Virus Propagation and Patch Distribution in Multiplex Networks: Modeling, Analysis, and Optimal Allocation

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***Abstract*— Efficient security patch distribution is of essential importance for updating anti-virus software to ensure effective and timely virus detection and cleanup. In this paper, we propose a mixed strategy of patch distribution to combine the advantages of the traditional centralized patch distribution strategy and decentralized patch distribution strategy. A novel network model that contains a central node and a multiplex network composed of patch dissemination network layer and virus propagation network layer is presented, and a competing spreading dynamical process on top of the network model that simulates the interplay between virus propagation and patch dissemination is developed. Such a new framework helps in effectively analyzing the impacts of patches distribution on virus propagation, and developing more realizable schemes for restraining virus propagation. Furthermore, considering the constraints of the capacity of the central node and the bandwidth of network links, an optimal allocation approach of patches is proposed, which could simultaneously optimize multiple dynamical parameters to effectively restrain the virus propagation with a given budget.**

***Index Terms*— Virus propagation, patch distribution, mixed strategy, multiplex network, optimization.**

I. Introduction

COMPUTER virus is a type of malicious software program (malware) that embeds itself in some other computer programs to attack the target system without the user's consent. Once activated, it could quickly diffuse into other computers [1]–[5]. The term virus is also commonly and loosely used to refer to other forms of malware, such as computer worms, Trojan horses, ransomware, spyware, adware, scareware, and so on [6], [7]. In recent years, the rapid developments of internet and the growing popularization of computing devices make it easier for viruses to produce a large-scale propagation and cause tremendous damages to human society.

A variety of countermeasures have been developed to restrain the outbreak of viruses [8]–[23]. The standard approach is to timely update the virus database of anti-virus software installed on the computers or smart phones via patch distribution executed by service providers or security companies. The conventional centralized patch distribution approach [16]–[22], including both the server/push mechanism in which servers assign patches to clients, and the client/pull mechanism in which clients download the patches from a server, has effectively prevented the fast diffusion of viruses. However, such an approach has also shown some unavoidable deficiency [16], [18], [20], [22], [24]–[31]: (1) myriad clients accessing the server and downloading patches within a short time period is similar to launching a DDoS attack. The direct result is that the remaining users can't get the normal service. The scheduled distribution of patches, though helpful, is not timely enough; (2) the network bandwidth is still limited particularly for mobile networks and wireless networks. The gusty transmission of mass data would lead to the network congestion and failures; (3) the service coverage of the server is not guaranteed in certain areas (e.g., rural or underground metro systems); (4) the virus writers could launch direct attacks on the server, thus delaying or preventing patch distribution.

As an alternative, decentralized patch distribution scheme was introduced [16], [24], which was empirically found to be effective, without suffering from bottlenecks or causing network congestion through ingenious design. A lot of decentralized patch distribution schemes have been developed, most of which are executed similar to that in epidemiology, where patch dissemination across networks is analogous to disease spreading in population networks and virus propagation in computer networks [19], [21], [22], [28], [32]–[35]. However, in these decentralized schemes, the patches cannot be disseminated to specified nodes at particular time, while if such can be done, the virus propagation could be restrained more effectively.

Intuitively, a combination of centralized approach and decentralized approach may work better. An example case

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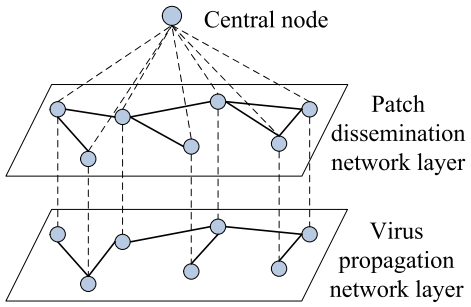

Fig. 1. The network model supporting the mixed patch distribution strategy, which contains a central node and a multiplex network formed by patch dissemination network layer and virus propagation network layer. The central node distributes patches to network nodes directly. The patch layer and virus layer share same nodes yet different topologies, supporting the dissemination of patches and the propagation of virus respectively.

could be that the central server distributes patches to specified nodes at particular time within its capacity limitation, and the decentralized part performs large-scale patch dissemination based on epidemic dynamics. Motivated by this, in this paper a mixed strategy is proposed to combine the advantages of centralized patch distribution strategy and decentralized patch distribution strategy.

Recently, lots of studies have been carried out to investigate the patch dissemination on networks, which contains the same set of network nodes yet different topology from that of the virus propagation network [20], [22], [25], [31], [36]. In details, the channels of patch dissemination are different from those of the virus propagation. For example, Gao and Liu [20] investigated disseminating the patch through MMS-based network. Sobhani and Keshavarz-Haddad [31] studied distributing the patch via peer-to-peer communication network. In this sense, the framework related to the competing spreading of viruses and patches on a two-layer network coupled by virus propagation layer and patch dissemination layer naturally arises, which makes it easier to analyze the influence of patch distribution on virus propagation and identify the most appropriate patch dissemination network to better restrain virus propagation.

