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ANALYSIS OF DYNAMICAL BEHAVIORS FOR A COMPUTER VIRUS SPREADING MODEL

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Abstract: A spreading model of computer virus on the Internet is presented in terms of the fact that computer virus process includes two stages: free spread stage and response stage. It is proven that the infected nodes are always increasing in the free spread stage. In response stage, it is proven that the viruses will all be cleared when the basic reproductive number is less than unity; otherwise, the relative density of infected nodes will tend to a positive constant. Numerical simulations are carried out to illustrate the main theoretical results.

AMS Subject Classification: 97M50, 97P70, 34D23, 34D05 Key Words: spreading model, scale-free network, the basic reproductive number, global attractivity

1. Introduction

Malicious computer viruses are defined as program codes that are intended to spread among computers and perform detrimental operations, see [19]. The computer viruses are major threats to the Internet security and privacy. Due to the fact that the development of antivirus software always lags behind the appearance of new viruses, it is urgent to macroscopically understand the way that computer viruses spread and to work out effective defense measures. Therefore many researchers are addressing questions linked to virus spreading models,

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dynamics and control strategies. Noting the appealing analogy between computer viruses and epidemic in the biological world, Cohen [8] and Murray [25] inventively suggested to exploit the tools developed in epidemic dynamics of infectious diseases to study the spreading behavior of computer viruses. From then on, some virus models on the Internet, which is regarded as homogeneous networks, have been proposed [12, 13, 26].

Since the modelling of the seminal works on the scale-free network, in which the probability of p(k) for any node with k links to other nodes is distributed according to the power law $p(k) = Ck^{-\gamma}$ (2 < γ < 3), suggested by Barabási and Albert [1], the interest in study of computer viruses on the Internet is enhanced by the evidence that the Internet exhibit scale-free topological properties, and some interesting and novel results were also obtained, for example, a virus process in an infinite-sized scale-free network do not possess any threshold [20, 21]. Up to now, many epidemic models on scale-free networks have been presented: SI models [23, 17], SIS models [20, 21, 2, 14, 5, 3, 6, 9], SIR models [5, 28, 18], SIRS models [7], SIQRS models [24], and SIERS model [15, 10], and so on. There is a unique positive endemic equilibrium when the basic reproductive number is more than unity, the fact is proved in these papers above. On the other hand, numerical simulations show the positive endemic equilibrium is globally attractive (this means the virus will persist on a positive steady level), but the mathematical proof of the global attractivity of the endemic equilibrium is difficult and only a few works are presented [16, 7, 10].

Considering the spread of computer viruses often experiences several stages, Wang and Liu discuss the dynamics of a kind of piecewise model in a homogeneous network in [22]. In this paper, we will extend these works to ones on the scale-free networks.

The rest of this paper is organized as following. The piecewise model is presented in Section 2. The properties of the model in the free spread stage and response stage are revealed in Section 3. Numerical simulations are given to demonstrate the main results in Section 4. The paper ends with a conclusion in Section 5.

2. Model formulation

Suppose the size of the network is a constant N during the period of virus spreading, we also suppose that the degree of each code is time invariant. Let $S_k(t), I_k(t)$ and $R_k(t)$ be the relative density of susceptible nodes, infected nodes and recovered nodes of connectivity k at time t, respectively, where $k = 1, 2, \dots, n$ and n is the maximum number of contact each node. In real world, a virus always occurs on a finite networks. The maximum connectivity

n of any node is related to the network age, measured as the number of nodes N [11]:

$$n = mN^{1/(\gamma - 1)}$$

in which m is the minimum connectivity of the network.

The spread of computer virus process on a networks is divided into two typical stages: free spread stage and response stage, and let the dividing point of the two phases be t_0 .

In free spread stage, the virus in the beginning of the free dissemination stage will need some time to attract the attention of people. Even if the antivirus mechanism to get the virus information, it also will require some time to develop anti-virus strategy, before that the infected node can not be treated into health state or removed state. Terminals only have two states: the susceptible and the infected. The dynamical equations for the density $S_k(t)$, $I_k(t)$, at the mean-field level, satisfy the following set of coupled different equations.

$$\dot{I}_k(t) = \lambda k(1 - I_k(t))\Theta(t), \quad t \le t_0 \tag{1}$$

with normalization conditions

$$S_k(t) + I_k(t) = 1, k = 1, 2, \dots, n,$$

where λ is the infection rate, and

$$\Theta(t) = \langle k \rangle^{-1} \sum_{i=1}^{n} \varphi(i) P(i) I_i(t).$$
 (2)

The factor $\Theta(t)$ represents the probability that any given link points to an infected node, where $P(i) \geq 0$ is the connectivity distribution; $\langle k \rangle = \sum_{i=1}^n i P(i)$ is the mean degree value, and $\varphi(i)$ denotes the infectivity of nodes with degree i. $\varphi(k)$ has many different forms, such as $\varphi(k) = k$ in [20, 21], $\varphi(k) = A$ in [23], $\varphi(k) = k^{\alpha}$, $0 < \alpha < 1$ in [27], and $\varphi(k) = ak^{\alpha}/(1 + bk^{\alpha})$, $0 < \alpha < 1$ in [11] and so on.

