A Network Virus Propagation Model with Distributed Infectious Period Delay in Internet

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Abstract. A novel propagation model with distributed infectious period delay of network virus is presented. The formula of basic reproduction number for the model is given. The viruses eventually disappear when the basic reproductive number is below the unity, otherwise the viruses are persistent.

1. Introduction
Network viruses have become a major problem for the security of computer networks. Facing with the grim situation of network virus destruction, it’s urgent for people to construct some effective mathematical model to predict propagation regularity of the network virus so that we can control the spread of virus efficiently.

The Internet network can be regarded as a complex network, where nodes and links represent computers connected to the Internet and communication links between them, respectively. An important discovery is the scale-free property [1], that is to say, the node degrees of Internet follow a power law distribution, \( p(k) \sim k^{-\gamma} \), where \( p(k) \) stands for the probability that a node chosen randomly from Internet is of degree \( k \). Noting that the similarities between network viruses and biological viruses, many scholars in the world have exploited the compartment modeling technique used in the spreading of biological viruses to study the spreading of network viruses in scale-free network [3-10].

Compared with ordinary differential equation (ODE) models, more realistic models should be functional differential equation (FDE) models, and time delay can describe the incubation period of the infectious disease, the immunity period of recovery of the disease and the infectious period of patients for FDE model. Unfortunately, only a few attention has been paid to the epidemic models with time delays on complex network [7-10]. In the real world, many infected nodes may have different infectious period due to the infected nodes’ different temporal, social, and physical contexts. Hence it may be more realistic to assume that the delay is considered in a distributed manner [11]. In this paper, we will presented a novel FDE SIS epidemic model with infinite distributed delay on scale-free network in this paper.

Without losing generality, let us assume the total number of nodes is fixed. Let \( S_k(t) \) and \( I_k(t) \) be the relative densities of susceptible and infected nodes of degree \( k \) at time \( t \), and \( S_k(t) \) and \( I_k(t) \), at the mean-field level, satisfy the following set of coupled functional different equations [6, 7]

\[
\begin{align*}
\frac{dI_k(t)}{dt} &= \lambda(k)S_k(t)\Theta(t) - \tilde{\lambda}(k)\int_{0}^{\infty}S_j(t-v)\Theta(t-v)f(v)dv, \\
S_k(t) &= 1 - I_k(t),
\end{align*}
\]

with the normalized condition
where \( \lambda(k) \) is the infection rate such as \( \lambda k[3] \), \( \lambda \epsilon(k) [4] \) and so on. The kernel function \( f(v) \) denotes the fraction of the infective nodes in which the time taken to become the removed is \( u \) and satisfies the following assumption:

\[
f(v) \geq 0, \quad \int_0^\infty f(v)dv = 1, \quad 0 \leq \int_0^\infty vf(v)dv < +\infty.
\]

There are many types of kernel function such that the gamma distribution, normal distribution, uniform distribution, and Delta-distribution and so on. \( \Theta(t) \) denotes the probability from any given link to an infected node. It satisfies the relation

\[
\Theta(t) = \frac{1}{\langle k \rangle} \sum \varphi(k) P(k) \theta(t),
\]

where \( \varphi(k) = ak^\beta/(1+bk^\beta) (a > 0, 0 \leq \alpha \leq 1, b \geq 0) \) represents the nonlinear infectivity related with the degree of an infected node, and \( \lim_{k \to +\infty} \varphi(k) = a/b \) when \( b \neq 0 \) [4]. This is consistent with feature that the infectivity of an infected node is limited because network bandwidth are limited. \( \langle k \rangle \) represents the average degree for the network.

Normally, there is an inevitable lag from the appear of a new virus to the release of the antivirus software targeting the network virus, and the network virus can propagation freely through the Internet during the lag. The model is suitable for the early spreading of network viruses, there is not the removed nodes because effective measures to clear the viruses have not appear.

The remainder of this paper is structured as follows. We discuss dynamical behaviors of system (1) in Section 2. We will give some numerical simulations to verify the main results in Section 3. At last, we summarize this work in Section 4.

### 2. Main Results

Let

\[
R_0 = \frac{1}{\langle k \rangle} \langle \lambda(k) \varphi(k) \rangle \int_0^\infty vf(v)dv
\]

Obviously, \( \int_0^\infty vf(v)dv \) represents the average infectious period of the patients according to the definition of mathematical expectation.

We shall know that \( R_0 \) is basic reproduction number. The basic reproduction number represents the average number of secondary infectious infected by an infected node during whole course of network virus in the case that all the nodes are susceptible [11].

System (1) can become the equivalent system (4) as follows.

\[
\begin{aligned}
I_i(t) & = \lambda(k) \int_0^\infty S_i(s) \Theta(s) df(v)dv, \\
S_i(t) & = 1 - I_i(t),
\end{aligned}
\]

and we only need to study the dynamical behaviors of system (4).

