Controlling Virus Spread Using an Intermittent Quarantine Strategy on Multiplex Networks

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1. Introduction

Computer viruses are spreading widely on the Internet, thereby significantly affecting the cyberspace security [1–4]. For instance, the WannaCry virus, one of the most influential viruses since Panda Burned Incense, has affected more than 100 countries and regions. Researchers from different fields, including computer science, network science, physics, and mathematics, have attempted to eliminate the spread of the virus [5–11]. In this study, we focused on network science to address this problem, in which the ultimate aim is to develop effective measures to control virus spreading using mathematical modeling and network theory.

Network science researchers have developed many effective models to investigate the spread of viruses on the Internet. Data analyses revealed that the topology of the Internet exhibits the heavy-tail degree distribution [12–14]; hence, Pastor-Csatornas and Vespignani proposed a mathematical susceptible-infected-susceptible (SIS) model on scale-free complex networks [5, 15]. They used a heterogeneous mean-field theory to describe the dynamics and revealed that a few hubs resulted in vanishingly low values of infection transmission probability that triggered virus spreading on the Internet. Based on this finding, we can design an effective approach to control virus spread. Target immunization [15–19] is one of the most popular strategies, which involve the immunization of hubs to suppress viruses significantly. To implement the target immunization strategy, the topology of the Internet must be determined, which limits its applications. Therefore, researchers proposed the acquaintance immunization strategy [20–23], which necessitates only information regarding local network structures. The progress in state-of-the-art network immunization has been reported in a recent review [24].

Computer users typically communicate with their friends through social networks [25–28]. Hence, the information spread by a virus will diffuse in the social network when the virus is spreading on the Internet. Therefore, researchers have developed models to describe the dynamics of virus-information spread. Scholars typically use epidemic spreading models to model the spread of viruses, such as SIS and susceptible-infected-removed (SIR) models. Therefore,
we herein present some of the advancements in the field of epidemic-information spreading dynamics that can be used to investigate the spread of virus information. Funk et al. [29] assume that an epidemic is spread on homogeneous networks, whereas information regarding the epidemic is also spread within the same network. Using a mean-field approach, they discovered that information spreading can significantly suppress the spread of an epidemic but cannot alter the outbreak threshold. Granell et al. [30, 31] used the unaware-aware-unaware (UAU)-SIS model, which assumes UAU information spreading dynamics on social networks and SIS epidemic spreading dynamics on face-to-face contact networks. They used a discrete Markovian approach to describe the interacting dynamics and revealed that the epidemic outbreak threshold was determined by the topologies of social networks, contact networks, and dynamic information spreading parameters. Subsequently, researchers investigated the effects of network topologies and interacting mechanisms on spreading dynamics [32–37]. Wang et al. [38–40] used an asymmetrical interacting SIR-SIRV model and discovered that the interlayer degree correlation facilitated the control of epidemic spread.

To immunize virus spreading on the Internet, different strategies can be adopted, e.g., barring Internet access, implementing patches, and turning on the firewall. Additionally, advanced information-epidemic spreading dynamics can be adopted to describe more realistic scenarios. Zhao et al. [41] developed a virus-information spreading model to design an optimal allocation strategy for patches. Once a virus spreads on the Internet, the simplest approach to stop the spreading is to bar Internet access until the virus is eliminated [42–44]. Because mathematical studies pertaining to information spreading dynamics are scarce, we herein propose a mathematical model to describe an interaction model based on multiplex networks. We assume that information regarding the virus spreads in the social network, i.e., the virus spreads on the Internet and then performs extensive numerical simulations to determine the optimal intervention strength.

2. Model Descriptions

In this section, an interacting spreading dynamics model based on the intermittent quarantine immunization strategy on multiplex networks with \( N_A = N_B = N \) nodes is described. The multiplex networks comprise two subnetworks, denoted as \( A \) and \( B \), and each subnetwork represents a communication platform. Each node in two subnetworks is matched one-to-one, which implies that a node exists in different communication platforms. The two subnetworks were constructed based on an uncorrelated configuration model with degree distributions \( P_A(k_A) \) and \( P_B(k_B) \), separately. When the network size is \( N \rightarrow \infty \), no inter- and interlayer degree correlations exist in the multiplex subnetwork (as shown in Figure 1(a)).