From a practical perspective, the initial conditions for system (1) satisfy

$$0 \le I_k(0) \le 1, \ \Theta(0) > 0.$$
 (3)

With the increasing of infected nodes, actively measures are taken to clear virus and prevent computers from being infected by virus again, and then it enters response stage. During this stage, the infected nodes are cured to transform into removed state, and then terminals have three states: susceptible, infected, and removed. But although the virus is cleared and patches are installed, removed nodes still may lost immunity to become susceptible again for

various reasons. Terminals now have three states: the susceptible, the infected and the removed. The spread of virus is governed by the following SIRS model on the Internet in the response stage:

$$\begin{cases} \dot{S}_k(t) = -\lambda k S_k(t) \Theta(t) + \gamma R_k(t), \\ \dot{I}_k(t) = \lambda k S_k(t) \Theta(t) - \mu I_k(t), & t \ge t_0 \\ \dot{R}_k(t) = \mu I_k(t) - \gamma R_k(t) \end{cases}$$
(4)

with normalization conditions

$$S_k(t) + I_k(t) + R_k(t) = 1, k = 1, 2, \dots, n$$

where $\Theta(t)$ is defined by formulation (2), μ, γ are positive constants which represent the removing rate of infected terminals and the rate of the immunization-lost for removed terminals.

The normalization imposes that $R_k(t) = 1 - S_k(t) - I_k(t)$ for $k = 1, 2, \dots, n$, the system (4) is equivalent to the following system:

$$\begin{cases} \dot{S}_k(t) = -\lambda k S_k(t) \Theta(t) + \gamma (1 - S_k(t) - I_k(t)), \\ \dot{I}_k(t) = \lambda k S_k(t) \Theta(t) - \mu I_k(t), \end{cases} \quad t \ge t_0. \tag{5}$$

The initial conditions for system (5) in response stage satisfy

$$S_k(t_0) + I_k(t_0) = 1,$$
 $(R_k(t_0) = 0), k = 1, 2, \dots, n,$ (6)

where $I_k(t_0)$ is the value of $I_k(t)$ at t_0 in free spread stage.

3. Global dynamics of the model

In this section, the properties of model in the free spread stage are analyzed, and then the global dynamics of model in response stage are revealed.

Theorem 3.1. Suppose that $I_k(t)$ $(k = 1, 2, \dots, n)$ is a solution of (1), then $0 < I_k(t) \le 1$ for $0 < t \le t_0$, and $\lim_{t \to +\infty} I_k(t) = 1$ at an exponential rate if $t_0 = +\infty$.

Proof. It follows from (1) and (2) that $\Theta(t)$ satisfies

$$\frac{d\Theta(t)}{dt} = \frac{\lambda\Theta(t)}{\langle k \rangle} \sum_{i=1}^{n} i\varphi(i)P(i)(1 - I_i(t)), \quad 0 < t \le t_0$$
 (7)

which implies $\Theta(t) = \Theta(0) \exp\left\{\frac{\lambda}{\langle k \rangle} \int_0^t \sum_{i=1}^n i\varphi(i) P(i) (1 - I_i(s)) \mathrm{d}s\right\}$. Consequently, $\Theta(t) > 0$ due to $\Theta(0) > 0$. From (1), it is implied that

$$\dot{I}_k(t) > -\lambda k\Theta(t)I_k(t), \quad 0 < t \le t_0.$$

Hence $I_k(t) > I_k(0) \exp\{-\lambda k \int_0^t \Theta(s) ds\} \ge 0$ for $0 < t \le t_0$. The function $1 - I_k(t)$ satisfies the following equation

$$\frac{d(1 - I_k(t))}{dt} = -\lambda k\Theta(t)(1 - I_k(t)), \quad 0 < t \le t_0.$$
 (8)

By similar discussion, we have $1 - I_k(t) \ge 0$ for $0 < t < t_0$.

Equation (7) implies that

$$\frac{d\Theta(t)}{dt} \ge \lambda \Theta(t) \left[\langle k \rangle^{-1} \langle k \varphi(k) \rangle - n\Theta(t) \right], \quad 0 < t \le t_0.$$
 (9)

If $t_0 = +\infty$, one has that $\lim_{t \to +\infty} \Theta(t) \ge \frac{\langle k\varphi(k)\rangle}{n\langle k\rangle}$ from (9). There exist a $\tau > 0$, such that $\Theta(t) > \frac{\langle k\varphi(k)\rangle}{2n\langle k\rangle}$ for $t > \tau$.

