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Research Article

Stability and Bifurcation of a Computer Virus Propagation Model with Delay and Incomplete Antivirus Ability

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A new computer virus propagation model with delay and incomplete antivirus ability is formulated and its global dynamics is analyzed. The existence and stability of the equilibria are investigated by resorting to the threshold value R_0. By analysis, it is found that the model may undergo a Hopf bifurcation induced by the delay. Correspondingly, the critical value of the Hopf bifurcation is obtained. Using Lyapunov functional approach, it is proved that, under suitable conditions, the unique virus-free equilibrium is globally asymptotically stable if $R_0 < 1$, whereas the virus equilibrium is globally asymptotically stable if $R_0 > 1$. Numerical examples are presented to illustrate possible behavioral scenarios of the mode.

1. Introduction

With the rapid developments of information and communication technologies, computer has brought great convenience to our life. While enjoying the convenience from Internet, people have to confront the threat of virus intrusions. As the damaging programs, computer viruses parasitize themselves on a host mainly through the Internet and have also become an enormous threat to computers and network resources. So, understanding and predicting the dynamics of computer virus propagation are, therefore, an important pursuit. Consequently, a number of computer virus propagation models, ranging from conventional *SIR* compartment model [1–3] to its extensions [4–16], were proposed by borrowing from classical epidemic models to investigate the behaviors of computer virus propagation over network.

There is something strikingly different between computer viruses and biological viruses: computer viruses in latent status possess infectivity [17–19]. Consequently, recently proposed models can distinguish latent computers from infected ones by introducing the L and B compartments [17–19], named as the S(susceptible)-L(latent)-B(breaking-out)-S(susceptible) model, which represents the dynamics of virus by systems of ordinary differential equations.

One common feature shared by a computer virus is latency [20], which means that, when viruses enter in a host, they do not always immediately break out, but they hide themselves and only become active after a certain period. It is therefore easy to show that there is an inevitable delay from virus invasion to its outbreak. On the other hand, in real networks, the limited cost results in the incomplete antivirus ability. Indeed, when attempting to model computer virus propagation, some of characteristics of viruses and networks should be taken into consideration.

In this paper, a new computer virus propagation model, which incorporates simultaneously the above-mentioned aspects, is established. The aim is to extend and analyze the *SLBS* computer virus propagation model without delay and incomplete antivirus ability first proposed by Yang et al. [17–19]. This study is motivated by the fact that the delay plays a key role and is inevitably a complex impact on the investigation of computer virus spreading behaviors [21]. The incorporation of the delay and incomplete antivirus ability of networks makes the model more realistic but its mathematical qualitative analysis may be difficult. In our model, the existence and stability of the equilibria are investigated by resorting to the threshold value R_0, a certain condition. By analysis, it is found that the model may undergo

a Hopf bifurcation induced by the delay. Correspondingly, the critical value τ_0 of the Hopf bifurcation is obtained. When delay $\tau < \tau_0$, the virus spreading is stable and easy to protect; whereas $\tau > \tau_0$, the virus spreading is unstable and out of control. Applying Lyapunov functional approach, it is proven that the unique virus-free equilibrium is globally asymptotically stable under certain condition if $R_0 < 1$, whereas the virus equilibrium is globally asymptotically stable if $R_0 > 1$. Numerical examples are presented to demonstrate the analytical results and to illustrate possible behavioral scenarios of the mode. Our results may provide some understanding of the spreading behaviors of computer viruses.

The organization of this paper is as follows. In the next section, we present the mathematical model to be discussed. In Section 3, we study the existence and local and global stability of the virus-free and virus equilibria, respectively, and investigate the Hopf bifurcation. In Section 4, numerical examples are presented to demonstrate the analytical results. Finally, some conclusions are given in Section 5.

2. Mathematical Model

Consider the typical *SLBS* mode [17–19], which is formulated as the following system of ordinary differential equations:

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= \mu - \beta S(t) L(t) - \beta S(t) B(t) + \gamma B(t) - \mu S(t),\\ \frac{\mathrm{d}L}{\mathrm{d}t} &= \beta S(t) L(t) + \beta S(t) B(t) - \alpha L(t) - \mu L(t),\\ \frac{\mathrm{d}B}{\mathrm{d}t} &= \alpha L(t) - \gamma B(t) - \mu(t) B.\end{aligned} \tag{1}$$

Here, it is assumed that a computer (or node) is categorized as internal or external depending on whether or not it is currently connected to the network. The total number of computers connected to the network is divided into three compartments: internal uninfected compartment, (*i.e.*, virus-free computers), internal infected compartment where computers are currently latent (latent computers, for short), and internal infected compartment where computers are currently breaking out (breaking-out computers, for short). Let $S(t)$, $L(t)$, and $B(t)$ denote their corresponding percentages at time t, respectively. This model involves four positive parameters: μ denotes the rate at which external virus-free computers are connected to the network and at which an internal node is disconnected from the network; β denotes the rate at which, when having connection to one latent or breaking-out computer, one virus-free computer can become infected; γ denotes the rate at which a breaking-out computer gets a scan by running the antivirus software; α denotes the rate at which the latent computer is triggered.

By carefully considering the natures of computer virus, the following assumptions are made.

(i) At time t, the transition from the latent to the breaking-out is given by $L(t-\tau)$, which means a latent computer moves into the breaking-out compartment after a period of time τ.

(ii) Since the antivirus ability is incomplete, at time t, the breaking-out computers may either be temporarily suppressed in their latency with probability e or be cured into the virus-free ones with probability $(1-e)$, where $e > 0$ is a constant. If $e = 0$, then the antivirus ability is fully effective, whereas $e = 1$ means that antivirus ability is utterly ineffective.

