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On the global stability of an epidemic model of computer viruses

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Abstract In this paper, we study the global properties of a computer virus propagation model. It is, interesting to note that the classical method of Lyapunov functions combined with the Volterra–Lyapunov matrix properties, can lead to the proof of the endemic global stability of the dynamical model characterizing the spread of computer viruses over the Internet. The analysis and results presented in this paper make building blocks towards a comprehensive study and deeper understanding of the fundamental mechanism in computer virus propagation model. A numerical study of the model is also carried out to investigate the analytical results.

Keywords Dynamical models · Computer viral propagation · Global stability · Computer virus · Lyapunov function · Volterra–Lyapunov stability

Introduction

By development of computer technologies and network applications, the Internet has become a powerful mechanism for propagating computer virus. Because of this, computers connected to the Internet become much vulnerable to digital threats. Computer viruses, including the narrowly defined

Hassan Saberi Nik saberi_hssn@yahoo.com viruses and network worms, are loosely defined as malicious codes that can replicate themselves and spread among computers. In this scenario, the large number of existing computer viruses and their high level of destructivity appear as an important risk factor for corporations and individuals. Developing a mathematical model for the computer viral propagation is of critical importance not only for understanding better the behavior of computer virus but also for stopping the spread of the virus. These models lead to a better understanding and prediction of the scale and speed of computer virus propagation (Murray 1988; Yang and Yang 2015; Piqueira et al. 2008). Due to the high similarity between computer virus and biological virus, some classic epidemic models were established for computer virus propagation, such as the SIRS model (Han and Tan 2010; Ren et al. 2012; Bradley et al. 2008), the SEIR model (Yuan and Chen 2008), the SEIRS model (Mishra and Saini 2007), and the SEIQV model Wang et al. (2010), which share a common assumption that an infected computer in which the virus resides is in latency can not infect other computers (Yang et al. 2013; Li and Knickerbocker 2007).

The study of the endemic global stability is not only mathematically important, but also essential in predicting the evolution of the disease in the long run, so that prevention and intervention strategies can be effectively designed, and public health administrative efforts can be properly scaled. There are some methods, i.e., those based on the monotone dynamical systems (Li et al. 1999), and Lyapunov functions (Chong et al. 2014; Liu et al. 2015), to conduct global stability analysis for epidemic models (Xu and Ma 2010; Bhunu and Mushayabasa 2013; Imran et al. 2014). In addition, the method of Lyapunov functions has been known for many decades. The challenge in the application of this method is that there is no systematic way to construct Lyapunov functions (particularly, the determination of the appropriate coefficients

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is often a matter of luck), so that its success largely depends on trial and error as well as on specific problems. In this paper, we apply the method of Lyapunov functions combined with the Volterra-Lyapunov matrix properties which lead to the proof of the endemic global stability (Redheffer 1985a, b; Rinaldi 1990; Wang and Liao 2012; Chavez et al. 2002). In fact, we incorporate the Volterra-Lyapunov matrix theory (Rinaldi 1990) into Lyapunov functions, under certain conditions. which can leverage the difficulty of determining specific coefficient values, and, as such wider application of Lyapunov functions to dynamical systems could be promoted. Although the method of Lyapunov functions has been widely applied to various dynamical systems, the main contributions of our analysis is based on the less well-known results of Volterra-Lyapunov stable matrices. We are able to investigate more complex model systems with nonlinear incidence rates, with the aid of the Volterra-Lyapunov matrix properties.

Recently, Yang et al. (2013) proposed a computer virus propagation model, which incorporates the two features mentioned above. One major difficulty in studying the qualitative properties of this model lies in the construction of suitable Lyapunov functions. They used linear combinations of quadratic functions in independent variables as the candidate Lyapunov functions. Equipped with this tool, it is proved that the dynamic behavior of the model is determined by a threshold R_0 . Specifically, the virus-free equilibrium is globally asymptotically stable if $R_0 \leq 1$, whereas the viral equilibrium is locally asymptotically stable if $R_0 > 1$. In the present work, we employ the Volterra–Lyapunov metod, to investigate the endemic global stability for the computer virus propagation model (Yang et al. 2013).

