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A theoretical method for assessing disruptive computer viruses

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HIGHLIGHTS

- To assess the prevalence of disruptive malware, a new epidemic model is proposed.
- A criterion for the global stability of the virus-free equilibrium is given.
- A criterion for the existence of a unique viral equilibrium is presented.
- Some interesting results are drawn from extensive simulation experiments.
- On this basis, some policies of suppressing disruptive malware are recommended.

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ABSTRACT

To assess the prevalence of disruptive computer viruses in the situation that every node in a network has its own virus-related attributes, a heterogeneous epidemic model is proposed. A criterion for the global stability of the virus-free equilibrium and a criterion for the existence of a unique viral equilibrium are given, respectively. Furthermore, extensive simulation experiments are conducted, and some interesting phenomena are found from the experimental results. On this basis, some policies of suppressing disruptive viruses are recommended.

1. Introduction

The convenience and popularization of the Internet have brought huge benefits to human society. Meanwhile, it offers a shortcut to spread computer viruses, inflicting large economic losses [1]. Consequently, the problem of how to effectively suppress digital viruses has long been a hot spot in the field of network security research. The epidemic modeling of computer infections is recognized as a feasible approach to the assessment of prevalence of electronic viruses as well as that of effectiveness of different virus-containing strategies [2]. Since the seminal work by Kephart and White [3,4], multifarious computer virus spreading models, ranging from the coarsest population-level models [5–12] and the intermediate network-level models [13–17] to the finest node-level epidemic models, have been proposed [18–23].

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Disruptive computer virusese are defined as those whose life period consists of two consecutive phases: the latent phase and the disruptive phase. In the latent phase, a disruptive virus staying in a host does not perform any disruptive operations. Rather, the virus tries to infect as many hosts as possible by sending its copies to them. In the disruptive phase, a disruptive virus staying in a host performs a variety of operations that disrupt the host, such as distorting data, deleting data or files, and destroying the operating system. For example, the notorious Melissa virus propagates by means of emails, paralyzing the email server. As the second instance, the CIH virus propagates through the Internet and emails, destroying the BIOS of all infected hosts on some prescribed dates. To assess the prevalence of disruptive viruses, a number of epidemic models, which are referred to as the Susceptible–Latent–Bursting–Susceptible (SLBS) models, have been suggested [24–28], which have been extended towards different directions [29–34]. Recently, Yang et al. [35] established a node-level SLBS model, where all nodes have the same infecting rate, the same curing rate, and the same disruptive rate. In real-world applications, different nodes may enjoy different attributes and different safety levels. Therefore, they may have different infecting rates, different curing rates, and different bursting rates. In such scenarios, heterogeneous SLBS models may be more appropriate.

This paper addresses the issue of assessing the prevalence of disruptive computer viruses in the situation that every node in a network has its own virus-related attributes. For that purpose, a heterogeneous epidemic model is proposed. A criterion for the global stability of the virus-free equilibrium and a criterion for the existence of a unique viral equilibrium are given, respectively. Furthermore, extensive simulation experiments are conducted, and some interesting results are drawn from the experimental results. On this basis, some measures of suppressing disruptive malware are recommended.

The remaining materials of this work are organized in the following pattern. Section 2 formulates the new epidemic model. Section 3 theoretically studies this model, and Section 4 conducts extensive simulation experiments. This work is closed by Section 5.

2. The new model

Given a population of hosts (nodes) numbered $1, 2, \ldots, N$. Let $G = (V, E)$ be the virus-spreading network, where $V = \{1, 2, \ldots, N\}$, and $\{i, j\} \in E$ if and only if disruptive viruses can propagate between node i and node j. From now on, G is assumed to be unvaried and connected. Let $\mathbf{A} = \left(a_{ij}\right)_{N\times N}$ denote the adjacency matrix of G. Then $\mathbf{A}$ is irreducible.

As with the traditional SLBS models, it is assumed that at any time every node in the network is in one of three possible states: *susceptible*, *latent*, and *disruptive*; susceptible nodes are those that are not infected with viruses (but susceptible to them), latent nodes are those that are infected with viruses and all of them are in the latent phase, and disruptive nodes are those that are infected with viruses and some of them are in the disruptive phase. Let $X_i(t) = 0$, 1, and 2 denote that at time t node i is susceptible, latent, and disruptive, respectively. Let

$$S_i(t) = \Pr\{X_i(t) = 0\}, \qquad L_i(t) = \Pr\{X_i(t) = 1\}, \qquad B_i(t) = \Pr\{X_i(t) = 2\}.$$

As $S_i(t) + L_i(t) + B_i(t) \equiv 1$, $1 \le i \le N$, the vector

$$\mathbf{I}(t) = (L_1(t), \ldots, L_N(t), \qquad B_1(t), \ldots, B_N(t))^T$$

probabilistically captures the state of the population at time t. Let

$$\Omega = \left\{(L_1, \ldots, L_N, B_1, \ldots, B_N)^T \in \mathbb{R}_+^{2N} \mid L_i + B_i \le 1, i = 1, 2, \ldots, N\right\}.$$

Then $\mathbf{I}(t) \in \Omega$ for all $t \ge 0$.

We intend to study the propagating behavior of disruptive virus by employing the mean-field approximation technique. To this end, let us impose a set of statistical hypotheses on the state transition of each node as follows.

(H_1) Due to the infection by a neighboring latent node j, a susceptible node i becomes latent at constant rate $\beta_j^L > 0$.