Based on the assumptions above, one can obtain the following computer virus propagation model:

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= \mu - \beta S(t) L(t) - \beta S(t) B(t)\\ &\quad + (1-e)\gamma B(t) - \mu S(t),\\ \frac{\mathrm{d}L}{\mathrm{d}t} &= \beta S(t) L(t) + \beta S(t) B(t)\\ &\quad - \alpha L(t-\tau) + e\gamma B(t) - \mu L(t),\\ \frac{\mathrm{d}B}{\mathrm{d}t} &= \alpha L(t-\tau) - \gamma B(t) - \mu(t) B.\end{aligned} \tag{2}$$

Let $S(t) + L(t) + B(t) = 1$. Thus, model (2) can be written as the following:

$$\begin{aligned}\frac{\mathrm{d}L}{\mathrm{d}t} &= \beta [1 - L(t) - B(t)] [L(t) + B(t)]\\ &\quad - \alpha L(t-\tau) + e\gamma B(t) - \mu L(t),\\ \frac{\mathrm{d}B}{\mathrm{d}t} &= \alpha L(t-\tau) - \gamma B(t) - \mu B(t).\end{aligned} \tag{3}$$

All the parameters are positive constants. The initial conditions are

$$(\phi_1(\theta), \phi_2(\theta)) \in C_+ = \left((-\tau, 0], R_+^2\right), \quad \phi_i(\theta) > 0,\ i = 1, 2, \tag{4}$$

where $R_+^2 = \{(L, B) \in R^2, L \geq 0, B \geq 0\}$.

3. Model Analysis

In this section, we intended to study the dynamical behaviors of model (3). First, a threshold value R_0 is defined as the number of virus-free computers that are infected by a single computer virus during its lift span. The threshold value R_0 plays a key role in the epidemic dynamics. By resorting to it, the existence and stability of the equilibria can be determined. Generally speaking, if $R_0 < 1$, the virus-free equilibrium is globally asymptotically stable, and when $R_0 > 1$, the virus equilibrium exists and is globally asymptotically stable. A direct computation gives

$$R_0 = \frac{\beta(\alpha + \mu + \gamma)}{(\mu + \alpha)(\mu + \gamma) - \alpha e \gamma}. \tag{5}$$

3.1. Stability of Virus-Free Equilibrium. It is clear that model (3) always admits unique virus-free equilibrium $E_0(0, 0)$. We

first consider its stability. For model (3), the corresponding characteristic equation at E_0 is

$$\lambda^2 + m_1\lambda + m_0 + (\alpha\lambda + n_0)\, e^{-\lambda\tau} = 0, \tag{6}$$

which is similar to the relative forms in [22–24], where

$$\begin{aligned} m_1 &= 2\mu + \gamma - \beta, \\ m_0 &= (\mu - \beta)(\mu + \gamma), \\ n_0 &= [(\mu + \gamma) - (\beta + e\gamma)]\,\alpha. \end{aligned} \tag{7}$$

Our aim is to investigate the stability behavior in the case $\tau \neq 0$. Obviously, (6) is a transcendental equation, and $i\eta$ $(\eta > 0)$ is its root if and only if η satisfies

$$-\eta^2 + m_1 i\eta + m_0 = -(\alpha i\eta + n_0)(\cos\eta\tau - i\sin\eta\tau). \tag{8}$$

Separating the real and imaginary parts, we have

$$\begin{aligned} -\eta^2 + m_0 &= -n_0\cos\eta\tau - \alpha\eta\sin\eta\tau \\ m_1\eta &= -\alpha\eta\cos\eta\tau + n_0\sin\eta\tau. \end{aligned} \tag{9}$$

Eliminating τ by squaring and adding (9), we obtain a polynomial in η as

$$\eta^4 + \left(m_1^2 - \alpha^2 - 2m_0\right)\eta^2 + m_0^2 - n_0^2 = 0, \tag{10}$$

where

$$\begin{aligned} m_1^2 - 2m_0 - \alpha^2 &= [(\mu - \beta) + (\mu + \gamma)]^2 \\ &\quad - 2(\mu - \beta)(\mu + \gamma) - \alpha^2 \\ &= (\mu + \gamma)^2 + (\mu - \beta)^2 - \alpha^2, \\ m_0 + n_0 &= (\mu - \beta)(\mu + \gamma) + (\mu + \gamma)\alpha - (\beta + e\gamma)\alpha \\ &= (\mu + \gamma)(\mu + \alpha) - \alpha e\gamma - \beta(\mu + \gamma + \alpha) \\ &= [(\mu + \gamma)(\mu + \alpha) - \alpha e\gamma](1 - R_0) > 0, \\ m_0 - n_0 &= (\mu - \beta)(\mu + \gamma) - (\mu + \gamma)\alpha + (\beta + e\gamma)\alpha \\ &= (\mu - \beta - \alpha)(\mu + \gamma) + \alpha\beta + \alpha e\gamma, \\ m_0^2 - n_0^2 &= (m_0 + n_0)(m_0 - n_0) \\ &= \{[(\mu + \gamma)(\mu + \alpha) - \alpha e\gamma](1 - R_0)\} \\ &\quad \times \{(\mu - \beta - \alpha)(\mu + \gamma) + \alpha\beta + \alpha e\gamma\}. \end{aligned} \tag{11}$$

Clearly, if $\mu > \alpha + \beta$, then $m_1^2 - 2m_0 - \alpha^2 > 0$ and $m_0^2 - n_0^2 > 0$. It follows from the Hurwitz criterion that the roots of (10) have negative real parts. Hence, we have the following.

Theorem 1. *When $R_0 < 1$, the virus-free equilibrium E_0 is locally asymptotically stable for all $\tau > 0$ provided that $\mu > \alpha + \beta$.*

Now, it is the turn to examine the global stability of virus-free equilibrium. The following theorem is obtained.