We organize the remainder of this paper as follows. In section "Model formulation", we analyze the computer virus propagation model and investigate the mathematical analysis of this model. In section "Some notations and preliminaries", we investigate the global stability of the model. Finally, we close the paper by conclusions and discussion.

Model formulation

We shall study the mathematical model of computer virus proposed by Yang et al. (2013). A computer is classified as internal and external depending on weather it is connected to internet or not. In this model, assumes that only internal computers are concerned, and all internal computers are categorized into three classes: uninfected computers (i.e., virus-free computers), infected computers that are currently latent (latent computers, for short), and infected computers that are currently breaking out (seizing computers, for short). Due to the fact that in the future, the total amount of computers in the world would tend to saturation, it is reasonable to suppose that this total number is constant. Let S(t), L(t), and B(t) denote, at time t, the percentages of uninfected, latent, and seizing computers in all internal computers, respectively. Then, S(t) + L(t) + B(t) = 1. Unless otherwise stated, let *S*, *L*, and *B* stand for S(t), L(t), and B(t), respectively. By carefully considering the features of a computer virus, the following hypotheses are made:

(H1) External computers are connected to the Internet at positive constant rate δ , and internal computers are disconnected from the Internet also at this rate.

(H2) All newly connected computers are virus free.

(H3) The percentage of internal computers infected at time t increases by $\beta S(L+B)$, where β is a positive constant. This hypothesis says that both seizing and latent computers have infectivity. In contrast, all the traditional models assumed that only seizing individuals have infectivity, i.e., at time t the force of infection can be described as $\beta Sf(B)$ (Britten 2003).

(H4) Latent computers break out at positive constant rate α . (H5) Latent computers are cured at positive constant rate γ_1 , while breaking-out computers are cured at positive constant rate γ_2 . For the graded cure rates, we have $\gamma_2 > \gamma_1 > 0$.

Based on the previous assumptions, one can derive the following computer virus propagation model:

$$\frac{dS}{dt} = \delta - \beta S(L+B) + \gamma_1 L + \gamma_2 B - \delta S,$$

$$\frac{dL}{dt} = \beta S(L+B) - \gamma_1 L - \alpha L - \delta L,$$
(1)
$$\frac{dB}{dt} = \alpha L - \gamma_2 B - \delta B.$$

Remark 2.1 From the biological point of view, the spread of virus is similar to sexually disease, worm is similar to flu, and logic bomb is similar to HIV. Kephart used the epidemic models to find out the rule in the computer virus (Kephart and White 1991) and he focused his attention on the effect of topological structure of the network on the spread of the virus.

Now, we will discuss the existence of all possible equilibria of the model system (1). We found that system (1) has two possible non-negative equilibria, namely, the virus-free equilibrium E_0 and the viral equilibrium E_1 .

Virus-free equilibrium E_0 and the basic reproduction number R_0

In this subsection, we shall investigate the existence of equilibria of system (1). The virus-free equilibrium (VFE) is always feasible, as at this equilibrium the infection eradicates from the population. We calculate the basic reproduction number, R_0 , using the next generation approach, developed in Van den Driessche and Watmough (2002).

Let consider the non-negative orthant of \mathbb{R}^n by \mathbb{R}^n_+ . We let $x(t) \in \mathbb{R}^n_+$, where $x_i(t)$ denotes the number of individuals in compartment *i* at time *t*. For ease of notation, we order the compartments, such that the first *m* (for $m \le n$) compartments correspond to the states with infection. Note that the definition of a state with infection needs to be made from an understanding of the system being modeled and not from the infections themselves (and that this definition may not be unique). For the model

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = f_i(x),\tag{2}$$

with non-negative initial conditions, $x(0) \in \mathbb{R}^n_+$, we define X_s as the set of all disease-free states

$$X_s = \{ x \in \mathbb{R}^n_+ | x_i = 0, 1 \le i \le m \}.$$

We rewrite the model (2) as

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \mathcal{F}_i(x) - \mathcal{V}_i(x),\tag{3}$$

where $\mathcal{F}_i(x)$ is the rate of new infections entering compartment *i*, and

$$\mathcal{V}_i = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x),$$

where $\mathcal{V}_i^+(x)$ is the rate of transfer into compartment *i* by any other means, and $\mathcal{V}_i^-(x)$ is the rate of transfer out of compartment *i*. We list five reasonable assumptions for these functions below.