(H_2) Due to the infection by a neighboring disruptive node j, a susceptible node i becomes latent at constant rate $\beta_j^B > 0$. As the major mission of a disruptive virus in the latent phase is to infect as many susceptible nodes as possible, whereas the major mission of a disruptive virus in the disruptive phase is to perform disruptive operations, we have $\beta_j^L > \beta_j^B$.

(H_3) Due to the outburst of disruptive viruses, a latent node i becomes disruptive at constant rate $\alpha_i > 0$.

(H_4) Due to the suppression by antivirus, a latent node i becomes susceptible at constant rate $\gamma_i^L > 0$, and a disruptive node i becomes susceptible at constant rate $\gamma_i^B > 0$. As a disruptive node has more chance to get treated than a latent node, we have $\gamma_i^B > \gamma_i^L$.

Fig. 1 shows these assumptions schematically.

Let Δt be a very small time interval. Hypotheses (H_1)–(H_4) imply that the probabilities of state transition of each node satisfy the following relations.

$$\begin{aligned}
\Pr\{X_i(t+\Delta t) = 1 \mid X_i(t) = 0\} &= \Delta t \sum_{j=1}^{N} a_{ij}\left[\beta_j^L L_j(t) + \beta_j^B B_j(t)\right] + o(\Delta t),\\
\Pr\{X_i(t+\Delta t) = 2 \mid X_i(t) = 0\} &= o(\Delta t),\\
\Pr\{X_i(t+\Delta t) = 0 \mid X_i(t) = 1\} &= \gamma_i^L \Delta t + o(\Delta t),\\
\Pr\{X_i(t+\Delta t) = 2 \mid X_i(t) = 1\} &= \alpha_i \Delta t + o(\Delta t),\\
\Pr\{X_i(t+\Delta t) = 0 \mid X_i(t) = 2\} &= \gamma_i^B \Delta t + o(\Delta t),\\
\Pr\{X_i(t+\Delta t) = 1 \mid X_i(t) = 2\} &= o(\Delta t).
\end{aligned}$$

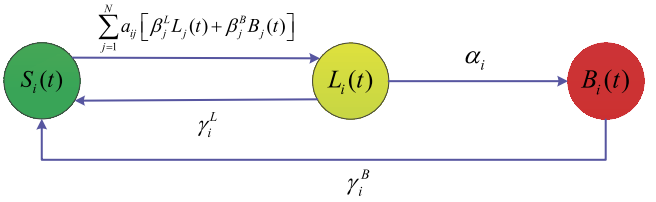

Fig. 1. Diagram of hypotheses (H_1)–(H_4).

As a result, we have

$$\begin{aligned}
\Pr\{X_i(t+\Delta t)=0 \mid X_i(t)=0\} &= 1-\Delta t\sum_{j=1}^{N} a_{ij}\left[\beta_j^L L_j(t)+\beta_j^B B_j(t)\right]+o(\Delta t),\\
\Pr\{X_i(t+\Delta t)=1 \mid X_i(t)=1\} &= 1-\gamma_i^L\Delta t-\alpha_i\Delta t+o(\Delta t),\\
\Pr\{X_i(t+\Delta t)=2 \mid X_i(t)=2\} &= 1-\gamma_i^B\Delta t+o(\Delta t).
\end{aligned}$$

It follows from the total probability formula that

$$\begin{aligned}
L_i(t+\Delta t) &= S_i(t)\cdot\Pr\{X_i(t+\Delta t)=1 \mid X_i(t)=0\}+L_i(t)\cdot\Pr\{X_i(t+\Delta t)=1 \mid X_i(t)=1\}\\
&\quad +B_i(t)\cdot\Pr\{X_i(t+\Delta t)=1 \mid X_i(t)=2\}\\
&= [1-L_i(t)-B_i(t)]\Delta t\sum_{j=1}^{N} a_{ij}\left[\beta_j^L L_j(t)+\beta_j^B B_j(t)\right]+L_i(t)\left[1-\gamma_i^L\Delta t-\alpha_i\Delta t\right]+o(\Delta t)
\end{aligned}$$

and

$$\begin{aligned}
B_i(t+\Delta t) &= S_i(t)\cdot\Pr\{X_i(t+\Delta t)=2 \mid X_i(t)=0\}+L_i(t)\cdot\Pr\{X_i(t+\Delta t)=2 \mid X_i(t)=1\}\\
&\quad +B_i(t)\cdot\Pr\{X_i(t+\Delta t)=2 \mid X_i(t)=2\}\\
&= \alpha_i L_i(t)\Delta t+B_i(t)\left[1-\gamma_i^B\Delta t\right]+o(\Delta t).
\end{aligned}$$

Rearranging the terms, dividing both sides by Δt, and letting $\Delta t\to 0$, we get the following dynamical model.

$$\begin{cases}
\dfrac{dL_i(t)}{dt} = [1-L_i(t)-B_i(t)]\displaystyle\sum_{j=1}^{N} a_{ij}\left[\beta_j^L L_j(t)+\beta_j^B B_j(t)\right]-\left(\gamma_i^L+\alpha_i\right)L_i(t), \quad i=1,2,\ldots,N,\\
\dfrac{dB_i(t)}{dt} = \alpha_i L_i(t)-\gamma_i^B B_i(t), \quad i=1,2,\ldots,N,
\end{cases} \tag{1}$$

where $\mathbf{I}(0)\in\Omega$. In what follows, we refer to the model as the *heterogeneous SLBS model*.

For the purpose of brevity, rewrite model (1) in matrix notation as

$$\frac{d\mathbf{I}(t)}{dt}=\mathbf{F}(\mathbf{I}(t)), \qquad \mathbf{I}(0)\in\Omega.$$

3. Analysis of the new model

This section addresses the dynamics of the heterogeneous SLBS model.