Theorem 2. *When $R_0 < 1$, the virus-free equilibrium E_0 is globally asymptotically stable for all $\tau > 0$.*

Proof. By use of the Lyapunov Direct Method, consider the following function:

$$V(L, B) = L + B. \tag{12}$$

It is clear that V is a positive definite. Then the derivative of V is

$$\begin{aligned} \dot{V}(L, B) &= \frac{\mathrm{d}}{\mathrm{d}t}(1 - S) \\ &= -\mu + \beta S(L + B) - (1 - e)\gamma B + \mu S \\ &= \mu(S - 1) + \beta S(1 - S) - (1 - e)\gamma B \\ &= (S - 1)(\mu - \beta S) - (1 - e)\gamma B. \end{aligned} \tag{13}$$

If $\mu > \alpha + \beta$ hold, then $(S - 1)(\mu - \beta S) < 0$. Furthermore, $\dot{V}(L, B) < 0$ by the Lyapunov-LaSalle type theorem shows that $\lim_{t\to\infty} L(t) = 0$ and $\lim_{t\to\infty} B(t) = 0$. Hence, when $R_0 < 1$, the virus-free equilibrium E_* is globally asymptotically stable. □

3.2. Stability of Virus Equilibrium. Next, we examine the stability of virus equilibrium. After direct computations, the unique virus equilibrium $E_*(L_*, B_*)$ of model (3) reads

$$\begin{aligned} L_* &= \frac{(\gamma + \mu)B_*}{\alpha} = \frac{(\gamma + \mu)(R_0 - 1)}{R_0(\gamma + \mu + \alpha)}, \\ B_* &= \frac{\beta\alpha(\gamma + \mu + \alpha) - \alpha(\gamma + \mu)(\mu + \alpha) + \alpha^2 e\gamma}{\beta(\gamma + \mu + \alpha)^2} \\ &= \frac{\alpha(R_0 - 1)}{R_0(\gamma + \mu + \alpha)}. \end{aligned} \tag{14}$$

If $R_0 < 1$, the $E_*(L_*, B_*)$ does not exist. It suffices to show the local asymptotical stability of E_* for model (3). Indeed, the Jacobian matrix of the linearized system of this system evaluated at E_* is

$$\begin{pmatrix} \beta - 2\beta(L_* + B_*) - \mu - \alpha e^{-\lambda\tau} - \lambda & \beta - 2\beta(L_* + B_*) + e\gamma \\ \alpha e^{-\lambda\tau} & -(\mu + \gamma) - \lambda \end{pmatrix}. \tag{15}$$

Its characteristic equation is

$$\lambda^2 + p_1\lambda + p_0 + (\alpha\lambda + q_0)\, e^{-\lambda\tau} = 0, \tag{16}$$

where

$$\begin{aligned} p_1 &= 2\mu + \gamma - \beta + 2\beta(L_* + B_*), \\ p_0 &= (\mu - \beta)(\mu + \gamma) + 2\beta(\mu + \gamma)(L_* + B_*), \\ q_0 &= (\mu + \gamma)\alpha - [\beta + e\gamma - 2\beta(L_* + B_*)]\,\alpha. \end{aligned} \tag{17}$$

In the case $\tau \neq 0$, for our purpose, if $i\eta$ $(\eta > 0)$ is a solution of (16), separating real and imaginary parts, we derive that

$$-\eta^2 + p_1 i\eta + p_0 = -(\alpha i\eta + q_0)(\cos\eta\tau - i\sin\eta\tau). \tag{18}$$

Separating the real and imaginary parts yields

$$\begin{aligned} -\eta^2 + p_0 &= -q_0 \cos \eta\tau - \alpha\eta \sin \eta\tau, \\ p_1\eta &= -\alpha\eta \cos \eta\tau + q_0 \sin \eta\tau. \end{aligned} \tag{19}$$

Eliminating τ by squaring and adding (19), we obtain a polynomial in η as

$$\eta^4 + \left(p_1^2 - \alpha^2 - 2p_0\right)\eta^2 + p_0^2 - q_0^2 = 0. \tag{20}$$

For convenience, let $h_1 \triangleq p_1^2 - \alpha^2 - 2p_0$ and $h_2 \triangleq p_0^2 - q_0^2$. If $h_1 > 0$ and $h_2 > 0$, then both of the two roots of (20) have negative real parts. By the Hurwitz criterion, E_* is locally asymptotically stable for all $\tau > 0$. However, if $h_1 < 0$ and $h_2 > 0$, then (20) has the positive root $\eta_1 = (1/2)(\alpha^2 + 2p_0 - p_1^2 + \sqrt{(p_1^2 - \alpha^2 - 2p_0)^2 - 4(p_0^2 - q_0^2)})$. It follows that (16) has a positive root. Say, the characteristic equation (16) has a pair of imaginary roots $\pm i\eta_0$, and corresponding delay τ_0 is given by (19):

$$\tau_0 = \frac{1}{\eta}\arccos\left[\frac{\eta^2\left(1 - \alpha p_1\right) - p_0}{\eta^2\alpha^2 + q_0}\right] + \frac{2k\pi}{\eta}, \quad k = 0, 1, 2, 3, \ldots. \tag{21}$$

Furthermore, we can also verify the transversality condition $\mathrm{d}\Re((\lambda(\tau)))/\mathrm{d}\tau\|_{\tau=\tau_0} > 0$. Then, we establish the following theorem.