(A1) If $x \in \mathbb{R}^n_+$, then $\mathcal{F}_i(x)$, $\mathcal{V}_i^+(x)$, $\mathcal{V}_i^-(x) > 0$ for $1 \le i \le n$. This implies that no rate of movement can be negative.

(A2) If $x_i = 0$, then $\mathcal{V}_i^-(x) = 0$. If there are no individuals in a compartment, there can be no movement of individuals out of that compartment.

(A3) $\mathcal{F}_i(x) = 0$ for i > m. There can be no infections entering classes that are defined as noninfectious.

(A4) If $x \in X_s$, then $\mathcal{F}_i(x) = 0$ and $\mathcal{V}_i^+(x) = 0$ for $1 \le i \le m$. If there is no infection in the population, there can be no input into the infectious populations.

(A5) if F(x) is set to 0, then all eigenvalues of the corresponding Jacobian of (2) evaluated at a disease-free equilibrium point x_{dfe} , have negative real part. Note that the disease-free equilibrium point is not required to be unique.

For a disease-free equilibrium point, x_{dfe} , of (2), where x_{dfe} and f(x) satisfy assumptions (A1)–(A5), we define $m \times m$ matrices, *F* and *V*

$$F_{ij} = \frac{\partial \mathcal{F}_i}{\partial x_j} |_{x_{\text{dfe}}}, \quad 1 \le i, j \le m,$$
$$V_{ij} = \frac{\partial \mathcal{V}_i}{\partial x_i} |_{x_{\text{dfe}}}, \quad 1 \le i, j \le m.$$

For this model, the matrices F and V, for the new infection and the remaining transfer, are, respectively, given by

$$F = \begin{bmatrix} \beta & \beta \\ 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \gamma_1 + \alpha + \beta & 0 \\ -\alpha & \gamma_2 + \delta \end{bmatrix}.$$

Then, the basic reproduction number is defined, as the spectral radius of the next generation matrix, FV^{-1} . For this model, the basic reproduction number, which is defined as the number of previously uninfected computers that are infected by a single infected computer during its life cycle, can be derived as

$$R_0 = \frac{\beta(\gamma_2 + \alpha + \delta)}{(\gamma_2 + \delta)(\gamma_1 + \alpha + \delta)}.$$

Clearly, system (1) always has a virus-free equilibrium $E_0 = (1, 0, 0)$.

The endemic equilibrium

A positive equilibrium of (1), if one exists, is called an endemic equilibrium, and denoted by

$$E_1 = (S^*, L^*, B^*),$$

satisfy the following equilibrium equations:

$$\begin{split} \delta &-\beta S^{*}(L^{*}+B^{*})+\gamma_{1}L^{*}+\gamma_{2}B^{*}-\delta S^{*}=0,\\ \beta S^{*}(L^{*}+B^{*})-\gamma_{1}L^{*}-\alpha L^{*}-\delta L^{*}=0,\\ \alpha L^{*}-\gamma_{2}B^{*}-\delta B^{*}=0, \end{split}$$
(4)

where,

$$\begin{cases} S^* = \frac{1}{R_0} = \frac{(\gamma_2 + \delta)(\gamma_1 + \alpha + \delta)}{\beta(\gamma_2 + \alpha + \delta)}, \\ L^* = \frac{(R_0 - 1)(\gamma_2 + \delta)}{R_0(\gamma_2 + \alpha + \delta)}, \\ B^* = \frac{\alpha(R_0 - 1)}{R_0(\gamma_2 + \alpha + \delta)}. \end{cases}$$

The authors in Yang et al. (2013) answered the basic questions of the stability of the DFE and the local dynamics of the endemic equilibrium, for the system (1). The global stability of the endemic equilibrium for this model, however, is much more difficult to analyze. Below, we will combine the method of Lyapunov functions and Volterra–Lyapunov stable matrices to address this challenge.

