
- [14] M. G. M. Gomes, L. J. White, and G. F. Medley, "The reinfection threshold," *Journal of Theoretical Biology*, vol. 236, no. 1, pp. 111–113, 2005.
- [15] Y. Zhou and H. Liu, "Stability of periodic solutions for an SIS model with pulse vaccination," *Mathematical and Computer Modelling*, vol. 38, no. 3–4, pp. 299–308, 2003.
- [16] R. M. Anderson and R. M. May, "Regulation and stability of host-parasite population interactions: I. Regulatory processes," *The Journal of Animal Ecology*, vol. 47, no. 1, pp. 219–267, 1978.
- [17] F. Zhang and Z.-Z. Li, "Global stability of an SIR epidemic model with constant infectious period," *Applied Mathematics and Computation*, vol. 199, no. 1, pp. 285–291, 2008.
- [18] Z. Jiang and J. Wei, "Stability and bifurcation analysis in a delayed SIR model," *Chaos, Solitons and Fractals*, vol. 35, no. 3, pp. 609–619, 2008.
- [19] V. Capasso and G. Serio, "A generalization of the Kermack-McKendrick deterministic epidemic model," *Mathematical Biosciences*, vol. 42, no. 1–2, pp. 43–61, 1978.
- [20] R. Xu and Z. Ma, "Stability of a delayed SIRS epidemic model with a nonlinear incidence rate," *Chaos, Solitons and Fractals*, vol. 41, no. 5, pp. 2319–2325, 2009.
- [21] P. Georgescu and H. Zhang, "A Lyapunov functional for a SIRI model with nonlinear incidence of infection and relapse," *Applied Mathematics and Computation*, vol. 219, no. 16, pp. 8496–8507, 2013.
- [22] S. Liu, S. Wang, and L. Wang, "Global dynamics of delay epidemic models with nonlinear incidence rate and relapse," *Nonlinear Analysis: Real World Applications*, vol. 12, no. 1, pp. 119–127, 2011.
- [23] C. Castillo-Garsow, G. Jordan-Salivia, and A. Rodriguez Herrera, "Mathematical models for the dynamics of tobacco use, recovery, and relapse," Technical Report Series BU-1505-M, Cornell University, 2000.
- [24] A. Korobeinikov, "Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission," *Bulletin of Mathematical Biology*, vol. 68, no. 3, pp. 615–626, 2006.
- [25] H. N. Moreira and Y. Wang, "Global stability in an $S \rightarrow I \rightarrow R \rightarrow I$ model," *SIAM Review*, vol. 39, no. 3, pp. 496–502, 1997.
- [26] S. Blower, "Modelling the genital herpes epidemic," *Herpes*, vol. 11, supplement 3, pp. 138A–146A, 2004.
- [27] P. van den Driessche and X. Zou, "Modeling relapse in infectious diseases," *Mathematical Biosciences*, vol. 207, no. 1, pp. 89–103, 2007.
- [28] A. Abta, A. Kaddar, and H. T. Alaoui, "Global stability for delay SIR and SEIR epidemic models with saturated incidence rates," *Electronic Journal of Differential Equations*, vol. 2012, no. 23, pp. 1–13, 2012.
- [29] C. Vargas-De-Leon, "On the global stability of infectious diseases models with relapse," *Abstraction & Application*, vol. 9, pp. 50–61, 2013.
- [30] Z. Shuai and P. van den Driessche, "Global stability of infectious disease models using Lyapunov functions," *SIAM Journal on Applied Mathematics*, vol. 73, no. 4, pp. 1513–1532, 2013.
- [31] R. Xu, "Global dynamics of an SEIRI epidemiological model with time delay," *Applied Mathematics and Computation*, vol. 232, pp. 436–444, 2014.
- [32] Y. Kuang, *Differential Equations With Applications in Population Dynamics*, Academic Press, New York, NY, USA, 1993.