Theorem 3. *With $R_0 > 1$, model (3) has a unique virus equilibrium E_*. Furthermore,*

(1) *E_* is locally asymptotically stable when $\tau < \tau_0$ and is unstable when $\tau > \tau_0$, where*

$$\tau_0 = \frac{1}{\eta}\arccos\left[\frac{\eta^2\left(1 - \alpha p_1\right) - p_0}{\eta^2\alpha^2 + q_0}\right] + \frac{2k\pi}{\eta}, \quad k = 0, 1, 2, 3, \ldots; \tag{22}$$

(2) *when $\tau = \tau_0$, the model (3) undergoes a Hopf bifurcation.*

Now, we are ready to examine the global stability of virus equilibrium E_*. Then we have the following.

Theorem 4. *When $R_0 > 1$, the virus equilibrium E_* is globally asymptotically stable.*

Proof. We consider the following function:

$$V_1(L, B) = \frac{1}{2}[(L - L_*) + (B - B_*)]^2 + \frac{1}{2}\omega(L - L_*)^2, \tag{23}$$

where ω is a positive constant to be determined. The derivative of V_1 is

$$\begin{aligned} \dot{V}_1(L, B) &= \left(S - S_*\right)\frac{\mathrm{d}S}{\mathrm{d}t} + \omega\left(L - L_*\right)\frac{\mathrm{d}L}{\mathrm{d}t} \\ &= \left(S - S_*\right)\left[-\beta\left(S - S_*\right)\left(1 - S - S_*\right)\right. \\ &\qquad - (1 - e)\gamma\left(L - L_*\right) \\ &\qquad \left. - \left[(1 - e)\gamma + \mu\right]\left(S - S_*\right)\right] \\ &\quad + \omega\left(L - L_*\right)\left[\beta\left(S - S_*\right)\left(1 - S - S_*\right)\right. \\ &\qquad - \alpha\left(L\tau - L_*\right) - e\gamma\left(S - S_*\right) \\ &\qquad \left. - \left(e\gamma + \mu\right)\left(L - L_*\right)\right] \\ &= \left[\beta\left(S + S_* - 1\right) - (1 - e)\gamma - \mu\right] \\ &\quad \times \left(S - S_*\right)^2 - \omega\left(e\gamma + \mu\right)\left(L - L_*\right)^2 \\ &\quad - \omega\alpha\left(L - L_*\right)\left(L\tau - L_*\right) \\ &\quad + \left[\omega\beta\left(1 - S - S_*\right) - \omega e\gamma - (1 - e)\gamma\right] \\ &\quad \times \left(S - S_*\right)\left(L - L_*\right) \\ &= \beta\left(S - 1\right)\left(S - S_*\right)^2 + \left[\beta S_* - (1 - e)\gamma - \mu\right] \\ &\quad \times \left(S - S_*\right)^2 - \omega\left(e\gamma + \mu\right)\left(L - L_*\right)^2 \\ &\quad - \omega\alpha\left(L - L_*\right)\left(L\tau - L_*\right) \\ &\quad + \left[\omega\beta\left(1 - S - S_*\right) - \omega e\gamma - (1 - e)\gamma\right] \\ &\quad \times \left(S - S_*\right)\left(L - L_*\right) \\ &= \beta\left(S - 1\right)\left(S - S_*\right)^2 - \frac{(\mu + \gamma)(1 - e)\gamma}{\alpha + \mu + \gamma} \\ &\quad \times \left(S - S_*\right)^2 - \omega\left(e\gamma + \mu\right)\left(L - L_*\right)^2 \\ &\quad - \omega\alpha\left(L - L_*\right)\left(L\tau - L_*\right) \\ &\quad + \left[\omega\beta\left(1 - S - S_*\right) - \omega e\gamma - (1 - e)\gamma\right] \\ &\quad \times \left(S - S_*\right)\left(L - L_*\right) \\ &= -\left[\frac{(\mu + \gamma)(1 - e)\gamma}{\alpha + \mu + \gamma}(S_* - S)^2\right. \\ &\qquad + \omega\left(e\gamma + \mu\right)\left(L - L_*\right)^2 \\ &\qquad - \left[\omega\beta\left(S - 1\right) + \omega\beta S_* + \omega e\gamma + (1 - e)\gamma\right] \\ &\qquad \left. \times \left(S_* - S\right)\left(L - L_*\right)\right] \\ &\quad + \beta\left(S - 1\right)\left(S - S_*\right)^2 \\ &\quad - \omega\alpha\left(L - L_*\right)\left(L\tau - L_*\right). \end{aligned} \tag{24}$$

Let

$$\begin{aligned} \frac{(\mu + \gamma)(1 - e)\gamma}{\alpha + \mu + \gamma} \times \omega\left(e\gamma + \mu\right) &\geq 2\omega\beta\left(S - 1\right) \\ &+ 2\omega\beta S_* + 2\omega e\gamma + 2(1 - e)\gamma. \end{aligned} \tag{25}$$

That is,

$$\frac{(\mu + \gamma)(1 - e)\gamma}{\alpha + \mu + \gamma} \times \omega\left(e\gamma + \mu\right) - 2\omega\left(\beta S_* + e\gamma\right) \geq (1 - e)\gamma, \tag{26}$$

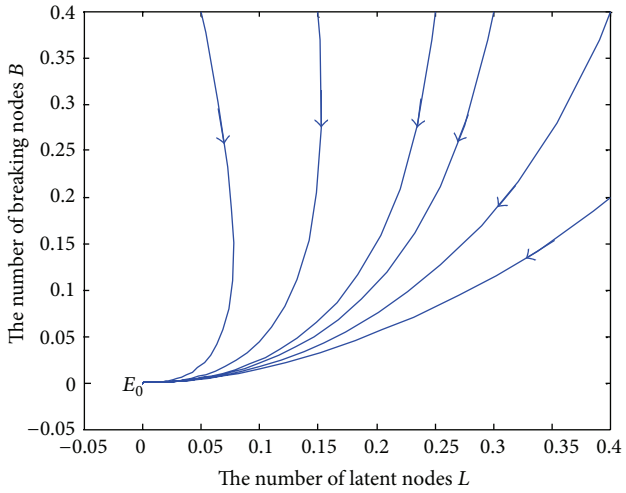

Figure 1: Phase diagram of $L(t)$ and $B(t)$ in the case $\beta = 0.02$, $\alpha = 0.23$, $\mu = 0.4$, $\gamma = 0.58$, $e = 0.35$, and $\tau = 1$ under the different values of $L(0)$ and $B(0)$.