Solving (8), it follows that

$$0 \le 1 - I_k(t) = (1 - I_k(\tau)) \exp\left\{-\int_{\tau}^{t} \lambda k \Theta(s) ds\right\}$$
$$\le (1 - I_k(\tau)) \exp\left\{-\frac{\lambda k \langle k \varphi(k) \rangle}{2n \langle k \rangle} (t - \tau)\right\}, \quad t > \tau.$$

which leads to $\lim_{t\to +\infty} I_k(t) = 1$. Thus, the proof is completed.

Theorem 3.1 indicates that the proportion of infected nodes with degree k is always increasing in the first stage. All nodes will be infected with exponential rate if no any anti-virus measure is taken.

However, with the increasing of the infected nodes, it will be noticed to enter response stage. The main results are given in the following Theorem 3.2 and Theorem 3.3.

For convenience, we discuss the system (5) which is equivalent to system (4), and define

$$R_0 = \frac{\lambda \langle k\varphi(k)\rangle}{\mu \langle k\rangle}.$$
 (10)

Theorem 3.2. There always exists a disease-free equilibrium $E^0 = \{1, \dots, 1, 0, \dots, 0\}$ for (5), E^0 is globally asymptotically stable when $R_0 < 1$; when $R_0 > 1$ the system (5) is uniformly persistent, i.e. there exists an $\varepsilon > 0$, such that

$$\liminf_{t \to +\infty} \{S_1(t), \cdots, S_n(t), I_1(t), \cdots, I_n(t)\} \ge \varepsilon,$$

where $\{S_1(t), \dots, S_n(t), I_1(t), \dots, I_n(t)\}$ is any solution of (5) satisfying (6).

Proof. Obviously, $E^0 = \{1, \dots, 1, 0, \dots, 0\}$ is always a equilibrium of system (5).

Define

$$V(t) = \Theta(t).$$

Let $G = \{\phi : \dot{V}(\phi) = 0\}$ and M is the largest set in G which is invariant with respect to system (5). Clearly, M is not empty since $E^0 \in M$.

Calculating the derivative of V(t) along solution of (5), we get

$$\begin{split} \dot{V}(t)\Big|_{(5)} &= \Theta\left[\frac{1}{\langle k\rangle}\sum_{k}\phi(k)p(k)(\lambda kS_{k}\Theta(t)-\mu I_{k}(t)\right] \\ &\leq \Theta\left[\frac{1}{\langle k\rangle}\sum_{k}\phi(k)p(k)(\lambda k\Theta(t)-\mu I_{k}(t)\right] \\ &= \mu(R_{0}-1)\Theta(t)\leq 0. \end{split}$$

It follows from $S_k(t)+I_k(t)+R_k(t)=1$ that $M=E^0$. Consequently, the disease-free equilibrium E^0 is globally attractive according to the LaSalle invariant principle.

The Jacobian matrix of (5) in E^0 is a $2n \times 2n$ matrix as

$$J = \left(\begin{array}{cc} A_{11} & A_{12} \\ \mathbf{0} & A_{22} \end{array}\right)$$

in which $A_{11} = -\gamma \mathbf{I}_{n \times n}$, $A_{12} = -B - \gamma \mathbf{I}_{n \times n}$, $A_{22} = B - \mu \mathbf{I}_{n \times n}$ and

$$B = \frac{\lambda}{\langle k \rangle} \begin{pmatrix} \varphi(1)P(1) & \varphi(2)P(2) & \cdots & \varphi(n)P(n) \\ 2\varphi(1)P(1) & 2\varphi(2)P(2) & \cdots & 2\varphi(n)P(n) \\ \cdots & \cdots & \cdots & \cdots \\ n\varphi(1)P(1) & n\varphi(2)P(2) & \cdots & n\varphi(n)P(n) \end{pmatrix}.$$

The characteristic polynomial can be calculated as follows.

$$(z+\gamma)^n(z+\mu)^{n-1}(z+\mu-\lambda\langle k\rangle^{-1}\langle k\varphi(k)\rangle)=0.$$

Note that all eigenvalue of J are negative when $R_0 < 1$, that is to say, E^0 is locally asymptotically stable, furthermore, we know E^0 is globally asymptotically stable according to global attractivity of E^0 .

On the other hand, there exists a unique positive eigenvalue z of J if and only if $R_0 > 1$, and E^0 is unstable. Then, a Theorem of Lajmanovich and York [4] implies the uniform persistence of system (5) when $R_0 > 1$. The proof is completed.

Theorem 3.3. If $R_0 > 1$, system (5) has a unique endemic equilibrium

$$E^* = \{S_1^*, \cdots, S_n^*, I_1^*, \cdots, I_n^*\},\$$

which is globally asymptotically attractivive, i.e., $\lim_{t\to+\infty} \{S_k(t), I_k(t)\} = \{S_k^*, I_k^*\}$ for any solution $\{S_1(t), \dots, S_n(t), I_1(t), \dots, I_n(t)\}$ of system (5) satisfying (6).