Some notations and preliminaries

Our goal here is to show that the endemic equilibrium is globally asymptotically stable. First, we introduce necessary concepts and notations that will facilitate our global stability analysis.

Notation 3.1 We write a matrix A > 0(<0) if A is symmetric positive (negative) definite. The following fundamental result on matrix stability was originally proved by Lyapunov:

Lemma 3.2 (Cross 1979). Let A be an $n \times n$ real matrix. Then, all the eigenvalues of A have negative (positive) real parts if and only if there exists a matrix H > 0, such that $HA + A^T H^T < 0(>0)$.

Definition 3.3 We say a nonsingular $n \times n$ matrix A is Volterra–Lyapunov stable if there exists a positive diagonal $n \times n$ matrix M, such that $MA + A^TM^T < 0$.

The following lemma determines all 2×2 Volterra–Lyapunov stable matrices.

Lemma 3.4 (Rinaldi 1990; Cross 1979). Let $D = \begin{bmatrix} d_{11} & d_{12} \\ d_{21} & d_{22} \end{bmatrix}$ be a 2 × 2 matrix. Then D is Volterra– Lyapunov stable if and only if $d_{11} < 0, d_{22} < 0$, and $det(D) = d_{11}d_{22} - d_{12}d_{21} > 0$.

The characterization of Volterra–Lyapunov stable matrices of higher dimensions, however, is much more difficult. We need the following definition.

Definition 3.5 We say a nonsingular $n \times n$ matrix A is diagonally stable (or positive stable) if there exists a positive diagonal $n \times n$ matrix M, such that $MA + A^TM^T > 0$.

From Definitions 3.3 and 3.5, it is clear that a matrix A is Volterra–Lyapunov stable if and only if its negative matrix, -A, is diagonally stable.

Notation 3.6 For any $n \times n$ matrix A, let \widetilde{A} denote the $(n-1) \times (n-1)$ matrix obtained from A by deleting its last row and last column.

The following generalized result was obtained by Redheffer (1985a, b) which will be frequently used in our global stability analysis. For simplicity, we only state the sufficient condition below.

Lemma 3.7 Redheffer (1985a, b). Let $D = [d_{ij}]$ be a nonsingular $n \times n$ matrix $(n \ge 2)$ and $M = diag(m_1, \ldots, m_n)$ be a positive diagonal $n \times n$ matrix. Let $E = D^{-1}$. Then, if $d_{nn} > 0$, $\widetilde{ME} + (\widetilde{ME})^T > 0$, and

 $\widetilde{M}\widetilde{D} + (\widetilde{M}\widetilde{D})^T > 0$, it is possible to choose $m_n > 0$, such that $MD + D^T M^T > 0$.

Lemma 3.8 (Chavez et al. 2002). Consider a disease model system written in the form:

$$\frac{dX_1}{dt} = F(X_1, X_2),
\frac{dX_2}{dt} = G(X_1, X_2),
G(X_1, 0) = 0,$$
(5)

where $X_1 \in \mathbb{R}^m$ denotes (by its components) the uninfected populations and $X_2 \in \mathbb{R}^n$ denotes (by its components) the infectious populations; $X_0 = (X_1^E, 0)$ denotes the diseasefree equilibrium of the system.

In addition, assume the conditions (C1) and (C2) below:

- C_1 : For $\frac{dX_1}{dt} = F(X_1, 0), X_1^E$ is globally asymptotically stable;
- $C_2: G(X_1, X_2) = AX_2 \hat{G}(X_1, X_2)$, with $\hat{G}(X_1, X_2) \ge 0$ for $(X_1, X_2) \in \Omega$, where the Jacobian matrix $A = \frac{\partial G}{\partial X_2}(X_1^E, 0)$ has all non-negative off-diagonal elements, and X is the region where the model makes biological sense.

Then, the DFE $X_0 = (X_1^E, 0)$ is globally asymptotically stable provided that $R_0 < 1$.