3.1. Preliminary knowledge

For fundamental matrix-theoretical knowledge, see Ref. [36]. For a square matrix $\mathbf{A}$, let $s(\mathbf{A})$ denote the maximum real part of an eigenvalue of $\mathbf{A}$, $s(\mathbf{A})$ the spectral radius of $\mathbf{A}$ (i.e., the maximum modulus of an eigenvalue of $\mathbf{A}$).

Lemma 1. *Let* $\mathbf{A}$ *be an irreducible nonnegative square matrix. Then* (a) $\rho(\mathbf{A})>0$, (b) $\rho(\mathbf{A})$ *is a simple eigenvalue of* $\mathbf{A}$, *and* (c) *there is a positive eigenvector belonging to* $\rho(\mathbf{A})$.

This lemma is referred to as the Perron–Frobenius Theorem, see Theorem 8.4.4 in [36].

Lemma 2. *Consider the smooth n-dimensional system of differential equations*

$$\frac{d\mathbf{x}(t)}{dt}=\mathbf{f}(\mathbf{x}(t)), \quad t\geq 0$$

and the corresponding system of differential inequalities

$$\frac{d\mathbf{y}(t)}{dt} \leq \mathbf{f}(\mathbf{y}(t)), \quad t \geq 0$$

with $\mathbf{x}(0) = \mathbf{y}(0)$. *Suppose*

$$f_i(x_1 + a_1, \ldots, x_{i-1} + a_{i-1}, x_i, x_{i+1} + a_{i+1}, \ldots, x_n + a_n) \geq f_i(x_1, \ldots, x_n), \quad i = 1, \ldots, n,\ a_1, \ldots, a_n \geq 0.$$

Then $\mathbf{y}(t) \leq \mathbf{x}(t)$, $t \geq 0$.

This lemma is referred to as the Chaplygin Lemma, see Theorem 31.4 in [37].

Lemma 3. *Let* $C \subset \mathbb{R}^n$ *be nonempty, bounded, closed, and convex. Let* $f : C \to C$ *be a continuous function. Then* f *has a fixed point.*

This lemma is referred to as the Brouwer Fixed Point Theorem, see Theorem 4.10 in [38].

3.2. Positive invariance

Lemma 4. *Ω is positively invariant for model (1). That is,* $\mathbf{I}(0) \in \Omega$ *implies* $\mathbf{I}(t) \in \Omega$ *for all* $t > 0$.

Proof. The boundary of Ω, denoted $\partial\Omega$, consists of the following $3N$ hyperplanes:

$$H_i = \{(L_1, \ldots, L_N, B_1, \ldots, B_N)^T \in \Omega \mid L_i = 0\}, \quad 1 \leq i \leq N,$$
$$H_{N+i} = \{(L_1, \ldots, L_N, B_1, \ldots, B_N)^T \in \Omega \mid B_i = 0\}, \quad 1 \leq i \leq N,$$
$$H_{2N+i} = \{(L_1, \ldots, L_N, B_1, \ldots, B_N)^T \in \Omega \mid L_i + B_i = 1\}, \quad 1 \leq i \leq N.$$

For $1 \leq i \leq N$, H_i, H_{N+i}, and H_{2N+i} have

$$\mathbf{n}_i = (0, \ldots, 0, \underbrace{-1}_{i}, 0, \ldots, 0)^T,$$

$$\mathbf{n}_{N+i} = (0, \ldots, 0, \underbrace{-1}_{N+i}, 0, \ldots, 0)^T,$$

and

$$\mathbf{n}_{2N+i} = (0, \ldots, 0, \underbrace{1}_{i}, 0, \ldots, 0, \underbrace{1}_{N+i}, 0, \ldots, 0)^T$$

as their respective outer normal vectors. Let $\hat{\mathbf{I}} = (\hat{L}_1, \ldots, \hat{L}_N, \hat{B}_1, \ldots, \hat{B}_N)^T$ be a smooth point of $\partial\Omega$. We distinguish among three possibilities.

Case 1: Some $\hat{L}_i = 0$. Then, $\langle \mathbf{F}(\hat{\mathbf{I}}), \mathbf{n}_i \rangle = -(1 - \hat{B}_i) \sum_j a_{ij} \left[\beta_j^L \hat{L}_j + \beta_j^B \hat{B}_j \right] \leq 0$.

Case 2: Some $\hat{B}_i = 0$. Then, $\langle \mathbf{F}(\hat{\mathbf{I}}), \mathbf{n}_{N+i} \rangle = -\alpha_i \hat{L}_i \leq 0$.

Case 3: Some $\hat{L}_i + \hat{B}_i = 1$. Then, $\langle \mathbf{F}(\hat{\mathbf{I}}), \mathbf{n}_{2N+i} \rangle = -\gamma_i^L \hat{L}_i - \gamma_i^B \hat{B}_i < 0$.

Combining the above discussions, we get that $\mathbf{F}(\hat{\mathbf{I}})$ is pointing to Ω. The claim follows.

The lemma can be explained as follows. $L_i(t)$ stands for the probability that node i is latent at time t, and $B_i(t)$ stands for the probability that node i is disruptive at time t. Hence, we have $0 \leq L_i(t) \leq 1$, $0 \leq B_i(t) \leq 1$, and $L_i(t) + B_i(t) \leq 1$ for all $t \geq 0$. That is, we have $\mathbf{I}(t) \in \Omega$ for all $t \geq 0$.

