On computer virus spreading using node-based model with time-delayed intervention strategies

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Dear editor,

Computer virus has become a major factor that poses a severe threat to contemporary information systems [1]. Some common models, e.g., threshold models, compartment-based models, and node-based models have been employed to investigate the propagation mechanism of computer viruses. In particular, node-based models have been recently attracting great attention because many real-world networks have been shown to have a highly structured property [2]. Mieghem et al. [3] proposed the first node-based model to investigate the impact of network structure on the virus propagation process. As far as we know, some researchers have made some achievements in studying the incubation period of computer viruses [4,5]. However, most existing studies have assumed that nodes in the incubation period are not contagious, which is not true in many real-world cases [6]. To the best of our knowledge, no existing result considers the computer virus spread with incubation period using a node-based approach.

This study models the incubation period as a time delay and investigates the impacts of network structure and incubation period on the virus propagation process. Two intervention strategies are proposed to guarantee cybersecurity of computer nodes in the incubation period. Specifically, a continuous intervention strategy and an intermittent intervention strategy are proposed to ensure that a node in the incubation period or infective state can transfer to a susceptible node ultimately. Additionally, the stability of the virus-free equilibrium is analyzed and some sufficient conditions associated with the incubation interval and network topology are provided.

Notations. For a vector a, a^{T} denotes its transpose, while $\|a\|$ denotes its Euclidean norm. $\|M\|_2$ denotes the spectral norm of a matrix M. I_n is the $n \times n$ identity matrix. The real and imaginary parts of a complex number are denoted as $\mathrm{Re}(\cdot)$ and $\mathrm{Im}(\cdot)$, respectively. A graph $G = (V, E)$ with n nodes denotes the propagation network of a computer virus and its adjacency matrix is given by $A = [a_{ij}] \in \mathbb{R}^{n \times n}$, where the element $a_{ij} = 1$ if there is a link between nodes i and j. Otherwise, $a_{ij} = 0$.

Problem formulation. Let S, L and I denote the susceptible, latent, and infected state of a node, respectively. Figure 1(a) shows the transmission process of the three states, where $p_i(t)$ and $q_i(t)$ denote the probability of node i in states I and L at time t, respectively. Then, the expression $s_i(t) = 1 - p_i(t) - q_i(t)$ denotes the probability of node i in state S. In Figure 1(a), the constants β_i^I and β_i^L denote the infection probability of a susceptible node becoming an infective state through contact with infective and latent nodes, respectively. δ_i denotes the probability of an infective node curing or self-curing to a susceptible node. In this study, we assume that every latent node can become an infective node in a sponta-

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neous manner with a probability 1 when $t > \tau$, while the probability becomes α when $0 < t < \tau$. Here, α is called the control rate of our intervention strategies in sequel. Without loss of generality, it is assumed that all state transition processes are independent. For simplification, we assume the infection rates β_i^I, β_i^L and the curing rate δ_i are homogeneous for all nodes, i.e., $\beta_i^I = \beta_I$, $\beta_i^L = \beta_L$ and $\delta_i = \delta$, $\forall i \in \{1, \ldots, n\}$.
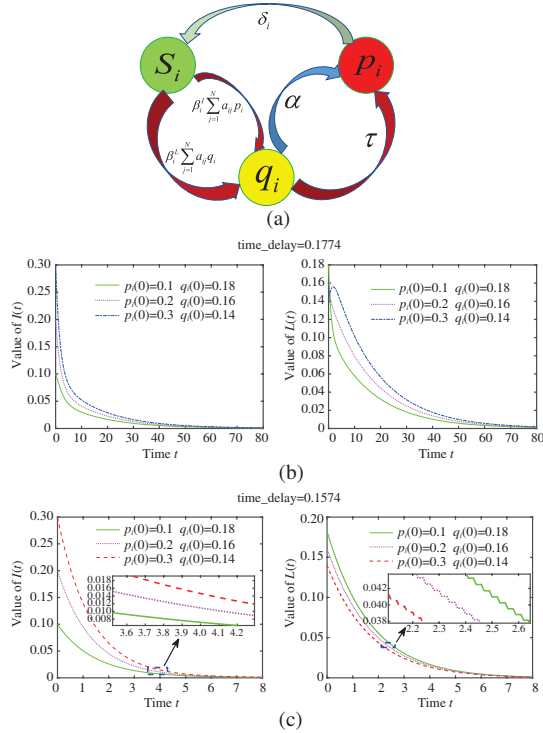
Figure 1 (Color online) Individual infection process (a); the evolution of the average densities of $q_i(t)$ and $p_i(t)$ with model (1) (b) and model (2) (c).