Assume that subnetworks \( A \) and \( B \) represent the social and virus networks, respectively. Information is typically spread on social network \( A \), whereas virus spreads on subnetwork \( B \). To describe information spreading, we used the classic SIR epidemiological model [45–47], in which each node can be susceptible, informed, or recovered at a specified time. At each time step, each informed node first transmits the information to every susceptible neighbor with probability \( \beta_A \) and then recovers with probability \( \gamma_A \). It is noteworthy that the susceptible node in subnetwork \( A \) is informed once its counterpart in subnetwork \( B \) becomes infected. Hence, virus spread promotes information spreading (as shown in Figure 1(b)).

For virus spreading on subnetwork \( B \), we used a generalized SIR model. The virus spreading dynamics is the same as the information spreading dynamics on subnetwork \( A \) but with different infection and recovery probabilities, i.e., \( \beta_B \) and \( \gamma_B \), respectively. We assume that the susceptible node in subnetwork \( B \) adopts an intermittent quarantine strategy to control virus spread. Specifically, the susceptible node \( i_B \) in subnetwork \( B \) will be quarantined for a period \( t_q \). If the corresponding node \( i_A \) in subnetwork \( A \) is in the informed state. After period \( t_q \), the susceptible node \( i_B \) is reconnected to its neighbors, i.e., the intermittent quarantine strategy is induced (as shown in Figure 1(c)). From the above descriptions, we know the state of the node \( i_A \) depends on the information spreading and thus further determines the adoption intermittent quarantine strategy for the node \( i_A \). Hence, virus spreading on subnetwork \( B \) is suppressed. According to the above descriptions, the differences between the information diffusion and virus spreading are listed as follows: (i) different dynamical parameters, i.e., transmission and recovery probabilities, and (ii) an additional intermittent quarantine state is induced in the virus spreading.

We randomly selected one node as the virus seed in subnetwork \( B \), subsequently, we set its counterpart in subnetwork \( A \) as the information seed. The remaining nodes were set in a susceptible state. The effective information transmission and virus transmission probabilities are expressed as \( \lambda_A = \beta_A/\gamma_A \) and \( \lambda_B = \beta_B/\gamma_B \), respectively. The interacting spreading dynamics terminate once no node is available in the informed or infected state. In Table 1, we illustrate the definitions of the parameters in our model.

3. Results

The results of this study are presented in this section.

3.1. Theoretical Analysis. To analyze the results of this study quantitatively, we used a heterogeneous mean-field approach with time delays. We denote \( S^A(t) \), \( I^A(t) \), and \( R^A(t) \) as the fractions of the susceptible, informed, and recovered nodes with degree \( k_A \) in subnetwork \( A \) at time \( t \), respectively. Similarly, we denote \( S^B(t) \), \( I^B(t) \), \( R^B(t) \), and \( V^B(t) \) to represent the fraction of nodes in the susceptible, infected, recovered, and vaccinated states with degree \( k_B \) in subnetwork \( B \) at time \( t \), respectively. Once the parameters above are obtained, the fraction of nodes in each state can be determined using the degree distributions of the two networks, denoted as \( P_A(k_A) \) and \( P_B(k_B) \), separately. For instance, the fraction of nodes in the susceptible state in subnetwork \( A \) is \( S_A(t) = \sum_{k_A} S^A_{k_A}(t)P_A(k_A) \).
Inspired by [38, 39], we developed a heterogeneous mean-field approach to investigate virus-information spreading dynamics. For the information spreading dynamics on subnetwork $A$, a susceptible node (e.g., node $i_A$) becomes infected via one of two approaches. (i) Node $i_A$ can receive the information from its infected neighbors with probability $s^A_{k_A} (t)\beta_A k_A \Theta_A (t)$, where $\Theta_A (t)$ is the probability that node $i_A$ is connected to an infected neighbor. For uncorrelated networks, $\Theta_A (t)$ is expressed as

$$\Theta_A (t) = \frac{\sum k_A (k_A' - 1) P_A (k_A') \rho^A_{k_A} (t)}{\langle k_A \rangle},$$