3.3. The virus-free equilibrium

A state of a differential dynamic system is an *equilibrium* if the orbit starting from the state is always in the state. Clearly, model (1) always admits the trivial equilibrium $\mathbf{E}_0 = (0, \ldots, 0)^T$. We shall refer to the equilibrium as the *virus-free equilibrium*.

Given a differential dynamic system. An equilibrium of the system is *asymptotically stable* if any orbit of the system that starts from near the equilibrium keeps close to it. An equilibrium of the system is *globally attracting* if all orbits of the system approach the equilibrium. An equilibrium of the system is *globally stable* if it is asymptotically stable and globally attracting simultaneously. This subsection aims to examine the global stability of $\mathbf{E}_0$. For that purpose, let $\mathbf{E}_N$ denote the identity matrix of order N, and let

$$\mathbf{B} = \mathbf{A} \cdot \operatorname{diag}\left(\beta_i^L\right), \qquad a = \min_{1 \leq i \leq N} \gamma_i^L. \tag{2}$$

Theorem 1. *Consider model (1). The virus-free equilibrium* $\mathbf{E}_0$ *is globally stable if* $s(\mathbf{B} - a\mathbf{E}_N) < 0$.

Proof. Let $I_i(t) = L_i(t) + B_i(t)$, $1 \le i \le N$. By calculations, we get

$$\begin{aligned}\frac{dI_i(t)}{dt} &= [1 - I_i(t)] \sum_{j=1}^{N} a_{ij}\beta_j^L I_j(t) - \gamma_i^L I_i(t) - [1 - I_i(t)] \sum_{j=1}^{N} a_{ij}\left(\beta_j^L - \beta_j^B\right) B_j(t) - \left(\gamma_i^B - \gamma_i^L\right) B_i(t) \\ &\le \sum_{j=1}^{N} a_{ij}\beta_j^L I_j(t) - aI_i(t), \quad i = 1, \ldots, N.\end{aligned}$$

Let $\mathbf{w}(t) = (w_1(t), \ldots, w_N(t))^T$ and consider the comparison system

$$\frac{dw_i(t)}{dt} = \sum_{j=1}^{N} a_{ij}\beta_j^L w_j(t) - aw_i(t), \quad i = 1, \ldots, N,$$

with $w_i(0) = I_i(0)$, $1 \le i \le N$. Rewrite the system in matrix notation as

$$\frac{d\mathbf{w}(t)}{dt} = (\mathbf{B} - a\mathbf{E}_N)\,\mathbf{w}(t).$$

As $s(\mathbf{B} - a\mathbf{E}_N) < 0$, it follows from the fundamental theory on linear differential system that $\mathbf{w}(t) \to \mathbf{0}$. By Lemma 2, we have $\mathbf{I}(t) \le \mathbf{w}(t)$ for all $t > 0$. Hence, $\mathbf{I}(t) \to \mathbf{0}$ as $t \to +\infty$.

Theorem 1 shows that it helps annihilate the disruptive computer viruses in a network to meet the condition in the theorem by deleting edges from the network or/and reducing the infecting rates or/and enhancing the curing rates

3.4. The viral equilibrium

If model (1) admits a nontrivial equilibrium, we shall refer to it as a *viral equilibrium*. This subsection addresses the existence of a viral equilibrium of model (1). For that purpose, let

$$\mathbf{C} = \mathbf{A} \cdot \operatorname{diag}\left(\beta_i^L + \frac{\alpha_i}{\gamma_i^B}\beta_i^B\right), \qquad b = \min_{1 \le i \le N}\left\{\gamma_i^L + \alpha_i\right\}. \tag{3}$$

Lemma 5. *Consider model (1) and suppose* $\rho(\mathbf{C}) > b$. *Define a continuous mapping* $\mathbf{H} = (h_1, \ldots, h_N) : (0, \infty)^N \to (0, 1)^N$ *as follows.*

$$h_i(\mathbf{x}) = \frac{\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)x_j}{\gamma_i^L + \alpha_i + \frac{\alpha_i + \gamma_i^B}{\gamma_i^B}\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)x_j}, \quad \mathbf{x} = (x_1, \ldots, x_N)^T \in (0, \infty)^N, \ 1 \le i \le N.$$

Then $\mathbf{H}$ *admits a unique fixed point.*

Proof. First, let us show the monotonicity of $\mathbf{H}$. Let $\mathbf{x}, \mathbf{y} \in (0, \infty)^N$, $\mathbf{x} \le \mathbf{y}$, i.e., $x_i \le y_i$, $1 \le i \le N$. Then,

$$\begin{aligned}h_i(\mathbf{x}) &= \frac{\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)x_j}{\gamma_i^L + \alpha_i + \frac{\alpha_i + \gamma_i^B}{\gamma_i^B}\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)x_j} \\ &\le \frac{\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)y_j}{\gamma_i^L + \alpha_i + \frac{\alpha_i + \gamma_i^B}{\gamma_i^B}\sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)y_j} = h_i(\mathbf{y}), \quad 1 \le i \le N,\end{aligned}$$

which implies $\mathbf{H}(\mathbf{x}) \le \mathbf{H}(\mathbf{y})$. The monotonicity of $\mathbf{H}$ is proven.