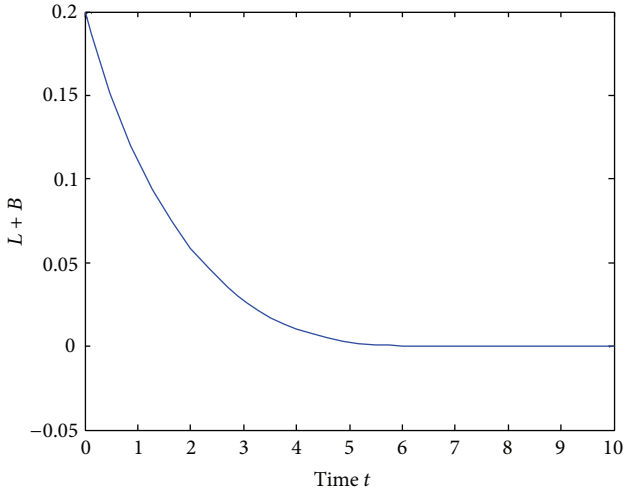

Figure 2: Evolutions of $L(t) + B(t)$ in the case $\beta = 0.05$, $\alpha = 0.3$, $\mu = 0.4$, $\gamma = 0.58$, $e = 0.18$, and $\tau = 2$ under the values of $L(0) = 0.1$ and $B(0) = 0.1$.

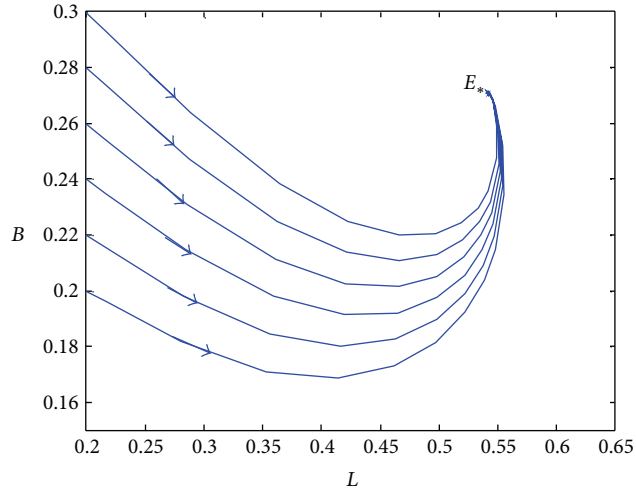

Figure 3: Phase diagram of $L(t)$ and $B(t)$ in the case $\beta = 0.4$, $\alpha = 0.1$, $\mu = 0.05$, $\gamma = 0.15$, $e = 0.5$, $\tau = 1$, $R_0 = 5.33 > 1$, and $E_* = (0.542, 0.271)$ under the different values of $L(0)$ and $B(0)$.

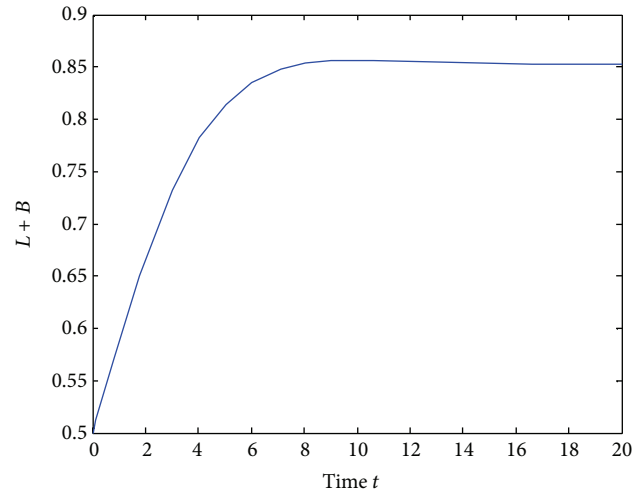

Figure 4: Evolutions of $L(t) + B(t)$ in the case $\beta = 0.56$, $\alpha = 0.15$, $\mu = 0.05$, $\gamma = 0.15$, $e = 0.5$, $\tau = 3$, and $R_0 = 6.817 > 1$ under the values of $L(0) = 0.3$ and $B(0) = 0.2$.

under the condition of $(\mu + \gamma)(1 - e)\lambda(e\gamma + \mu) - 2(\beta S_* + e\gamma)(\alpha + \mu + \gamma) > 0$, from which we can conclude that $\omega > (1-e)\gamma(\alpha+\mu+\gamma)/((\mu+\gamma)(1-e)\lambda(e\gamma+\mu)-2(\beta S_*+e\gamma)(\alpha+\mu+\gamma))$. In addition, suppose that τ is small enough, then $L\tau \approx L$. They lead to $V < 0$. Applying the Lyapunov-LaSalle type theorem, it shows that $\lim_{t\to\infty} L(t) = L_*$ and $\lim_{t\to\infty} B(t) = B_*$. Hence, when $R_0 > 1$, the virus equilibrium E_* is globally asymptotically stable. □