Global stability of disease-free equilibrium

We will use the theorem by Castillo-Chavez et al. (2002), to prove the global stability result.

Theorem 3.9 The fixed point $E_0 = (1,0,0)$ is a globally asymptotically stable equilibrium of system (1) provided that $R_0 < 1$ and the assumptions in Eq. (5) are satisfied.

Proof Applying Lemma 3.8 to system (1), consider $X_1 = S$, $X_2 = \begin{bmatrix} L \\ B \end{bmatrix}$.

When L = B = 0, the uninfected subsystem (i.e., the equation for *S*) becomes

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \delta - \delta S,\tag{6}$$

which has the solution

$$S(t) = 1 + e^{-\delta t} (S(0) - 1),$$
(7)

obviously, $S(t) \rightarrow 1$ as $t \rightarrow \infty$ regardless of the initial value S(0). Therefore, it shows that condition (C_1) in Lemma 3.8 holds for our model.

Next, the right-hand side of the infectious subsystem (i.e., the equations for L and B) can be written as

$$\frac{\mathrm{d}X_2}{\mathrm{d}t} = G(X_1, X_2) = \begin{bmatrix} -(\beta S - \gamma_1 - \alpha - \delta)L + \beta SB \\ \alpha L - (\gamma_2 + \delta)B \end{bmatrix}$$
$$= \begin{bmatrix} \beta(-\gamma_1 - \alpha - \delta) & \beta \\ \alpha & -(\gamma_2 + \delta) \end{bmatrix} \begin{bmatrix} L \\ B \end{bmatrix}$$
$$- \begin{bmatrix} -\beta SL + \beta L - \beta SB + \beta B \\ 0 \end{bmatrix}$$
$$= AX_2 - \hat{G}(X_1, X_2),$$

where

$$A = \begin{bmatrix} \beta(-\gamma_1 - \alpha - \delta) & \beta \\ \alpha & -(\gamma_2 + \delta) \end{bmatrix}$$

and

$$\hat{G}(X_1, X_2) = \begin{bmatrix} \beta L(1-S) + \beta B(1-S) \\ 0 \end{bmatrix}$$

It is obvious that, $S \le 1$, hence, it is clear that condition (C_2) holds for our model. We also notice that the matrix *A* is an M matrix, since all its off-diagonal elements are non-negative. Hence, this proves the global stability of the DFE (E_0) .

Global stability of the endemic equilibrium of the model system (1)

Our goal here is to show that the endemic equilibrium of the model system (1) is globally asymptotically stable. It is, however, interesting to note that the classical method of Lyapunov functions combined with the Volterra–Lyapunov matrix properties Redheffer (1985a, b) can lead to the proof of the endemic global stability. The details are provided below.

To prove global stability result, we propose the following Lyapunov function:

$$V = w_1 (S - S^*)^2 + w_2 (L - L^*)^2 + w_3 (B - B^*)^2,$$
(8)

where w_1 , w_2 , and w_3 are positive constants. Calculating the time derivative of *V* along the trajectories of the system (1), we obtain

$$\begin{split} \dot{V} &= 2w_1(S - S^*)\dot{S} + 2w_2(L - L^*)\dot{L} + 2w_3(B - B^*)\dot{B} \\ &= 2w_1(S - S^*)[-\beta S(L + B) + \beta S^*(L^* + B^*) \\ &+ \gamma_1(L - L^*) + \gamma_2(B - B^*) - \delta(S - S^*)] \\ &+ 2w_2(L - L^*)[\beta S(L + B) - \beta S^*(L^* + B^*) \\ &- \gamma_1(L - L^*) - \alpha(L - L^*) - \delta(L - L^*)] \\ &+ 2w_3(B - B^*)[\alpha(L - L^*) - \gamma_2(B - B^*) - \delta(B - B^*)] \end{split}$$