By using similar method in [4, 10], it is easy to know that system (4) has always a virus-free equilibrium \( E_0((1,0),1) \), and it also has a unique endemic equilibrium \( E_1((S_i', I_i')) \) when \( R_0 >1 \).

Now we give the main results in this paper.

**Theorem.** If \( R_0 <1 \), the disease-free equilibrium \( E_0 \) of system (4) is globally attractive and the relative average density of infected nodes \( I(t) = \sum p(k)I_i(t) \) satisfies \( \lim_{t \to +\infty} I(t) = 0 \). If \( R_0 >1 \), the viruses are persistent, i.e.,

\[
\liminf_{t \to +\infty} I(t) = \sum p(k)I_i(t) > \varepsilon.
\]
there where $\varepsilon$ is a positive constant.

**Proof:** First, we proof that the globally attractivity of the viruse-free equilibrium $E_0$ if $R_0 < 1$.

Consider the following system

\[
\begin{cases}
I_k(t) = \lambda(k) \int_{\tau}^{t} S_j(s) \Theta(s) ds \phi(v) dv, \\
S_j(t) = 1 - I_k(t),
\end{cases}
\]

where $h > 0$.

We have from (2) that

\[
\Theta(t) = \frac{\sum \phi(k) P(k) I_k(t)}{\langle k \rangle} \leq \frac{\sum \phi(k) P(k)}{\langle k \rangle} \Theta_0
\]

Note that

\[
\Theta(t) = \frac{\sum \phi(k) P(k) I_k(t)}{\langle k \rangle} \leq \frac{\sum \phi(k) P(k) \lambda(k) \int_{\tau}^{t} S_j(s) \Theta(s) ds \phi(v) dv}{\langle k \rangle}
\]

and

\[
0 < \int_{0}^{t} v f(v) dv \leq \int_{h}^{\infty} v f(v) dv.
\]

When $t > h$, we have from (6) that

\[
\Theta(t) \leq \frac{\sum \phi(k) P(k) \lambda(k) \int_{\tau}^{t} S_j(s) \Theta(s) ds \phi(v) dv}{\langle k \rangle} = \Theta_0 R_0
\]

Substituting it into (6), for $t > 2h$, we have

\[
\Theta(t) \leq \Theta_0 R_0^2
\]

By induction method, we have $\Theta(t) \leq \Theta_0 R_0^t$ for $t > nh$.

Hence $0 \leq \lim_{t \to \infty} \Theta(t) \leq \lim_{t \to \infty} \Theta(R_0^t) = 0$. Moreover, it follows that $\lim_{t \to \infty} I_k(t) = 0$ for system (5).

Let $h \to +\infty$, we have $\lim_{t \to \infty} I_k(t) = 0$ hold for system (4). So the equilibrium $E_0$ of system (4) is globally attractive when $R_0 < 1$. Consequently, $\lim_{t \to \infty} I_k(t) = \lim_{t \to \infty} \sum_{k} \phi(k) P(k) I_k(t) = 0$.

Second, we discuss the persistence of the network viruses if $R_0 > 1$.

Denote

\[
\begin{align*}
D &= \{(S_j, I_k, \cdots, S_j, I_k) : I_k(t) \geq 0, \tau \in (-\infty, 0], k = 1, 2, \cdots, n\}, \\
D_0 &= \{(S_j, I_k, \cdots, S_j, I_k) : I_k(t) \geq 0, \tau \in (-\infty, 0], k = 1, 2, \cdots, n\}, \\
\mathcal{D}_0 &= D \setminus D_0.
\end{align*}
\]

Obviously, system (4) is a dissipative system, and $D_0$ is positively invariant set of system (4). Let $\Omega$ be the omega limit set of system (4) on $\mathcal{D}_0$, $E_0$ is the unique equilibrium of system (4) on $\mathcal{D}_0$ and
0 = E\$, E\$ is isolated and acyclic. Hence the proof will be completed if we can prove that 
$W^s(E\$) \cap D_\$ = \phi, in which $W^s(E\$) is the stable manifold of $E\$ [12]. Suppose it is not true, there exists a 
solution $(S_\$(t), I_\$(t), \ldots, S_{n}(t), I_{n}(t))$ of system (4) in $D_\$ such that $S_{n}(t) \to 1, I_{n}(t) \to 0$ as $t \to +\infty$.

Noting that $\gamma > 1$, we can choose $0 < \gamma < 1$ such that $(1-\gamma)R_0 > 1$, and we can also choose a sufficiently 
large positive number $h$ such that $R_0 = 1/ \{ k \} \langle \lambda(k)\rho(k) \rangle^h_{k} \neq 1/ \{ v \} < 1$.

For $\gamma > 0$, there exists a $T > 0$ such that $S_{n}(t) > 1-\gamma, 0 \leq I_{n}(t) < \eta$, for $t > T$, $k = 1, 2, \ldots, n$.