Proof. First, we discuss the existence of endemic equilibrium for system (5). From $S_k(t) + I_k(t) + R_k(t) = 1$ that $0 \le I_k(t) \le 1$, we have from (2) that $\Theta(t) \le \langle k \rangle^{-1} \langle \varphi(k) \rangle \doteq \rho$.

Let $\dot{S}_k(t) = 0$, $\dot{I}_k(t) = 0$, a direct calculation yields

$$S_k = \frac{\mu \gamma}{A_k}, \quad I_k = \frac{\lambda k \gamma \Theta}{A_k},$$
 (11)

where $A_k = \lambda k(\mu + \gamma)\Theta + \mu\gamma$ and $\Theta = \langle k \rangle^{-1} \sum_{i=1}^n \varphi(i)P(i)I_i$.

Substituting I_k obtained above into Θ , an equation of the form $\Theta f(\Theta) = 0$ is obtained, where

$$f(\Theta) = 1 - \frac{\lambda \gamma}{\langle k \rangle} \sum_{k=1}^{n} \frac{k\varphi(k)P(k)}{A_k}.$$

If non-trivial solution Θ^* exists, it satisfies $f(\Theta^*) = 0$. Since $f'(\Theta) > 0$ and $f(\rho) > 0$, a non-trivial Θ^* exists if and only if $f(0) = 1 - R_0 < 0$, i.e. $R_0 > 1$. Substituting Θ^* into (11), we can know that system (5) has a unique positive equilibrium (the endemic equilibrium) $E^* = \{S_1^*, \dots, S_n^*, I_1^*, \dots, I_n^*\}$ when $R_0 > 1$.

Secondly, we discuss the attractivity of the endemic equilibrium.

By Theorem 3.2, there exists a constant $0 < \xi < \frac{1}{2}$ and a lager enough constant T > 0 such that $S_k(t) > \xi, I_k(t) > \xi$. Hence one can obtain that

 $\rho \xi < \Theta(t) < \rho$ for t > T. Substituting this into the first equation of (5), it yields

$$\dot{S}_k(t) \le -\lambda k \rho \xi S_k(t) + \gamma (1 - S_k(t)), \quad t > T.$$

By the standard comparison theorem in the theory of differential equations, for any given constant $0 < \xi_1 < \frac{\lambda k \rho \xi}{2[\lambda k \rho \xi + \gamma]}$, there exists a $T_1 > T$ such that $S_k(t) \leq X_k^{(1)} - \xi_1$ for $t > T_1$, where

$$X_k^{(1)} = \frac{\gamma}{\lambda k \rho \xi + \gamma} + 2\xi_1 < 1.$$

It then follows from the second equation of (5) that

$$\dot{I}_k(t) \le \lambda k \rho (1 - I_k(t)) - \mu I_k(t).$$

Hence, for a given constant $0 < \xi_2 < \min\{\frac{1}{2}, \xi_1, \frac{\mu}{2[\lambda k \rho + \mu]}\}$, there exists a $T_2 > T_1$ such that $I_k(t) \le Y_k^{(1)} - \xi_2$ for $t > T_2$, where

$$Y_k^{(1)} = \frac{\lambda k \rho}{\lambda k \rho + \mu} + 2\xi_2 < 1.$$

On the other hand,

$$\dot{S}_k(t) \ge \lambda k \rho S_k(t) + \gamma (1 - S_k(t) - Y_k^{(1)}), \quad t > T_2.$$

Then, for a given constant $0 < \xi_3 < \min\{\frac{1}{3}, \xi_2, \frac{\gamma(1-Y_k^{(1)})}{\lambda k \rho + \gamma}\}$, there exists a $T_3 > T_2$ such that

$$S_k(t) \ge x_k^{(1)} \doteq \frac{\gamma(1 - Y_k^{(1)})}{\lambda k \rho + \gamma} - \xi_3 > 0$$

for $t > T_4$. It follows that

$$\dot{I}_k(t) \ge \lambda k \rho \xi x_k^{(1)} - \mu I_k(t), \quad t > T_3.$$

For a given constant $0 < \xi_4 < \min\{\frac{1}{4}, \xi_3, \frac{\lambda k \rho \xi x_k^{(1)}}{\mu}\}$, there exists a $T_4 > T_3$ such that

$$I_k(t) \ge y_k^{(1)} \doteq \frac{\lambda k \rho \xi x_k^{(1)}}{\mu} - \xi_4 > 0$$

for $t > T_4$.

Furthermore, the inequalities $0 < x_k^{(1)} < S_k(t) < X_k^{(1)} < 1$ and $0 < y_k^{(1)} < I_k(t) < Y_k^{(1)} < 1$ hold because ξ is a small constant.