(1)

where $\langle k_A \rangle = \sum k_A P_A (k_A)$ is the average degree of subnetwork $A$. (ii) Node $i_A$ receives the infection once its corresponding node $i_B$ becomes infected, and the infection probability is $s^A_{k_A} (t)\beta_A \rho^B_{k_B} (t)\delta_{k_A} (k_B)$, where $\Theta_B (t)$ is the probability that a susceptible node is connected to an infected neighbor in subnetwork $B$. $\Theta_B (t)$ is expressed as

$$\Theta_B (t) = \frac{\sum k_B (k_B' - 1) P_B (k_B') \rho^B_{k_B} (t)}{\langle k_B \rangle},$$

(2)

where $\langle k_B \rangle = \sum k_B P_B (k_B)$ is the average degree of subnetwork $B$. Combining the two approaches above, the evolution of $S^A_{k_A} (t)$ can be expressed as follows:

$$\frac{dS^A_{k_A} (t)}{dt} = -S^A_{k_A} (t) \left[ \beta_A k_A \Theta_A (t) + \beta_B \Theta_B (t) \sum k_B P_B (k_B) \right].$$

(3)

Once the susceptible node is infected by the information, it is regarded as infected. Hence, the evolution of $I^A_{k_A} (t)$ is expressed as

$$\frac{dI^A_{k_A} (t)}{dt} = S^A_{k_A} (t) \left[ \gamma_A + \gamma_B \sum k_B P_B (k_B) \right].$$

Table 1: Definitions of parameters and abbreviations.

| Parameters/abbreviations | Definitions |
|--------------------------|-------------|
| $\beta_A$                | Information transmission rate |
| $\beta_B$                | Virus transmission rate |
| $\gamma_A$               | Information recovery rate |
| $\gamma_B$               | Virus recovery rate |
| $t_B$                    | Intermittent quarantine period |
| $\langle k_A \rangle$    | Average degree of subnetwork $A$ |
| $\langle k_B \rangle$    | Average degree of subnetwork $B$ |
| $\Theta_A (t)$           | The probability of a node connects to an infected neighbor in network $A$ |
| $\Theta_B (t)$           | The probability of a node connects to an infected neighbor in network $B$ |
| $S^A_{k_A} (t)$          | A node with degree $k_A$ in network $A$ is in the susceptible state |
| $S^B_{k_B} (t)$          | A node with degree $k_B$ in network $B$ is in the susceptible state |
| $I^A_{k_A} (t)$          | A node with degree $k_A$ in network $A$ is in the infected state |
| $I^B_{k_B} (t)$          | A node with degree $k_B$ in network $B$ is in the informed state |
| $R^A_{k_A} (t)$          | A node with degree $k_A$ in network $A$ is in the recovered state |
| $R^B_{k_B} (t)$          | A node with degree $k_B$ in network $B$ is in the recovered state |
\[ \frac{dI_B^A(t)}{dt} = s_{k_A}(t) \left[ \beta_A k_A \Theta_A(t) + \beta_B \Theta_B(t) \sum_{k_B} k_B P_B(k_B) \right] - \gamma_A I_B^A(t). \] (4)

where \( \gamma_A I_B^A(t) \) is the fraction of nodes recovered from the infected state. Therefore, the evolution of \( R_{k_A}(t) \) is expressed as

\[ \frac{dR_{k_A}(t)}{dt} = \gamma_A I_B^A(t). \] (5)

For virus spreading on subnetwork \( B \), the situation becomes more complex. A susceptible node \( i_B \) may be quarantined for a period \( t_b \) if its corresponding node \( i_A \) becomes the infected state. Mathematically, we use a new state \( V \) to represent the quarantined nodes. We use \( V_{k_A}(t) \) to denote the fraction of nodes with degree \( k_A \) in the quarantined state. The fraction of nodes becomes a quarantined node experiences more time steps than \( t_b \). Hence, for a randomly selected susceptible node, it becomes an infected node with probability \( S_{k_B}^B(t) \beta_B k_B \Theta_B(t) \). The evolution of \( S_{k_B}^B(t) \) is expressed as

\[ \frac{dS_{k_B}^B(t)}{dt} = -S_{k_B}^B(t) \beta_B k_B \Theta_B(t) - p \beta_A \Theta_A(t) \sum_{k_A} S_{k_A}^B(t) k_A P_A(k_A). \] (6)