Then, we add the expression βS^*L and βS^*B into the first and second square bracket. As a result, we obtain

$$\begin{split} \dot{V} &= 2w_1(S - S^*)[-\beta SL - \beta SB + \beta S^*L^* + \beta S^*B^* + \beta S^*L \\ &- \beta S^*L + \beta S^*B - \beta S^*B + \gamma_1(L - L^*) + \gamma_2(B - B^*) \\ &- \delta(S - S^*)] + 2w_2(L - L^*)[\beta SL + \beta SB - \beta S^*L^* \\ &- \beta S^*B^* + \beta S^*L - \beta S^*L + \beta S^*B - \beta S^*B - \gamma_1(L - L^*) \\ &- \alpha(L - L^*) - \delta(L - L^*)] + 2w_3(B - B^*)[\alpha(L - L^*) \\ &- \gamma_2(B - B^*) - \delta(B - B^*)], \end{split}$$

therefore, we have

$$\begin{split} \dot{V} &= 2w_1(S-S^*)[(-\beta L - \beta B - \delta)(S-S^*) + (-\beta S^* + \gamma_1) \\ &(L-L^*) + (-\beta S^* + \gamma_2)(B-B^*)] + 2w_2(L-L^*) \\ &[(\beta L + \beta B)(S-S^*) + (\beta S^* - \gamma_1 - \alpha - \delta)(L-L^*) \\ &+ \beta S^*(B-B^*)] + 2w_3(B-B^*)[\alpha(L-L^*) - (\gamma_2 + \delta) \\ &(B-B^*)] = 2w_1(-\beta L - \beta B)(S-S^*)^2 \\ &+ 2w_1(-\beta S^* + \gamma_1)(S-S^*)(L-L^*) \\ &+ 2w_1(-\beta S^* + \gamma_2)(S-S^*)(B-B^*)] + 2w_2(\beta L + \beta B) \\ &(L-L^*)(S-S^*) + 2w_2(\beta S^* - \gamma_1 - \alpha - \delta)(L-L^*)^2 \\ &+ 2w_2\beta S^*(L-L^*)(B-B^*) + 2w_3\alpha(L-L^*)(B-B^*) \\ &- 2w_3(\gamma_2 + \delta)(B-B^*)^2 = Y(WP + P^TW^T)Y^T, \end{split}$$

where $Y = [S - S^*, L - L^*, B - B^*], W = diag(w_1, w_2, w_3),$ and

$$P = \begin{bmatrix} -\beta L - \beta B - \delta & -\beta S^* + \gamma_1 & -\beta S^* + \gamma_2 \\ \beta L + \beta B & \beta S^* - \gamma_1 - \alpha - \delta & \beta S^* \\ 0 & \alpha & -\gamma_2 - \alpha \end{bmatrix}.$$
(10)

To discuss the global asymptotic stability of $E_1 = (S^*, L^*, B)$, we proceed to show that the matrix *P* defined in Eq. (10) is Volterra–Lyapunov stable or -P is diagonal stable. For this goal, we prove the following lemmas.

Lemma 3.10 For the matrix P defined in Eq. (10), -P, is diagonal stable.

Proof To prove the diagonal stability of -P and based on Lemma 3.7, we need to show that the following three conditions are satisfied:

Condition 1. We show that the matrix $D = -\tilde{P}$ is diagonal stable. From Eq. (10), we obtain

$$D = -\tilde{P} = \begin{bmatrix} \beta L + \beta B + \delta & \beta S^* - \gamma_1 \\ \\ -\beta L - \beta B & -\beta S^* + \gamma_1 + \alpha + \delta \end{bmatrix}$$

For this purpose, it is necessary to show that -D is Volterra–Lyapunov stable:

$$-D = \begin{bmatrix} -\beta L - \beta B - \delta & -\beta S^* + \gamma_1 \\ \beta L + \beta B & \beta S^* - \gamma_1 - \alpha - \delta \end{bmatrix}.$$