Let

$$\theta^{(j)} = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \varphi(i) P(i) y_i^{(j)}, \Theta^{(j)} = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \varphi(i) P(i) Y_i^{(j)},$$

for $j = 1, 2, \dots, n$. From the discussion above, it is clear that

$$0 < \theta^{(j)} \le \Theta(t) \le \Theta^{(j)} \le \rho.$$

Again, by (5), one has

$$\dot{S}_k(t) \le -\lambda k \theta_k^{(1)} S_k(t) + \gamma (1 - S_k(t) - y_k^{(1)}), \quad t > T_4.$$

Hence, for any given constant $0 < \xi_5 < \min\{\frac{1}{5}, \xi_4\}$, there exists a $T_5 > T_4$ such that $S_k(t) \le X_k^{(2)} - \xi_5$ for $t > T_5$, where

$$X_k^{(2)} = \min\{X_k^{(1)} - \xi_1 + \xi_5, \frac{\gamma(1 - y_k^{(1)})}{\lambda k \theta^{(1)} + \gamma} + 2\xi_5\}.$$

Thus,

$$\dot{I}_k(t) \le \lambda k \Theta^{(1)} X_k^{(2)} - \mu I_k(t), \quad t > T_5.$$

So, for any given constant $0 < \xi_6 < \min\{\frac{1}{6}, \xi_5\}$, there exists a $T_6 > T_5$ such that $I_k(t) \ge Y_k^{(2)} - \xi_6$ for $t > T_6$, where

$$Y_k^{(2)} \doteq \min\{Y_1^{(1)} - \xi_2 + \xi_6, \frac{\lambda k \Theta^{(1)}}{\mu} X_k^{(2)} + 2\xi_6\}.$$

Turning back to (5), it gives that

$$\dot{S}_k(t) \ge -\lambda k \Theta^{(2)} S_k(t) + \gamma (1 - S_k(t) - Y_k^{(2)}), \quad t > T_6.$$

So, for any given constant $0 < \xi_7 < \min\{\frac{1}{7}, \xi_6, \frac{\gamma(1-Y_k^{(2)})}{\lambda k \Theta^{(2)} + \gamma}\}$, there exists a constant $T_7 > T_6$ such that

$$I_k(t) \ge x_k^{(2)} \doteq \frac{\gamma(1 - Y_k^{(2)})}{\lambda k \Theta^{(2)} + \gamma} - \xi_7$$

for $t > T_7$. Thus,

$$\dot{I}_k(t) \ge \lambda k \theta^{(1)} x_k^{(2)} - \mu I_k(t), \quad t > T_7.$$

So, for any given constant $0 < \xi_8 < \min\{\frac{1}{8}, \xi_7, \frac{\lambda k \theta^{(1)} x_k^{(2)}}{\mu}\}$, there exists a $T_8 > T_7$ such that

$$I_k(t) \ge y_k^{(2)} \doteq \frac{\lambda k \theta^{(1)} x_k^{(2)}}{\mu} - \xi_8$$

for $t > T_8$. Similarly, step l ($l = 3, 4, \cdots$) of the calculation can be carried out and four sequences are obtained:

$$\begin{split} X_k^{(1)} &= \frac{\gamma}{\lambda k \rho \xi + \gamma} + 2\xi_1, \\ X_k^{(l)} &= \min \left\{ X_k^{(l-1)} - \xi_{4l-7} + \xi_{4l-3}, \frac{\gamma(1 - y_k^{(l-1)})}{\lambda k \theta^{(l-1)} + \gamma} + 2\xi_{4l-3} \right\}, \\ Y_k^{(1)} &= \frac{\lambda k \rho}{\lambda k \rho + \mu} + 2\xi_2, \\ Y_k^{(l)} &= \min \left\{ Y_k^{(l-1)} - \xi_{4l-6} + \xi_{4l-2}, \frac{\lambda k \Theta^{(l-1)}}{\mu} X_k^{(l)} + 2\xi_{4l-2} \right\}, \\ x_k^{(1)} &= \frac{\gamma(1 - Y_k^{(1)})}{\lambda k \rho + \gamma} - \xi_3, \quad x_k^{(l)} &= \frac{\gamma(1 - Y_k^{(l)})}{\lambda k \Theta^{(l)} + \gamma} - \xi_{4l-1}, \\ y_k^{(1)} &= \frac{\lambda k \rho \xi x_k^{(1)}}{\mu} - \xi_4, \qquad y_k^{(l)} &= \frac{\lambda k \theta^{(l-1)}}{\mu} x_k^{(l)} - \xi_{4l} \end{split}$$

for $l = 2, 3, 4, \cdots$.