4. Numerical Simulations and Discussion

In this section, numerical simulations are carried out to support the analytical conclusion and to illustrate possible behavioral scenarios of the model. Figure 1 exhibits the evolutions of $L(t)$ and $B(t)$ with time, where the virus-free equilibrium is globally asymptotically stable, consistent with Theorem 2. Furthermore, an equilibrium is virus-free if and only if $L(t) + B(t) = 0$, which means that the virus would be extinct in the network, as shown in Figure 2. Figure 3 plots the evolutions of $L(t)$ and $B(t)$ with time. One can observe that, for any initial state, the solution would approach a fixed level; that is, the virus equilibrium is globally asymptotically stable. Besides, an equilibrium is viral if and only if $L(t) + B(t) \neq 0$, and its global asymptotical stability means that the virus spreads in the network continuously and stably, as shown in Figure 4. Figure 5 illustrates the complex impacts of delay τ on the spreading behavior of the virus. The evolutions of comparing $\tau < \tau_0$ with $\tau > \tau_0$ between $L(t)$ and $B(t)$ are carried out. It can be seen that, virus equilibrium E_* is stable when $\tau < \tau_0$and then when delay τ increases to the critical

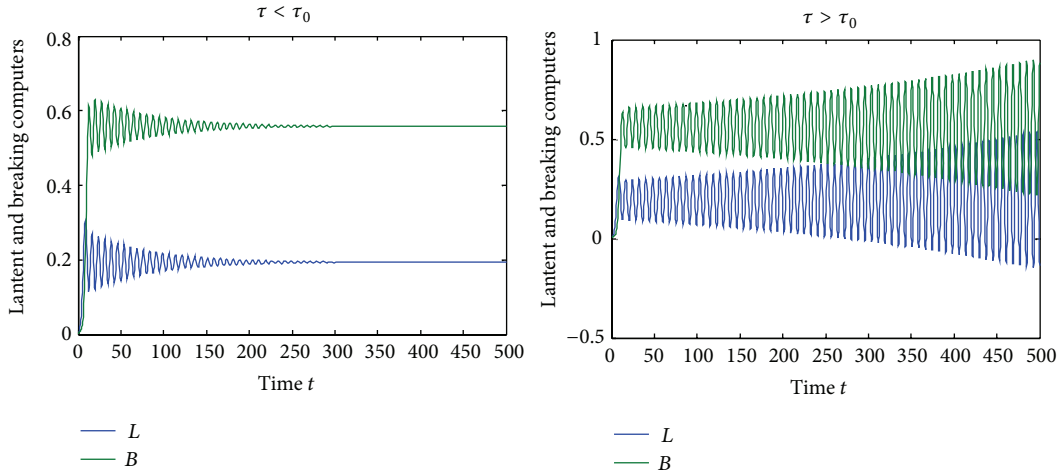

FIGURE 5: Evolutions of comparison of $\tau < \tau_0$ with $\tau < \tau_0$ between $L(t)$ and $B(t)$ in the case $\beta = 0.85$, $\alpha = 0.85$, $\mu = 0.2$, $\gamma = 0.095$, $e = 0.85$, $\tau = 2.3$ (left) and $\tau = 2.4$ (right), $R_0 = 4.05 > 1$, and $E_* = (0.193, 0.559)$ under the values of $L(0) = 0.006$ and $B(0) = 0.001$.