First, according to the propagation process mechanism illustrated in Figure 1(a) and the N-intertwined mean-field approximation approach [3], we establish a novel node-based susceptible-latent-infected-susceptible (SLIS) model with a continuous intervention strategy as follows:

$$\begin{cases} \dfrac{\mathrm{d}p_i(t)}{\mathrm{d}t} = q_i(t-\tau) - \delta p_i(t) + (1-\delta)\alpha q_i(t), \\ \dfrac{\mathrm{d}q_i(t)}{\mathrm{d}t} = s_i(t)\left[\beta_I \sum_{j=1}^{n} a_{ij}p_i(t) + \beta_L \sum_{j=1}^{n} a_{ij}q_i(t)\right] \\ \qquad\qquad - q_i(t-\tau) - \alpha q_i(t), \end{cases} \quad (1)$$

where the initial states $p_i(0) \in [0,1]$, $q_i(0) \in [0,1]$, for $i \in (1, \ldots, n)$. Furthermore, we propose an intermittent intervention strategy, which is updated periodically at time instants $t_k \in \{kT, k \in \mathbb{N}\}$ with a constant period T. Thus, the modified SLIS

model is formulated as follows:

$$\begin{cases} \dfrac{\mathrm{d}p_i(t)}{\mathrm{d}t} = q_i(t-\tau) - \delta p_i(t) + (1-\delta)\alpha q_i(t)\delta_k, \\ \dfrac{\mathrm{d}q_i(t)}{\mathrm{d}t} = s_i(t)\left[\beta_I \sum_{j=1}^{n} a_{ij}p_i(t) + \beta_L \sum_{j=1}^{n} a_{ij}q_i(t)\right] \\ \qquad\qquad - q_i(t-\tau) - \alpha q_i(t)\delta_k, \end{cases} \quad (2)$$

where $\delta_k = \delta(t - t_k)$, $\delta(t)$ is the Dirac delta function.

Main results. For convenience of stability analysis, we adopted the following notations:

$$\bar{A}_I = \begin{bmatrix} -\delta I_n & (1-\delta)\alpha I_n \\ \beta A & \beta_L A - \alpha I_n \end{bmatrix}, \quad B = \begin{bmatrix} 0 & I_n \\ 0 & -I_n \end{bmatrix},$$

$$\bar{A}_{II} = \begin{bmatrix} (1-\delta) I_n & 0 \\ \beta A & \beta_L A + I_n \end{bmatrix}, \quad C = \begin{bmatrix} 0 & (1-\delta)\alpha I_n \\ 0 & -\alpha I_n \end{bmatrix}. \quad (3)$$

Thus, the system (1) can be written in compact form as follows:

$$\dot{x}(t) = \bar{A}_I x(t) + B x(t-\tau), \quad (4)$$

while the system (2) can rewritten as follows:

$$\begin{cases} \dot{x}(t) = \bar{A}_{II} x(t) + B x(t-\tau), \\ x(t_k) = \hat{A} x(t_k^-) + B x(t_k^- - \tau), \end{cases} \quad t \neq t_k, \quad (5)$$

where $x = [\, p_1 \cdots p_n \; q_1 \cdots q_1 \,]^{\mathrm{T}}$, $\hat{A} = \bar{A}_{II} + C$, $\bar{A}_I$, $\bar{A}_{II}$, B and C are defined in (3).

Lemma 1 plays a key role in the stability of the SLIS system (4).

Lemma 1. For system (1), if it follows the below condition:

$$a > 0, \quad abd + a^2 c - d^2 > 0, \quad (6)$$

where

$$\begin{cases} a = 1 + \alpha + \delta - \beta_L \mathrm{Re}\mu_i, \; b = -\beta_L \mathrm{Im}\mu_i, \\ c = \alpha\delta + \delta - (\alpha\beta_I + \alpha\delta\beta_I + \beta_I + \delta\beta_L)\mathrm{Re}\mu_i, \\ d = (-\alpha\beta_I - \alpha\delta\beta_I - \beta_I - \delta\beta_L)\mathrm{Im}\mu_i, \end{cases}$$

μ_i is the i-th eigenvalue of A, then there exists a positive definite matrix P such that

$$PG + G^{\mathrm{T}}P = -I_{2n}, \quad (7)$$

where $G = \bar{A}_I + B$.