Clearly, $-D_{11} < 0$. Next, we show $-D_{22} < 0$; according to (4), we have

$$\beta S^*(L^*+B^*)=(\gamma_1+\alpha+\delta)L^*,$$

and it is obvious that

 $\beta S^*L^* \leq (\gamma_1 + \alpha + \delta)L^*,$

hence, $-D_{22} < 0$. Now, we show $-D_{12} < 0$, that is

$$-\beta S^* + \gamma_1 < 0,$$

using (4), (5), we can see that

$$\beta S^*(L^*+B^*)-(\gamma_1+\delta)L^*-(\gamma_2+\delta)B^*=0,$$

since $0 < \gamma_1 < \gamma_2$, we have

$$\begin{split} \beta S^*(L^*+B^*) &- (\gamma_1+\delta)L^* - (\gamma_2+\delta)B^* < \beta S^*(L^*+B^*) \\ &- (\gamma_1+\delta)L^* - (\gamma_1+\delta)B^*, \end{split}$$

therefore

$$\beta S^*(L^* + B^*) > (\gamma_1 + \delta)(L^* + B^*),$$

hence, $-D_{12} < 0$. It is easy to see $-D_{21} > 0$. Therefore, -D is Volterra–Lyapunov stable based on Lemma 3.4.

Condition 2. We show that the matrix $E = -P^{-1}$ is diagonal stable. In fact, we show that -E is Volterra-Lyapunov stable:

$$-E = -(-\widetilde{P^{-1}}) = \frac{1}{det(-P)} \begin{bmatrix} -E_{11} & -E_{12} \\ -E_{21} & -E_{22} \end{bmatrix},$$

where

$$-E_{11} = -(\gamma_2 + \delta)(-\beta S^* + \gamma_1 + \alpha + \delta) + \alpha\beta S^*$$

$$-E_{12} = -(\gamma_2 + \delta)(-\beta S^* + \gamma_1) + \alpha(\beta S^* - \gamma_2),$$

$$-E_{21} = -(\beta L + \beta B)(\gamma_2 + \delta),$$

$$-E_{22} = -(\beta L + \beta B + \delta)(\gamma_2 + \delta).$$

It is obvious that $-E_{21} < 0$ and $-E_{22} < 0$. Below, we show $-E_{11} = 0$ and $-E_{12} > 0$.

The (1, 1) entrie of this -E is written as

$$-E_{11} = -(\gamma_2 + \delta)(-\beta S^* + \gamma_1 + \alpha + \delta) + \alpha \beta S^*,$$

multiplying the (4) by α , and using (5) we have

$$\beta S^* \alpha L^* + \beta S^* \alpha B^* - (\gamma_1 + \alpha + \delta) \alpha L^* = 0.$$
⁽¹¹⁾

Therefore

$$\beta S^* (\gamma_2 + \delta) B^* + \beta S^* \alpha B^* - (\gamma_1 + \alpha + \delta) (\gamma_2 + \delta) B^* = 0,$$
(12)

from where

$$\beta S^*(\gamma_2 + \alpha + \delta) = (\gamma_1 + \alpha + \delta)(\gamma_2 + \delta), \tag{13}$$





hence, $-E_{11} = 0$.

It is easy to see det(-E) > 0, see the Appendix of this paper. Therefore, -E is Volterra–Lyapunov stable based on Lemma 3.4.



Fig. 3 Phase plane portraits of *L* vs. *S* for system (1). The basic reproduction number is $R_0 = 1.4$. The five curves correspond to different initial conditions with L(0) = 0.1, 0.3, 0.5, 0.7, 0.9, respectively

Condition 3. It is obvious that $-P_{33} > 0$.

Hence, Lemma 3.7 guarantees that -P is diagonal stable.







Theorem 3.11 The matrix P defined in Eq. (10) is Volterra–Lyapunov stable.

Proof Based on Lemmas 3.7 and 3.10, there exists a positive diagonal matrix W, such that $W(-P) + (-P)^T W^T > 0$. Thus $WP + P^T W^T < 0$.

Theorem 3.12 The endemic equilibrium, $E_1 = (S^*, L^*, B^*)$, of model (1) is globally asymptotically stable.

Proof Based on Lemmas 3.7 and 3.10 and Theorem 3.12, we obtain $\frac{dV}{dt} < 0$ when $X \neq X^*$ and X is not on the S-axis (a set of measure zero). It implies that the endemic equilibrium of the model system (1) is globally asymptotically stable.