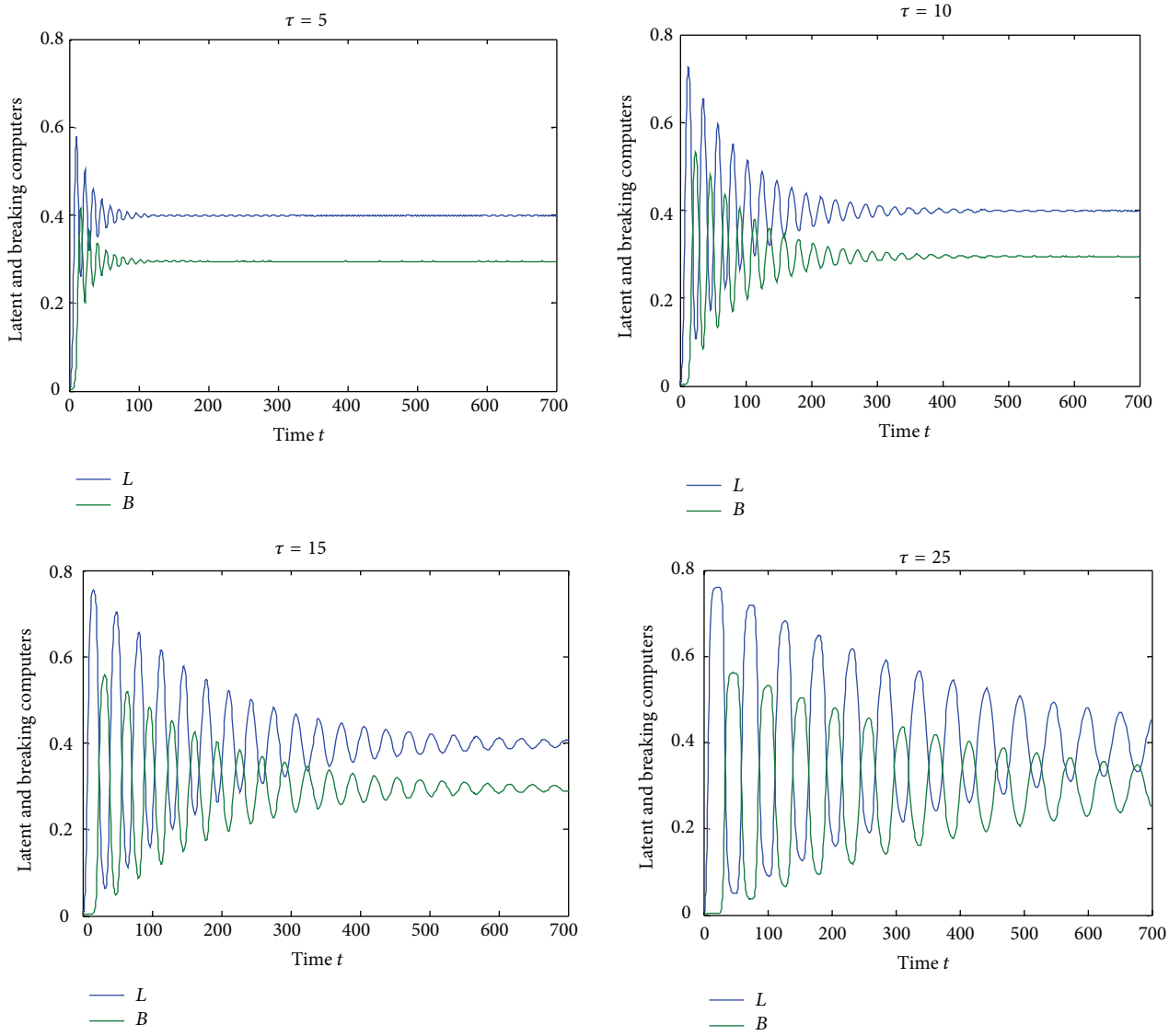

FIGURE 6: Evolutions of $L(t)$ and $B(t)$ with the different values of time delay τ in the case $\beta = 0.85$, $\alpha = 0.85$, $\mu = 0.2$, $\gamma = 0.95$, $e = 0.85$, $R_0 = 3.26 > 1$, and $E_* = (0.399, 0.295)$ under the values of $L(0) = 0.006$ and $B(0) = 0.001$.

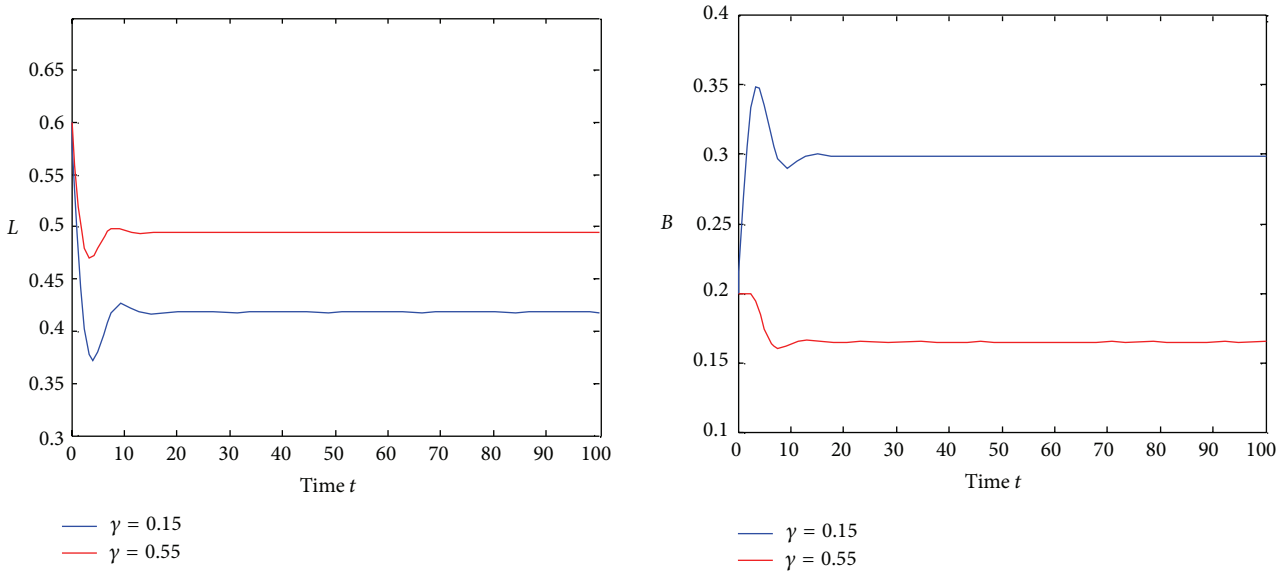

FIGURE 7: Evolutions of $L(t)$ and $B(t)$ with the different scan rates in the case $\beta = 0.85$, $\alpha = 0.25$, $\mu = 0.2$, $e = 0.15$, and $\tau = 2.5$ under the values of $L(0) = 0.6$ and $B(0) = 0.2$.