Since the first two $X_k^{(l)}, Y_k^{(1)}$ are monotone decreasing sequences and the last two $x_k^{(l)}, y_k^{(l)}$ are strictly monotone increasing sequences, there exists a large positive integer M such that

$$X_{k}^{(l)} = \frac{\gamma(1 - y_{k}^{(l-1)})}{\lambda k \theta^{(l-1)} + \gamma} + 2\xi_{4l-3},$$

$$Y_{k}^{(l)} = \frac{\lambda k \Theta^{(l-1)}}{\mu} X_{k}^{(l)} + 2\xi_{4l-2},$$

$$x_{k}^{(l)} = \frac{\gamma(1 - Y_{k}^{(l)})}{\lambda k \Theta^{(l)} + \gamma} - \xi_{4l-1},$$

$$y_{k}^{(l)} = \frac{\lambda k \theta^{(l-1)}}{\mu} x_{k}^{(l)} - \xi_{4l}$$
(12)

for $l \geq M$.

It is clear that

$$x_k^{(l)} \le S_k(t) \le X_k^{(l)}, \quad y_k^{(l)} \le I_k(t) \le Y_k^{(l)}, \quad t \ge T_{4l}.$$
 (13)

Hence, the sequential limits of (12) exist. Let $\lim_{l\to+\infty} \Omega_k^{(l)}$, where

$$\Omega_k^{(l)} \in \left\{ x_k^{(l)}, y_k^{(l)}, X_k^{(l)}, Y_k^{(l)}, \theta_k^{(l)}, \Theta_k^{(l)} \right\}$$

and $\Omega_k = \{x_k, y_k, X_k, Y_k, \theta, \Theta\}$. It follows that $\lim_{l \to +\infty} \xi_i = 0$ from $0 < \xi_l < \frac{1}{l}$. By taking $l \to +\infty$ in the sequences of (12), a direct computation yields

$$x_k = \frac{\gamma(1 - Y_k)}{\lambda k\Theta + \gamma}, \quad X_k = \frac{\gamma(1 - y_k)}{\lambda k\theta + \gamma},$$
 (14)

where

$$\theta = \langle k \rangle^{-1} \sum_{k=1}^{n} \varphi(k) P(k) y_k, \Theta = \langle k \rangle^{-1} \sum_{k=1}^{n} \varphi(k) P(k) Y_k.$$

Further,

$$y_{k} = \frac{1}{H_{k}} \lambda k \gamma \theta \left[(\lambda k \theta + \gamma) \mu - \lambda k \gamma \Theta \right],$$

$$Y_{k} = \frac{1}{H_{k}} \lambda k \gamma \Theta \left[(\lambda k \Theta + \gamma) \mu - \lambda k \gamma \theta \right]$$
(15)

in which $H_k = (\lambda k\Theta + \gamma)(\lambda k\theta + \gamma)\mu^2 - \lambda^2 k^2 \gamma^2 \theta\Theta$. Substituting (15) into θ and Θ , it is obtained that

$$1 = \frac{\lambda \gamma}{\langle k \rangle} \sum_{k=1}^{n} \frac{\varphi(k)P(k)}{H_{k}} k \left[(\lambda k \theta + \gamma)\mu - \lambda k \gamma \Theta \right],$$

$$1 = \frac{\lambda \gamma}{\langle k \rangle} \sum_{k=1}^{n} \frac{\varphi(k)P(k)}{H_{k}} k \left[(\lambda k \Theta + \gamma)\mu - \lambda k \gamma \theta \right].$$
(16)

By subtracting the above two equations, it is obtained that

$$\frac{\lambda^2 \gamma(\mu + \gamma)}{\langle k \rangle} (\theta - \Theta) \sum_{k=1}^n \frac{\varphi(k) P(k)}{H_k} k^2 \equiv 0.$$

This implies that $\theta = \Theta$. Hence $\langle k \rangle^{-1} \sum_{k=1}^{n} \varphi(k) P(k) (y_k - Y_k) = 0$, which is equivalent to $y_k = Y_k$ for $0 \le k \le n$. Then it follows from (13) and (14) that

$$\lim_{t \to \infty} S_k(t) = x_k = X_k, \quad \lim_{t \to \infty} I_k(t) = y_k = Y_k.$$

Finally, by substituting $\theta = \Theta$ into (15) and (16), in view of (12) and (14), it is easy get that $x_k = X_k = S_k^*, y_k = Y_k = I_k^*$. This completes the proof.

According to Theorems 3.1-3.3, when $R_0 < 1$, the virus will be cleaned and all terminals will become healthy; otherwise, when $R_0 > 1$, the virus will persist and the proportions of susceptible, infected and removed nodes will converge to the unique stationary positive levels eventually. R_0 is the basic productive number for the spreading of virus.