Let 
\[ I_{n}(t) = \lambda(k) \int_{k}^{\infty} \left( \int_{0}^{k} S_{k}(s)\Theta(s)ds \right) f(v)dv \]
\[ \geq \lambda(k) \int_{0}^{k} \left( \int_{0}^{\infty} S_{k}(s)\Theta(s)ds \right) f(v)dv \]
\[ \geq \lambda(k)(1-\eta) \int_{0}^{k} \left( \int_{0}^{\infty} \Theta(s)ds \right) f(v)dv. \]

And we have 
\[ \Theta(t) \geq \langle \lambda(k)\rho(k) \rangle \langle 1-\eta \rangle \int_{0}^{k} \langle \int_{0}^{\infty} \Theta(s)ds \rangle f(v)dv \]

By using similar methods in [10], we can conclude that it is impossible that $\Theta(t) < \rho(k)\eta / \langle k \rangle$ for $t > T$. Hence 
liminf_{t \to +\infty} \Theta(t) \neq 0$. Furthermore, there exists a $k_0 \in \{ 1, 2, \ldots, n \}$ such that 
liminf_{t \to +\infty} I_{k_0}(t) \neq 0$. Contradicting $\liminf_{t \to +\infty} I_{k}(t) = 0$ for $k = 1, 2, \ldots, n$. Consequently,
\[ \liminf_{t \to +\infty} I_{k}(t) = \sum_{k} p(k)\lambda_{k}(t) > 0. \]

Hence, the virus is uniformly persistent according to Lemmal in [12].

Remark. Through the above theoretical analysis, we conclude that the network viruses will 
disappear when $R_0 < 1$, the network viruses will be persistent when $R_0 > 1$. Hence $R_0$ is basic 
reproduction number for system (1).

3. Numerical Simulation

Now we give some numerical simulations to support the results obtained in Section 2. The simulations are based on a scale-free network in which the degree distribution is $P(k) = c(k-\gamma)^{\gamma-2}$ and 
c satisfies $\sum_{k} p(k) = 1$. The maximum degree $k_{max} = 100$, the minimum degree $k_{min} = 1$.

Consider system (1) with parameters $\lambda(k) = \lambda k, \alpha = 0.5, \sigma = 0.75, b = 0.02, \eta = 0.02$, and let kernel function be a 
class of gamma distribution function and Delta distribution function, respectively, i.e.,

(a) The gamma distribution $f(v) = \frac{v^{a-1}}{(a-1)!b^{a}}e^{-v/b}$, where $b$ is a real number. Here we take $a = 1, b = 1$ and 
$f(v) = e^{-v}.$

(b) $f(v) = \delta(t-1)$, and the system (1) become the following system with discrete delay 
\[ \begin{aligned} 
\frac{d I_{k}(t)}{dt} &= \lambda(k)S_{k}(t)\Theta(t) - \lambda(k)S_{k}(t-1)\Theta(t-1), \\
S_{k}(t) &= 1 - I_{k}(t). 
\end{aligned} \] (7)

For the initial conditions $I_{k}(s) = 0.15$ and $I_{k}(s) = 0, k \neq 5$ for $s \in (-\infty, 0]$, Fig. 1-2 show the relative 
average density of infected nodes $I(t) = \sum_{k} p(k)\lambda_{k}(t).$

It is clearly seen from Fig. 1-2 that network viruses will tend to extinction if $R_0 < 1$, and network 
viruses are persistent if $R_0 > 1$. These results qualitatively agree with Theorem in Section 2.
Although the average infectious period $\int_{0}^{\infty} ve^{-v} dv = 1$, the dynamical behaviors of model (1) with $f(v) = e^{v}$ is very different from system (7) one with discrete infectious period delay in the process of epidemic spreading, this implies that it is very important to discuss the epidemic spreading model with distributed delay in heterogeneous network.

**Figure 1.** Evolutions of $I(t)$ for system (1) with $\lambda = 0.02, \tau = 1$ and $R_0 = 0.3205 < 1$

**Figure 2.** Evolutions of $I(t)$ for system (1) with $\lambda = 0.8, \tau = 1$ and $R_0 = 1.2820 > 1$.

We would like to point that it is interesting but challenging to discuss the global attractivity of equilibrium $E$. We will investigate it in the future.

### 4. Conclusion
We introduce a novel SIS model with infinite distributed delay describe the propagation of network viruses on the scale-free network. The basic reproduction number for the model has been given. The global stability of the disease-free equilibrium has been shown when the basic reproduction number is below one, whereas the persistence of the network viruses has been proved when the basic reproduction number is above one. These results help to develop policies of prevention and control for network viruses.

### 5. Acknowledgments
This work is supported by the Hebei Provincial Natural Science Foundation of China under Grant A2016506002.
6. References

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