Next, let us show that $\mathbf{H}$ has a fixed point. It is well known that the connectedness of G implies the irreducibility of $\mathbf{A}$, which in turn implies the irreducibility of $\mathbf{C}$. According to Lemma 1, $\rho(\mathbf{C})$ is a simple eigenvalue of $\mathbf{C}$, and $\rho(\mathbf{C})$ has a positive eigenvector $\mathbf{v} = (v_1, \ldots, v_N)^T$. Let

$$\varepsilon_1 = \frac{1}{\rho(\mathbf{C})} \min_{1\le i\le N} \left\{ \frac{\left(\rho(\mathbf{C}) - \gamma_i^L - \alpha_i\right)\gamma_i^B}{\alpha_i + \gamma_i^B} \right\},$$
$$\varepsilon_2 = \frac{1}{\rho(\mathbf{C})} \max_{1\le i\le N} \left\{ \frac{\left(\rho(\mathbf{C}) - \gamma_i^L - \alpha_i\right)\gamma_i^B}{\alpha_i + \gamma_i^B} \right\}.$$

Then, $0 < \varepsilon_1 \le \varepsilon_2$. Thus,

$$h_i(\varepsilon_1\mathbf{v}) = \frac{\varepsilon_1 \sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) v_j}{\gamma_i^L + \alpha_i + \varepsilon_1 \frac{\alpha_i+\gamma_i^B}{\gamma_i^B} \sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) v_j}$$
$$= \frac{\varepsilon_1 v_i \rho(\mathbf{C})}{\gamma_i^L + \alpha_i + \varepsilon_1 \frac{\alpha_i+\gamma_i^B}{\gamma_i^B} v_i \rho(\mathbf{C})} \ge \varepsilon_1 v_i, \quad 1 \le i \le N,$$

which implies $\mathbf{H}(\varepsilon_1\mathbf{v}) \ge \varepsilon_1\mathbf{v}$. And

$$h_i(\varepsilon_2\mathbf{v}) = \frac{\varepsilon_2 \sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) v_j}{\gamma_i^L + \alpha_i + \varepsilon_2 \frac{\alpha_i+\gamma_i^B}{\gamma_i^B} \sum_{j=1}^{N} a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) v_j}$$
$$= \frac{\varepsilon_2 v_i \rho(\mathbf{C})}{\gamma_i^L + \alpha_i + \varepsilon_2 \frac{\alpha_i+\gamma_i^B}{\gamma_i^B} v_i \rho(\mathbf{C})} \le \varepsilon_2 v_i, \quad 1 \le i \le N,$$

which implies $\mathbf{H}(\varepsilon_2\mathbf{v}) \le \varepsilon_2\mathbf{v}$. It follows from the monotonicity of $\mathbf{H}$ that $\mathbf{H}|_K$, the restriction of $\mathbf{H}$ on the compact convex set

$$K = [\varepsilon_1 v_1, \varepsilon_2 v_1] \times [\varepsilon_1 v_2, \varepsilon_2 v_2] \times \cdots \times [\varepsilon_1 v_N, \varepsilon_2 v_N],$$

maps K to K. It follows from Lemma 3 that $\mathbf{H}$ has a fixed point in K, denoted $\mathbf{L}^* = (L_1^*, \ldots, L_N^*)^T$.

Finally, let us show that $\mathbf{L}^*$ is the unique fixed point of $\mathbf{H}$. On the contrary, suppose $\mathbf{H}$ has another fixed point $\mathbf{M}^* = (M_1^*, \ldots, M_N^*)$. Let

$$\theta = \max_i \left\{ \frac{L_i^*}{M_i^*} \right\}, \qquad i_0 = \arg\max_i \left\{ \frac{L_i^*}{M_i^*} \right\}.$$

Without loss of generality, we may assume $\theta > 1$, it follows that

$$L_{i_0}^* = h_{i_0}(\mathbf{L}^*) \le h_{i_0}(\theta\mathbf{M}^*) = \frac{\theta \sum_j a_{i_0 j}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) M_j^*}{\gamma_{i_0}^L + \alpha_{i_0} + \theta \frac{\alpha_{i_0}+\gamma_{i_0}^B}{\gamma_{i_0}^B} \sum_j a_{i_0 j}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) M_j^*}$$
$$< \theta \frac{\sum_j a_{i_0 j}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) M_j^*}{\gamma_{i_0}^L + \alpha_{i_0} + \frac{\alpha_{i_0}+\gamma_{i_0}^B}{\gamma_{i_0}^B} \sum_{j=1}^{N} a_{i_0 j}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right) M_j^*} = \theta h_{i_0}(\mathbf{M}^*) = \theta M_{i_0}^*,$$

which contradicts the assumption that $L_{i_0}^* = \theta M_{i_0}^*$. Hence, the fixed point is unique. The theorem is proven.

Theorem 2. *Model (1) admits a unique viral equilibrium if* $\rho(\mathbf{C}) > b$.

Proof. It is easily verified that $(L_1, \ldots, L_N, B_1, \ldots, B_N)^T$ is an equilibrium of model (2) if and only if

$$B_i = \frac{\alpha_i}{\gamma_i^B} L_i, \quad 1 \le i \le N,$$

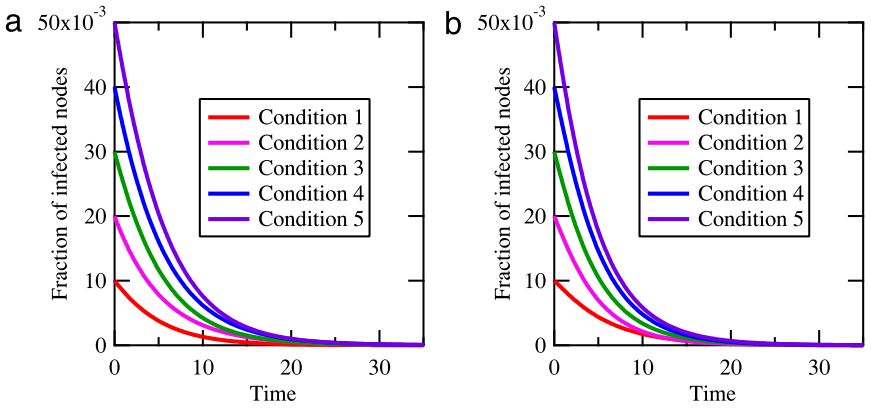

Fig. 2. The time plots of $I(t)$ for two models in the first class.

and

$$L_i = \frac{\sum_j a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_j^B}\beta_j^B\right)L_j}{\gamma_i^L + \alpha_i + \frac{\alpha_i+\gamma_i^B}{\gamma_i^B}\sum_j a_{ij}\left(\beta_j^L + \frac{\alpha_j}{\gamma_i^B}\beta_j^B\right)L_j}, \quad 1 \le i \le N.$$

The claim follows from Lemma 5.