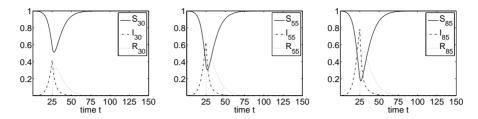


Fig. 1: The time series of system with parameter $\lambda=0.16,~\mu=0.4,$ and $\gamma=0.14.$ Then $R_0=0.6429.$

Remark 1. One may find that SIRS model describing virus spread in the response stage in [7] is similar to model (4) in this paper, the authors investigated the global stability of the endemic equilibrium by constructing an Lyapunov function. The main verifying tools for global attractivity of the positive equilibrium in this paper are the comparison theorem and iteration principle. In addition, author also try to discuss the global attractivity of the endemic equilibrium for an SEIRS epidemic model by using the comparison theorem and iteration principle in [10], but there are some flaws in their proof, for example, the inequality about ξ_{10} and so on. One may find $Y_k^{(2)} < 1$ and $Z_k^{(2)} < 1$, which is obtained from the construction of the sequences, does not guarantee that the right side of inequality is positive. These flaws lead to failure of sequences construction.

4. Numerical simulations

In this section, we present the result of numerical simulations. The following numerical simulation are implemented based on the scale-free network with $p(k) = Ck^{-\gamma}$, and C satisfies $\sum_{k=1}^{n} p(k) = 1$.

Assume that the network is finite network, let n=100 and r=2.5, which is a suitable assumption. The infectivity of nodes with degree k is choose as $\varphi(k)=\frac{ak^{\alpha}}{1+bk^{\alpha}}$, where $a=0.5, b=0.02, \alpha=0.75$. The dividing point of free spread stage and response stage is $t_0=25$. The initial values are $I_k(0)=0, S_k(0)=1$ $(k=1,2,\cdots,100,)$ and $I_5(0)=0.005, S_5(0)=0.995$.

In Figs. 1–4, the time series of systems (1) and (4) with degree 30, 55 and 85 are manifested. But it should be noted that the time series of systems with other degrees are analogous.

Figs. 1–2 show the outcome of (1) and (4) when $R_0 < 1$. The virus will disappear and all the terminals will become susceptible even if it is longer time

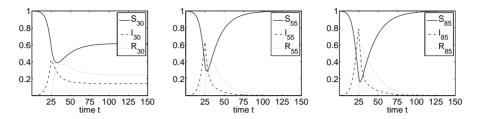


Fig. 2: The time series of system with parameter $\lambda=0.16,~\mu=0.31,$ and $\gamma=0.14.$ Then $R_0=0.8296.$

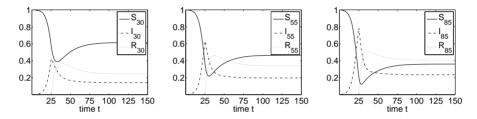


Fig. 3: The time series of system with parameter $\lambda = 0.16$, $\mu = 0.19$, and $\gamma = 0.11$. Then $R_0 = 1.3536$.

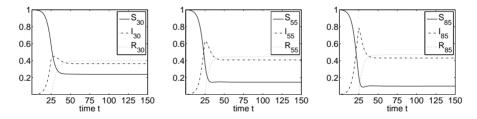


Fig. 4: The time series of system with parameter $\lambda = 0.16$, $\mu = 0.12$, and $\gamma = 0.11$. Then $R_0 = 2.1432$.

before taking anti-virus measures. Moreover, the smaller R_0 is, the faster the virus disappears.

Figs. 3–4 show the outcome of (1) and (4) when $R_0 > 1$. One can see that when $R_0 > 1$, the virus is uniformly persistent, which means that the density of infected nodes will converge to a stationary positive level. Further, the larger R_0 is, the faster and higher the density of infected nodes reach to the stationary positive level and the higher the stationary positive level is.

5. Conclusion

To describe the propagation of computer virus spread, a piecewise model of

computer virus concluding free spread stage and response stage is constructed on the Internet. The results show that the density of infected nodes is always increase in the free spread stage because no any anti-virus measure is taken. In the response stage, it is proven that the disease-free equilibrium is globally stable and so the viruses will disappear when the basic reproductive number is less than unity. Otherwise, the endemic equilibrium is globally attractive and so the density of infected nodes will tend to a positive constant. The main verifying tools for global attractivity of the positive equilibrium are the comparison theorem and iteration principle, and these methods may be generalized to the other models such as SIS, SIRS models with time delay. These will be aims for our next works.