The proof of Lemma 1 can be found in Appendixes A and B.

Now, we present the main result of system (4) with a continuous intervention strategy.

Theorem 1. For system (4), assume that there exists a positive constant τ^* such that

$$\tau < \tau^* = 1/\left\| PB\bar{A}_I P^{-1}\bar{A}_I^{\mathrm{T}}B^{\mathrm{T}}P \right. \\ \left. + PB^2 P^{-1}(B^{\mathrm{T}})^2 P + 2\epsilon P \right\|, \quad (8)$$

where P is defined by (7) and $\epsilon > 1$. Then, the virus-free equilibrium of (4) is asymptotically stable.

The proof of Theorem 1 can be found in Appendix B. Theorem 1 indicates that with appropriate propagation parameters satisfying the conditions (8), the virus will be eradicated in the cyber network.

Corollary 1. If the network topology of the cyber network is undirected, then the condition (6) of Lemma 1 can be further reduced to

$$1 + \alpha + \delta - \beta_L\mu_1 > 0,$$

$$\alpha\delta + \delta - (\alpha\beta_I + \alpha\delta\beta_I + \beta_I + \delta\beta_L)\mu_1 > 0,$$

where $\mu_{\max}$ is the maximum eigenvalue of A.

The proof of Corollary 1 can be found in Appendix B. For large-scale networks, it is both time-consuming and resource-intensive to solve Eq. (6). This corollary is important for determining whether a virus dies out on an undirected network and provides a quick solution scheme as well.

Another main result for the system (5) with an intermittent intervention strategy is presented as follows.

Theorem 2. When $\tau \leqslant d$, the virus-free equilibrium of (5) is exponentially stable, if there exists a positive constant H such that $\|\Phi(t,s)\| \leqslant He^{x_{t_0}(t-s)}$, $H\|B\| + x_{t_0} \geqslant 0$, $t \in [t_{k-1}, t_k]$, and $K, h > 0$ result in

$$\prod_{j=1}^{k} H_j e^{\gamma(t-t_0)} \leqslant Ke^{-ht_{k+1}}, \quad (9)$$

where $k \in \mathbb{N}$, $x(t) \overset{\triangle}{=} x(t, t_0, x_{t_0})$ is the solution of (2), $x_{t_0} = [p_1(0)\cdots p_n(0)\; q_1(0)\cdots p_n(0)]^{\mathrm{T}}$ are the initial conditions of (2), $\gamma = H\|B\|_2 + x_{t_0}$, $H_j = H[l + d_j \max\{l,1\}]$, $d_j = \int_{t_j}^{t_j+d}\|B\|_2 e^{x_{t_0}(t_j-s)}\mathrm{d}s$, $\hat{A} = \bar{A}_{II} + C$, $l = \|\hat{A}\|_2 + \|B\|_2 e^{\gamma d}$ and $\Phi(t, t_0)$ is the fundamental matrix of system $\dot{x}(t) = \bar{A}_{II}x(t)$.

Numerical simulations. The cyber network is modeled using a scale-free Barabási-Albert network with a power-law degree distribution $p_\varrho = \omega\varrho^{-\iota}$. In our simulation, we take $n = 300$, $\omega = 2$, $\iota = 5$ and $\varrho = 4$.

Example 1. Consider system (1) with $\tau = 0.1774$, and $\beta_I = 0.04$, $\beta_L = 0.05$, $\alpha = 0.3$, $\delta = 0.3$. Matrix P can be obtained from (7) and $\tau^* = 0.1874$. Figure 1(b) shows that the computer virus die out under the proposed intervention strategy, which validates Theorem 1. Some other simulation results can be found in Appendix C.

Example 2. For the system (2), the propagation parameters are same as those considered in Example 1. Here, $\tau = 0.1574$. The period is taken as $T = 1$ and $\gamma = 0.2$. Figure 1(c) shows that the computer virus perish in the cyber network.

From Examples 1 and 2, we can observe that with the same initial conditions, the convergence rate of the closed-loop system with the pulse intervention strategy is faster than the convergence rate obtained under continuous intervention strategy. More results are presented in Appendix C.

Conclusion. We proposed a new SLIS model using a node-based approach to investigate the dynamic behavior of a computer virus. We proved that the virus-free equilibrium is asymptotically stable or exponentially stable under the two proposed intervention strategies. Our findings can serve as a reference to better understand the propagation mechanism of a computer virus with latent period and the impact of the cyber network structure on the computer virus propagation.

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