We had tried our best to give a criterion for the local stability of the viral equilibrium, but failed, let alone a criterion for its global stability.

4. Simulation analysis

Let $I(t)$ denote the fraction of infected nodes at time t. That is,

$$I(t) = \frac{1}{N}\sum_{i=1}^{N}(L_i(t) + B_i(t)).$$

Although Theorem 1 gives a sufficient condition for the global stability of the virus-free equilibrium, and Theorem 2 offers a sufficient condition for the existence of a viral equilibrium, the following questions are yet to be answered.

Q_1 If the condition in Theorem 1 holds true, how fast does $I(t)$ approach zero?
Q_2 If the condition in Theorem 2 holds true, how about the dynamics of $I(t)$?
Q_3 If neither the condition in Theorem 1 nor that in Theorem 2 holds true, how about the dynamics of $I(t)$?

The aim of this section is to answer these questions by means of extensive computer simulations. The simulation experiments are carried out based on a synthetic scale-free network, a synthetic small-world network, and a real-world email network, respectively.

4.1. Simulations based on a scale-free network

Scale-free networks are a large class of networks having widespread applications [39]. Taking a randomly generated scale-free network with 100 nodes as the virus-spreading network, and taking 1575 random combinations of parameters satisfying hypotheses (H_1)–(H_4), we get 1575 different heterogeneous SLBS models. We categorize these models as three classes: the first class that consists of all models satisfying the condition in Theorem 1, the second class that consists of all models satisfying the condition in Theorem 2, and the third class that consists of all the remaining models. Numerical results reveal the following phenomena.

(a) There are 85 models in the first class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively fast speed, according with the result of Theorem 1. Fig. 2 shows five time plots of $I(t)$ for two models.
(b) There are 1446 models in the second class. Moreover, for each of the models, the fraction of infected nodes approaches a nonzero constant. Fig. 3 exhibits five time plots of $I(t)$ for two models.
(c) There are 44 models in the third class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively slow speed. Fig. 4 demonstrates five time plots of $I(t)$ for two models.

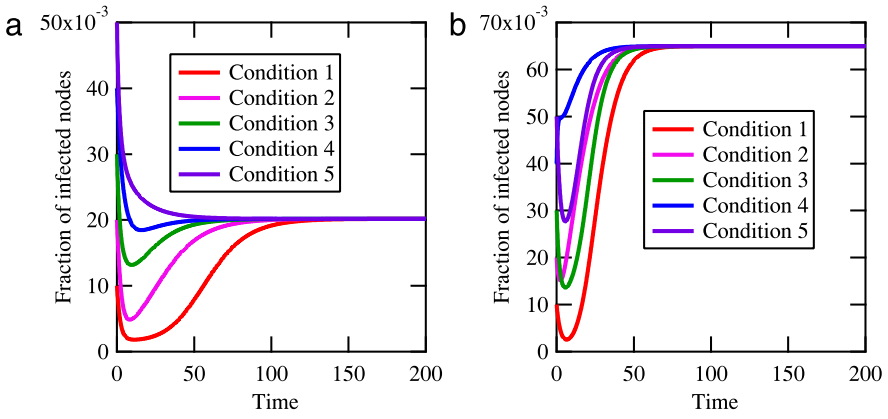

Fig. 3. The time plots of $I(t)$ for two models in the second class.

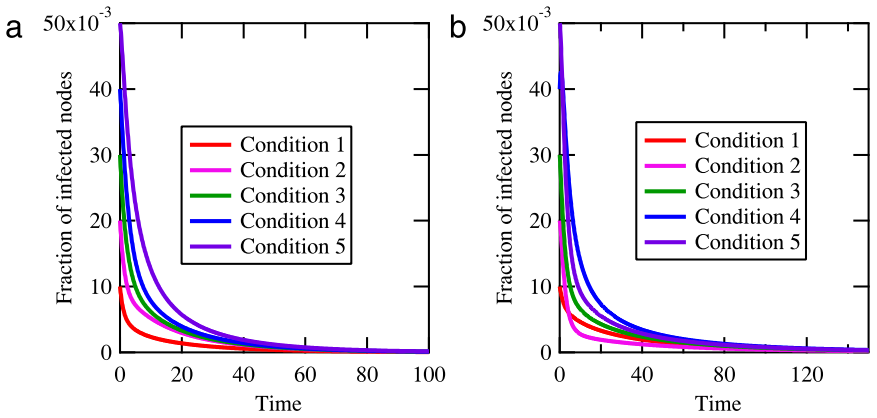

Fig. 4. The time plots of $I(t)$ for two models in the third class.

4.2. Simulations based on a small-world network

Small-world networks are another large class of networks having widespread applications [40]. Taking a randomly generated small-free network with 100 nodes as the virus-spreading network, and taking 1575 random combinations of parameters satisfying hypotheses (H_1)–(H_4), we get 1575 different heterogeneous SLBS models. Again, we categorize these models as three classes: the first class that consists of all models satisfying the condition in Theorem 1, the second class that consists of all models satisfying the condition in Theorem 2, and the third class that consists of all the remaining models. Numerical results reveal the following phenomena.