Numerical results

In this subsection, we carry out numerical simulations and discuss results. Consider system (1) with $\alpha = 0.6$, $\beta = 0.3$, $\delta = 0.1$, $\gamma_1 = 0.1$, $\gamma_2 = 0.3$. We plot the phase plane portrait of *L* vs. *S* and *B* vs. *S* in Figs. 1, 2 for $R_0 = 0.9375$, a typical case of $R_0 < 1$, where the *DFE* is globally asymptotically stable. This is evidenced in these figures by the fact that all the five orbits converge to the *DFE* at S = 1 and L = B = 0.

In addition, consider system (1) with $\alpha = 0.3$, $\beta = 0.4$, $\delta = 0.1$, $\gamma_1 = 0.1$, $\gamma_2 = 0.3$. Then, $R_0 = 1.4$ in this case, and the unique positive endemic equilibrium is located at $S^* = 0.71$, $L^* = 0.1$ and $B^* = 0.1$. We pick

five different initial conditions, and plot these five solution curves by the phase plane portrait of L vs. S and B vs. S in Figs. 3, 4. From which one can see all these five orbits converge to the endemic equilibrium, showing the global asymptotic stability of the endemic equilibrium.

Remark 3.13 The method considered in this paper is conceptually simple to implement, and can be applied to different models with nonlinear incidence rates. As can be expected, the implementation of this method will likely be hindered for epidemiological models with more complex incidence rates, or those with even higher dimensions, though such difficulty remains the same for all other existing methods in global stability analysis. As far as the current method is concerned, some symbolic computation software can be possibly used to leverage some of the algebraic difficulty.

Conclusion

We study a dynamical model characterizing the spread of computer viruses over the Internet. It is assumed that all infected computers possess infectivity, and latent computers have a lower cure rate than seizing computers. As we know, a computer user might try to clear viruses spontaneously even if he is not sure that viruses are staying in his computer possibly because:

- he is accustomed to running antivirus program regularly, or
- 2. he is informed that viruses are spreading over the Internet.

In this paper, the global stability of a computer virus propagation model, which incorporates the two features mentioned above, is investigated. One major difficulty in studying the qualitative properties of this model lies in the construction of suitable Lyapunov functions, so that its success largely depends on trial and error as well as on specific problems. By combining this classical approach with the Volterra-Lyapunov matrix analysis, we have leveraged the difficulty of determining specific coefficient values, and as such, wider application of Lyapunov functions to dynamical systems could be promoted. The method Volterra-Lyapunov stability in this work is applied for a model of a computer virus propagation model. The analytical expressions of the stability analysis are provided and their numerical implementation is discussed.

Most of us thought that the problem about propagation of computer viruses should be discussed on network; thus, the diffusion can be considered as well (Ma et al. 2016; Song et al. 2016; Yang and Yang 2017; Satorras et al. 2015; Yang et al. 2017). Within our proposed oscillatorlike epidemic model, the spatial effect is homogenized and considered using the variables using mean field theory. In the view of network, for example, regular network with nearest-neighbor connection, small-world type, each node will suffer from external forcing and stimuli from other nodes, which can be regarded as external stimuli with diversity. The local kinetics is critical for collective behaviors of the network. Our results in this model could be helpful for further discussion on collective transition of safety and propagation of computer viruses. In addition, we wish this problem can be further discussed on network in the future.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interests regarding the publication of this article.

Appendix

Proof of
$$det(-P) > 0$$
:
 $det(-P) = (\beta L + \beta B + \delta)[(\gamma_2 + \delta)(-\beta S^* + \gamma_1 + \alpha + \delta) - \alpha\beta S^*] + (\beta L + \beta B)[(\gamma_2 + \delta)(\beta S^* - \gamma_1) + \alpha(\beta S^* - \gamma_2)].$

Since $\widetilde{P^{-1}}_{11} = 0$, and $\widetilde{P^{-1}}_{12} < 0$, we have det(-P) > 0.

Hence, it is clear to see det(-E) > 0. The proof is then complete.

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