The evolution of \( V_{k_B}^B(t) \) is

\[ \frac{dV_{k_B}^B(t)}{dt} = p \beta_A \Theta_A(t) \sum_{k_A} S_{k_A}^B(t) k_A P_A(k_A). \] (7)

The evolution of \( I_{k_A}(t) \) occurs in one of two processes: (i) the susceptible nodes become infected by their infected neighbors with probability \( S_{k_B}^B(t) \beta_B k_B \Theta_B(t) \) and (ii) the quarantined node experiences more steps than \( t_b \) and is infected by neighbors with probability \( \sum_{t \neq t_b} V_{k_B}^B(t - t') \beta_B k_B \Theta_B(t) \). Therefore, the evolution of \( I_{k_B}^B(t) \) is expressed as

\[ \frac{dI_{k_B}^B(t)}{dt} = S_{k_B}^B(t) \beta_B k_B \Theta_B(t) + \sum_{t \neq t_b} V_{k_B}^B(t - t') \beta_B k_B \Theta_B(t) - \gamma_B I_{k_B}^B(t). \] (8)

The evolution of \( R_{k_B}(t) \) is

\[ \frac{dR_{k_B}(t)}{dt} = \gamma_B I_{k_B}^B(t). \] (9)

3.2. Numerical Simulations. We performed extensive Monte Carlo simulations on uncorrelated configuration networks using the method proposed in [48]. Specifically, we generated an uncorrelated configuration network as follows. (i) The network size was determined based on the average degree and degree distributions of the two subnetworks. We set the network sizes of subnetworks \( A \) and \( B \) as

\[ N_A = N_B = 10^4. \]

The degree distributions of the two networks were set based on a power law as \( P_A(k_A) = \xi_A^k k_A^{-\alpha_A} \) and \( P_B(k_B) = \xi_B^k k_B^{-\alpha_B} \), respectively, where \( \gamma_A \) and \( \gamma_B \) are the degree exponents of the two subnetworks, separately: \( \xi_A = \sum k_A^{\alpha_A} \) and \( \xi_B = \sum k_B^{\alpha_B} \). In the numerical simulations, we set \( \gamma_D = \gamma_D = 3.0 \) and the recovery probability \( \gamma_A = \gamma_B = 0.1 \). The multiplex networks are denoted as SFCSF multiplex networks. (ii) Degree sequences were generated for each subnetwork based on the degree distributions, and stubs were assigned for each node. (iii) For each, two stubs were selected randomly, and an edge was built until no stubs remained in the subnetwork. (iv) For each node in the two subnetworks, we performed one-to-one matching randomly.

We first investigated the final information spreading size \( R_A \) and the virus spreading size \( R_B \) for a specified quarantine value for period \( t_b \), as shown in Figure 2. For a specified value of \( \lambda_B \), \( R_B \) increased monotonically with \( \lambda_A \) (see Figure 2(a)) because the nodes in subnetwork \( A \) had a higher probability of accessing the information. In addition, \( R_A \) increased with \( \lambda_B \) because the spread of the virus promoted information spreading, as shown in Figures 2(a) and 2(b). In general, the virus spreading size \( R_B \) increased with \( \lambda_B \) for a specified \( \lambda_A \). However, we discovered an optimal information transmission probability for which the virus spreading suppressed significantly, as illustrated in Figures 2(c) and 2(d). Specifically, \( R_B \) first decreased with \( \lambda_A \) because the virus spread was sufficiently large, those nodes would be protected from being infected by the virus. Otherwise, the quarantined nodes would participate in virus spreading. Therefore, \( R_B \) increases with \( \lambda_B \). It is noteworthy that when \( \lambda_A \) is sufficiently large, many nodes are quarantined, and only a fraction of quarantined nodes will participate in virus spreading when their quarantine period is completed. Hence, \( R_B \) decreases with \( \lambda_A \).