(a) There are 136 models in the first class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively fast speed, according with the result of Theorem 1. Fig. 5 shows five time plots of $I(t)$ for two models.
(b) There are 1322 models in the second class. Moreover, for each of the models, the fraction of infected nodes approaches a nonzero constant, which is dependent upon the initial condition. Fig. 6 exhibits five time plots of $I(t)$ for two models.
(c) There are 44 models in the third class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively slow speed. Fig. 7 demonstrates five time plots of $I(t)$ for two models.

4.3. Simulations based on a real-world email network

Consider the email network at the University Rovira i Virgili in Tarragona in the south of Catalonia in Spain, which has 1133 nodes and 5451 edges [41]. Taking the network as the virus-spreading network, and taking 1575 random combinations of parameters satisfying hypotheses (H_1)–(H_4), we get 1575 different heterogeneous SLBS models. Again, we categorize these models as three classes: the first class that consists of all models satisfying the condition in Theorem 1, the second class that consists of all models satisfying the condition in Theorem 2, and the third class that consists of all the remaining models. Numerical results reveal the following phenomena (see Fig. 7).

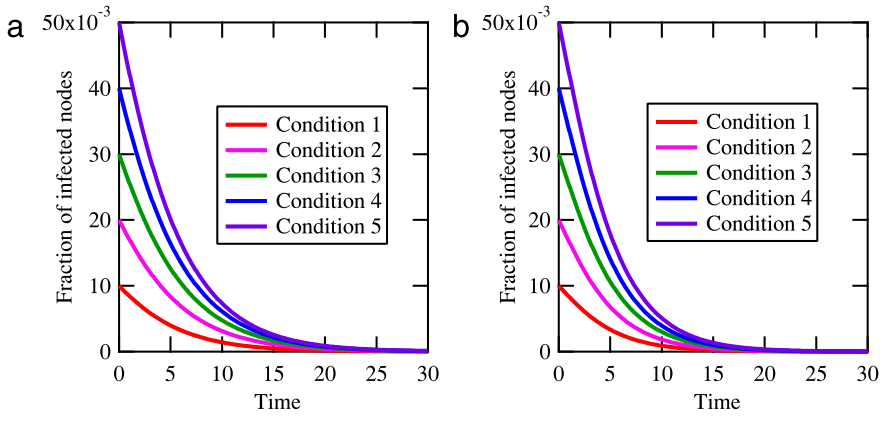

Fig. 5. The time plots of $I(t)$ for two models in the first class.

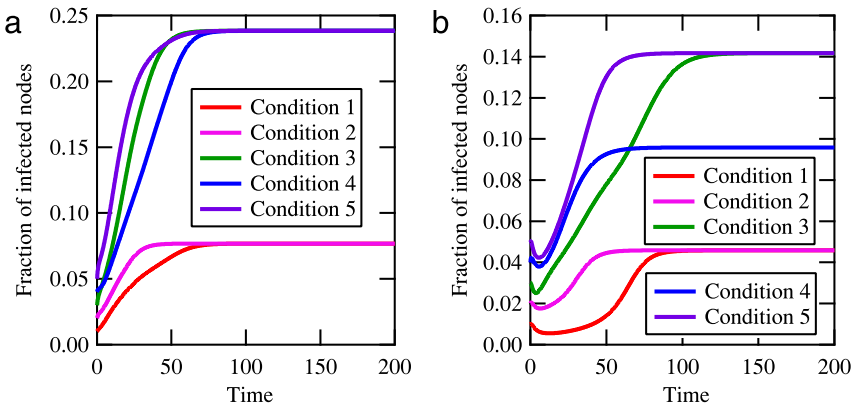

Fig. 6. The time plots of $I(t)$ for two models in the second class.

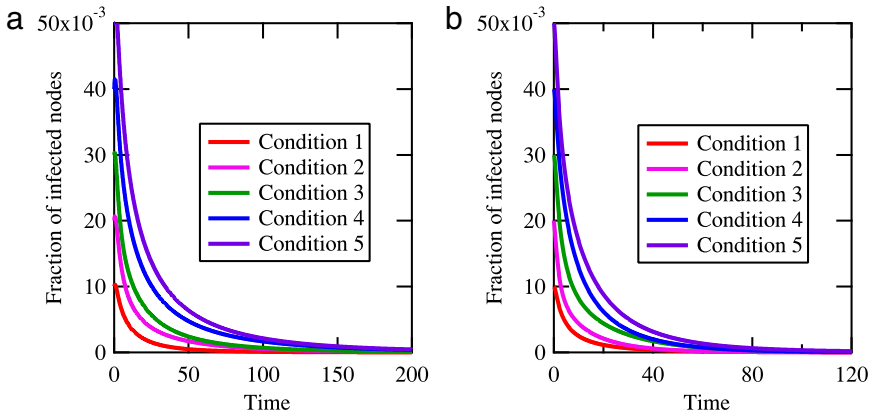

Fig. 7. The time plots of $I(t)$ for two models in the third class.

(a) There are 50 models in the first class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively fast speed. Fig. 8 shows five time plots of $I(t)$ for two models.

(b) There are 1501 models in the second class. Moreover, for each of the models, the fraction of infected nodes approaches a nonzero constant, which is dependent upon the initial condition. Fig. 9 exhibits five time plots of $I(t)$ for two models.