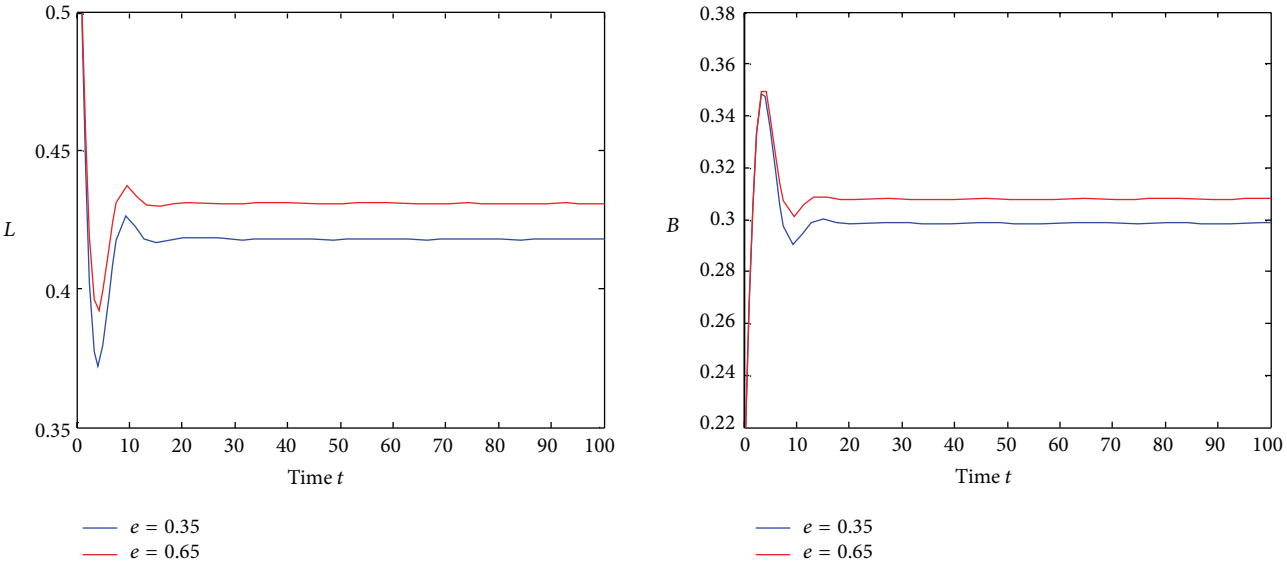

FIGURE 8: Evolutions of $L(t)$ and $B(t)$ with the different parameter e in the case $\beta = 0.85$, $\alpha = 0.25$, $\mu = 0.2$, $\gamma = 0.35$, and $\tau = 2.5$ under the values of $L(0) = 0.6$ and $B(0) = 0.2$.

value τ_0, it loses its stability and a Hopf bifurcation arises; then it exceeds the value of τ_0 beyond which the virus propagation will become unstable, in agreement with Theorem 3. In Figure 6, the effect of delay with $\tau \in (5, 10, 15, 25)$ on the number of latent and breaking-out computers is illustrated. The role of key parameters γ and e in the variation of the latent and breaking-out compartments is shown in Figures 7-8. As expected, one can observe that, for higher value of scan rates γ, the percentage of latent computers increases, in contrast to that of breaking-out ones. However, the percentages of both latent and breaking-out computers rise as e increases. Figure 9 shows the appearance of periodic solutions with the transmission from the stable state to the unstable one.

5. Conclusions

In real networks, the outbreak of computer virus usually lags and the antivirus ability of network is not fully complete. Aiming at characterizing these situations, a new computer virus propagation model is established. By theoretical analysis, the following conclusions can be obtained.

(1) If $R_0 < 1$ hold, the virus-free equilibrium E_0 is globally asymptotically stable under certain conditions for all $\tau > 0$, which implies that the virus would be extinct in the network. In such conditions, it is unnecessary for us to take practices in a real network. Say, the virus should be left alone.

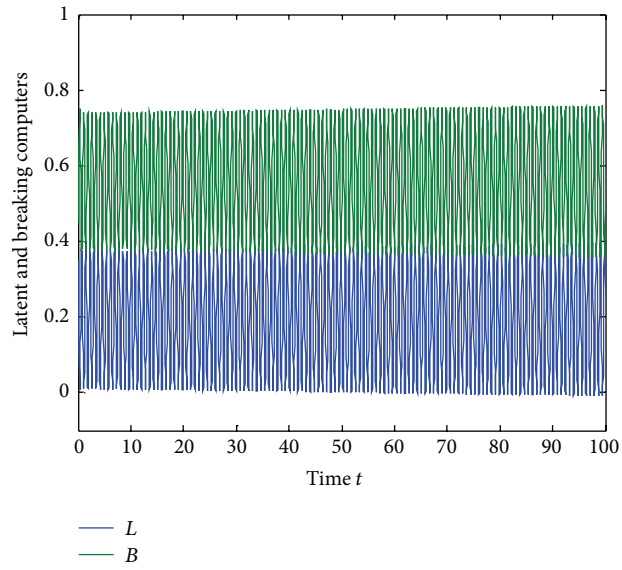

FIGURE 9: Appearance of periodic solutions in the case $\beta = 0.85$, $\alpha = 0.85$, $\mu = 0.2$, $\gamma = 0.095$, $e = 0.85$, $\tau = 2.375$, and $E_* = (0.193, 0.559)$ under the values of $L(0) = 0.35$ and $B(0) = 0.45$.

(2) If $R_0 > 1$ hold, the virus equilibrium E_0 is globally asymptotically stable, which means that the viruses spread in the network continuously and stably. In this case, some efforts can be made to keep the virus prevalence to below a proper level.

(3) The critical delay τ_0 where the Hopf bifurcation occurs is obtained, where

$$\tau_0 = \frac{1}{\eta}\arccos\left[\frac{\eta^2(1-\alpha p_1) - p_0}{\eta^2\alpha^2 + q_0}\right] + \frac{2k\pi}{\eta}, \quad k = 0, 1, 2, 3, \ldots. \tag{27}$$

(4) When the delay $\tau < \tau_0$, the virus propagation is stable. In such conditions, the spreading behavior of virus would be divinable.

(5) When the delay $\tau > \tau_0$, the virus propagation is unstable. In such conditions, the virus spreading would be out of control.

Moreover, numerical simulations are presented to demonstrate the analytical results and to illustrate possible behavioral scenarios of the model. It is shown that

(1) For virus equilibrium, the larger the delay is, the longer it takes to settle down towards its steady states.

(2) As expected, the increase of the scan rate can reduce the percentage of the breaking-out computers but increase the percentage of the latent ones, which suggests that we run the antivirus software as often as possible.

(3) As expected, the increase of the antivirus ability of the software can reduce the percentage of the infected (latent and breaking-out) computers in the network, which suggests that we invest more in their developments.

Our results may provide some understanding of the spreading behaviors of computer viruses.

Conflict of Interests

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

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