We further investigated the effects of the quarantine period on the virus-information spreading dynamics, as shown in Figure 3. For the case of \( t_b = 40 \) (see Figures 3(a)–3(d)), i.e., when the quarantine period was relatively small, a similar phenomenon to that shown in Figure 2 was observed. For a large value of \( t_b \), i.e., when \( t_b = 90 \) (as shown in Figures 3(e)–3(h)), a different phenomenon was observed, and the optimal information disappeared. Specifically, \( R_A \) increased monotonically with \( \lambda_A \) or \( \lambda_B \), whereas \( R_B \) decreases monotonically. When \( t_b \) was extremely large, the quarantined nodes did not participate in the virus spreading dynamics when they completed their quarantine period. Hence, we conclude that increasing the information transmission probability can contain virus spreading for a long quarantine period.

Next, we investigate whether an optimal quarantine period can significantly suppress virus spreading. Hence, we
systematically investigated the spread of virus information for different values of $\lambda_A$ and $\lambda_B$, as shown in Figure 4. We discovered that $R_A$ decreased with $t_b$, regardless of the values of $\lambda_A$ and $\lambda_B$. In terms of virus spreading, $R_B$ decreased monotonically with $t_b$ because the larger value of $t_b$ resulted in fewer nodes participating in virus spreading when they complete their quarantine period.

Finally, we investigated the effects of the average degree of the multiplex networks on virus-information spreading, as shown in Figure 5). It is noteworthy that varying the values of $\langle k_A \rangle$ and $\langle k_B \rangle$ do not qualitatively affect the phenomena presented in Figure 2. In other words, an optimal information transmission probability exists at which the spread of virus will be significantly suppressed.

Figure 2: Virus-information spreading dynamics on SFCSF networks. (a) Final information outbreak size $R_A$ and (b) epidemic outbreak size $R_B$ vs. information transmission probability $\lambda_B$ under different epidemic transmission probabilities $\lambda_B = 0.3, 0.5, 0.8$. (c) $R_A$ and (d) $R_B$ vs. $\lambda_B$ under $\lambda_A = 0.3, 0.5, 0.8$. We set the intermittent breaking period as $t_b = 60$, and average degree of two subnetworks are $\langle k_A \rangle = \langle k_B \rangle = 8$. 

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Figure 3: Virus-information spreading dynamics on SFCSF networks. (a) Final information outbreak size $R_A$ and (b) epidemic outbreak size $R_B$ vs. information transmission probability $\lambda_A$ under different epidemic transmission probabilities $\lambda_B = 0.3, 0.5, \text{ and } 0.8$ when $t_b = 40$. (c) $R_A$ and (d) $R_B$ vs. $\lambda_B$ under $\lambda_A = 0.3, 0.5, \text{ and } 0.8$ when $t_b = 40$. (e) $R_A$ and (f) $R_B$ as a function of $\lambda_A$ under $\lambda_B = 0.3, 0.5, \text{ and } 0.8$ when $t_b = 90$. (g) $R_A$ and (h) $R_B$ vs. $\lambda_B$ under $\lambda_A = 0.3, 0.5, \text{ and } 0.8$ when $t_b = 90$. Average degree of two subnetworks are $\langle k_A \rangle = \langle k_B \rangle = 8$.

Figure 4: Continued.
Figure 4: Virus-information spreading dynamics on SFCSF networks. Average degrees are $\langle k_A \rangle = 8$ and $\langle k_B \rangle = 8$. We set the intermittent breaking period as $t_b = 40$.

Figure 5: Continued.
4. Conclusions

Controlling the spread of virus on the Internet is vital to cyberspace security. The spread of virus on the Internet triggers information spreading on social networks. Hence, we proposed an interacting virus-information spreading dynamics model for multiplex networks, in which a node receiving information is intermittently quarantined for a specified period. The spreading dynamics were described using a time-delay heterogeneous mean-field approach. By performing Monte Carlo simulations, we investigated the spreading dynamics of SFCSF networks and identified two situations. For relatively small intermittent periods, virus spreading suppressed significantly at the optimal information transmission probability. For large intermittent periods, i.e., the intermittent period was larger than the virus spreading time. Therefore, increasing the information diffusion probability will likely facilitate the control of viruses. Furthermore, we discovered that increasing the intermittent period suppressed virus spreading. Finally, we changed the average degrees of the two subnetworks and discovered that the above mentioned phenomena were not qualitatively affected.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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