(c) There are 24 models in the third class. Moreover, for each of the models, the fraction of infected nodes approaches zero at a relatively slow speed. Fig. 10 demonstrates five time plots of $I(t)$ for two models.

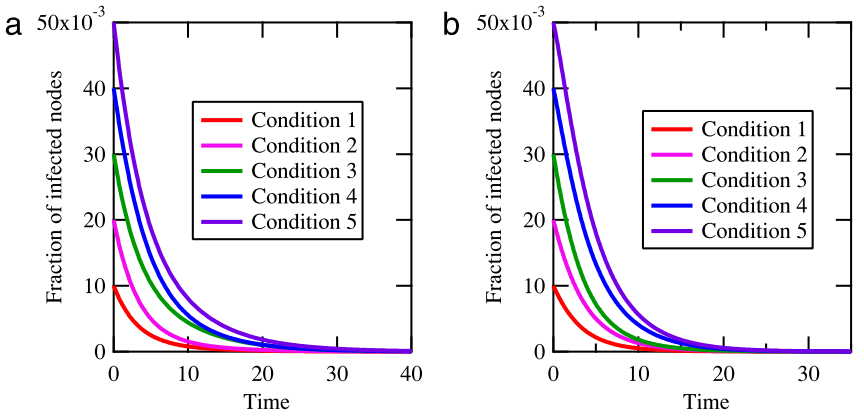

Fig. 8. The time plots of $I(t)$ for two models in the first class.

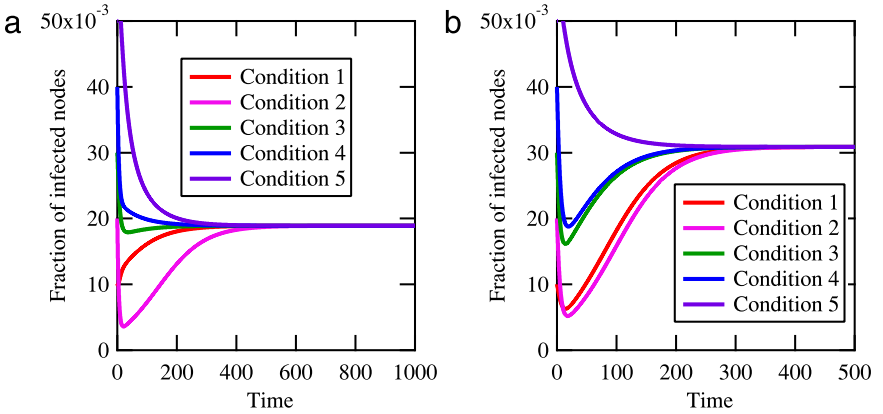

Fig. 9. The time plots of $I(t)$ for two models in the second class.

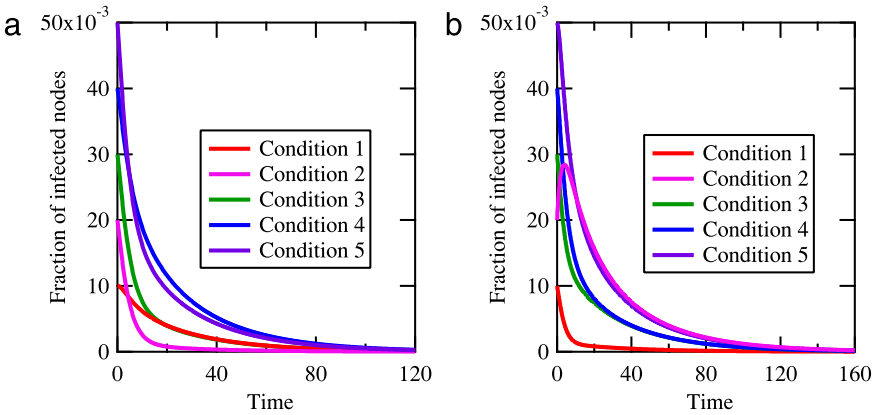

Fig. 10. The time plots of $I(t)$ for two models in the third class.

4.4. Brief summary

Based on the previous experimental results, we conclude the following results.

(a) For each heterogeneous SLBS model satisfying the condition in Theorem 1, the fraction of infected nodes approaches zero at a relatively fast speed.
(b) For each heterogeneous SLBS model satisfying the condition in Theorem 2, the fraction of infected nodes approaches a nonzero zero constant, which may be dependent upon the initial condition, unless initially there is no infected node.
(c) For each heterogeneous SLBS model satisfying neither the condition in Theorem 1 nor that in Theorem 2, the fraction of infected nodes approaches zero at a relatively slow speed.

The following strategies are recommended to control disruptive computer viruses.

(a) Delete some edges of the virus-spreading network.
(b) Reduce the infecting/disruptive rates of viruses.
(c) Enhance the curing rates.

5. Conclusions and remarks

For the purpose of assessing the prevalence of disruptive computer viruses, a heterogeneous node-level SLBS model has been proposed. A criterion for the global stability of the virus-free equilibrium has been given, and a criterion for the existence of a unique viral equilibrium has been presented. Furthermore, extensive simulation experiments have been conducted, and thereby some interesting results have been concluded. On this basis, some measures of containing the prevalence of disruptive malware have been suggested.

Towards this direction, numerous work has yet to be done. First, the cost-effective strategies of suppressing disruptive computer viruses should be studied [42–45]. Second, some key factors such as pulse should be incorporated in the proposed model [46,47]. Third, it is worthwhile to extend this work to time-varying networks [48–50]. Last, but not least, the methodology developed in this work can be extended to some other types of malicious epidemics such as the rumor spreading [51–55].

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