References

- [1] A. Barabási, R. Albert, Emergence of scaling in random networks, *Science*, **286** (1999), 509-512.
- [2] A.L. Lloyd, R.M. May, How viruses spread among computers and people, *Science*, **292** (2001), 1316-1317.
- [3] A. d'Onofrio, A note on the global behavior of the network-based SIS epidemic model, *Nonlinear Analysis: Real World Application*, **9**, No 4 (2008), 1567-1572.
- [4] A. Lajmanovich, J.A. Yorke, A deterministic model for gonorrhea in a nonhomogenous population, *Mathematical Biosicience*, **28** (1976), 221-236.
- [5] C. Griffin, R. Brooks, A note on the spread of worms in scale-free networks, *IEEE Trans. Syst. Man Cybernet. Part B: Cybernet.* **36** (2006), 198-202.
- [6] C. Castellano, R. Pastor-Satorras, Thresholds for epidemic spreading in networks, *Physical Review Letters*, 105 (2010), 218701.
- [7] C. Li, C. Tsai, S. Yang, Analysis of epidemic spreading of an SIRS model in complex heterogeneous networks, *Communications in Nonlinear Science and Numerical Simulation*, **19**, No 4 (2014), 1042-1054.
- [8] F. Cohen, Computer viruses theory and experiments, *Computer Security*, **6**, No 1 (1987), 22-35.

- [9] G.H. Zhu, X.C. Fu, G.R. Chen, Global attractivity of a network-based epidemic SIS model with nonlinear infectivity, *Communications in Nonlinear Science and Numerical Simulation*, **17**, No 6 (2012), 2588-2594.
- [10] G. Zhu, X. Fu, G. Chen, Spreading dynamics and global stability of a generalized epidemic model on complex heterogeneous networks, *Applied Mathematical Modelling*, **36**, No 12 (2012), 5808-5817.
- [11] H. Zhang, X. Fu, Spreading of epidemics on scale-free networks with non-linear infectivity. *Nonlinear Analysis*, **70**, No 9 (2009), 3273-3278.
- [12] J.R.C. Piqueira, B.F. Navarro, L.H.A. Monteiro, Epidemiological models applied to viruses in computer networks, *Journal of Computer Science*, 1 (2005), 31-34.
- [13] J.R.C. Piqueira, A.A. de Vasconcelos, C.E.C.J. Gabriel, V.O. Araujo, Dynamic models for computer viruses, *Computer Security*, **27**, No 7-8 (2008), 355-359.
- [14] J.C. Wierman, D.J. Marchette, Modeling computer virus prevalence with a susceptible-infected-susceptible model with reintroduction, *Computational Statistics and Data Analysis* 45, No 1 (2004), 3-23.
- [15] J. Liu, T. Zhang, Epidemic spreading of an SEIRS model in scale-free networks, Communications in Nonlinear Science and Numerical Simulation, 16, No 8 (2011), 3375-3384.
- [16] L. Chen, J. Sun, Global stability and optimal control of an SIRS epidemic model on heterogeneous networks, *Physica A*, **10** (2014), 196-204.
- [17] M. Karsai,. Kivel, R.K. Pan, K. Kaski, et al., Small but slow world: how network topology burstiness slow down spreading, *Physical Review E*, **83** (2011), 025102.
- [18] M. Draief, A. Ganesh, L. Massouili, Thresholds for virus spread on networks, Annals of Applied Probability, 18 (2008), 59-378.
- [19] P. Szor, The Art of Computer Virus Research and Defense, Addison-Wesley Education Publishers Inc. (2005).
- [20] R. Pastor-Satorras, A. Vespignani, Epidemic spreading in scale-free networks, *Physical Review Letters*, 86, No 14 (2001), 3200-3203.

[21] R. Pastor-Satorras, A. Vespignani, Epidemic dynamics and endemic states in complex networks, *Physical Review Letters*, **63** (2001), 066117.

- [22] S. Wang, Q. Liu, Bifurcation analysis of a model for network worm propagation with time delay, *Mathematics and Computer Modelling*, **52**, No 3-4 (2010), 435-447.
- [23] T. Zhou, J.G. Liu, W.J. Bai, et al., Behaviors of susceptible-infected epidemics on scale-free networks with identical infectivity, *Physical Review E*, 74 (2006), 056109.
- [24] T. Li, Y. Wang, Z. Guan, Spreading dynamics of a SIQRS epidemic model on scale-free networks, Communications in Nonlinear Science and Numerical Simulation, 19, No 3 (2014), 686-692.
- [25] W.H. Murray, The application of epidemiology to computer viruses, *Computer Security*, **7**, No 2 (1988), 130-150.
- [26] X. Han, Q. Tan, Dynamical behavior of computer virus on Internet, Applied Mathematics and Computer, 217, No 6 (2010), 2520-2526.
- [27] X. Cheng, X. liu, Z. Chen, Z. Yuan, Spreading behavior of SIS model with non-uniform transmission on scale-free networks, *Beijing University of Posts and Telecommunications*. *Journal* **16**, No 1 (2009), 27-31.
- [28] Y. Moreno, R. Pastor-Satorras, A. Vespignani, Epidemic outbreaks in complex heterogeneous networks, *The European Physical Journal B*, **26**, No 4 (2002), 521-529.