Actually, the competing spreading in multiplex networks has recently attracted great attentions in the field of statistical physics [37]–[44]. The multiplex network is an extension of traditional single network and regarded as a major milestone of network science [45]–[60]. Based on this, we present a novel network model that contains a central node and a multiplex network formed by two layers: patch dissemination network layer and virus propagation network layer (see Fig. 1). Our mixed patch distribution strategy runs on the network model in which the central node distributes patches to network nodes directly and the patch layer and virus layer support the dissemination of patches and the propagation of virus respectively. A real world example is that when a virus propagates through Bluetooth network (formed by the geographic location of mobile users), the security service providers could distribute the patch to some important smart phones directly and the patch could also spread via the MMS network (constructed from the address books of mobile users) to realize large scale dissemination. In addition, a spreading dynamical model, termed as virus-patch model, is presented to simulate the competing spreading processes of the virus and patch on multiplex networks. Besides, microscopic Markov chain approach (MMCA) equations are proposed to calculate the probability of network node being in any state at any time, which can help us make quantitative theoretical analysis.

Furthermore, considering the restrictions of capacity of central node and the network bandwidth, the number of patches distributed by centralized approach and disseminated through decentralized approach should be limited in the mixed strategy. In this sense, an interesting question naturally poses itself. Namely, how to optimally allocate patches to most effectively hinder the virus propagation under the constraint of restricted resources. Though studies on epidemic dynamics have spanned a long period of time [61]–[63], it is only recently that control engineers start to focus on such a topic [64]. In this paper, we show how to use the forward-backward propagation algorithm to control the MMCA equations, which can produce the optimal patch allocation solutions to virus prevention [65].

The contributions of the present work can be summarized as follows:

- We propose a mixed patch distribution strategy which combines the advantages of centralized patch distribution scheme and decentralized patch distribution scheme. To our knowledge, this is the first study on patch distribution by a mixed strategy.
- We present a novel network model that supports the running of our mixed patch distribution strategy. A virus-patch model is given to simulate the competing spreading of virus and patches on the proposed network model. The MMCA equations are also proposed to calculate the probability of network node being in any state at any time. Both of them make it easier for us to find more appropriate parameters of network topologies and spreading dynamics to better restrain the viruses.
- Subject to the limitations of capacity of central node and bandwidth of network links, an optimal patch allocation scheme is given which could optimize the patches distribution performed by central node and patches dissemination in network simultaneously. The effectiveness of the optimization scheme is validated by comparative analysis.

The remainder of this paper is organized as follows: Section II surveys existing works on patch distribution and optimal allocation of resources. Section III introduces the system model. Section IV performs model analysis. Section V presents an optimal allocation scheme of patches for best restraining the virus propagation under given budgets. Section VI verifies the efficiency of the optimization algorithm. Conclusion is shown in Section VII.

II. Related Work

To overcome the disadvantages of centralized patch distribution strategy, Toyoizumi and Kara [16] and Kara [24] first proposed the idea of decentralized patch distribution strategy, in which a predator is introduced. Such an antivirus program could be replicated and disseminated from one computer to

another across the network, similar to the propagation of a virus. Gupta and Duvarney [18] empirically found that, with a proper design, the predator could effectively restrain the virus propagation without suffering from bottlenecks or causing network congestion. Tamimi and Khan [66] presented the predator and virus interaction model to analyze the fight of predator against virus in the presence of system patching. After that, an extensive modeling study of decentralized patch distribution strategies was carried out. Eshghi *et al.* [28] devised optimal patching policies that attain the minimum aggregated cost due to the spread of malware and the surcharge of patching. Zhu *et al.* [67] presented a decentralized patch distribution scheme based on epidemiology, where an epidemic dynamical model, SIPS model, was presented to simulate the evolution of viruses and patches. However, this SIPS model is compartmental, which cannot accommodate the complete information of network structures, thus the assessment of the effectiveness of patch distribution is not accurate. As an improvement, Yang *et al.* [22] proposed a node-level SIPS model, which takes into full account the influence of patch distribution. In addition, they assumed that the patch forwarding network is different from the virus spreading network, which is of practical importance to study the combined impacts of these two different networks on the virus propagation.

The framework of competing spreading on multiplex networks has attracted much attention recently. Granell *et al.* [37] first presented the analysis of interrelation between two processes, accounting for the spreading of an epidemic and the information awareness to prevent its infection on top of multiplex networks. Wang *et al.* [68] investigated the co-evolution mechanisms and dynamics between information and disease spreading by utilizing real data and found there is an optimal information transmission rate enabling to evidently suppresses disease spreading. Wei *et al.* [41] studied various mutual influences of two spreading processes on two-layer networks. The epidemic thresholds of interacting networks were contrasted and proven by using the corresponding isolated networks for three scenarios, including competing spreading process, cooperative spreading process and the combination of the two processes. Yang *et al.* [69] considered the bi-virus competing spreading on top of bilayer-network with generic infection rates which takes the first step toward enhancing the accuracy of multi-virus competing spreading models. Moreover, Granell *et al.* [37] proposed a more general scenario of competing spreading on multiplex networks, where a mass media is introduced. Then, they analyzed the influence of mass media on the final outcome of the epidemic incidence. This framework is similar to ours, however, its competing spreading model cannot capture the interplay dynamics of virus and patches appropriately.

The ultimate goal of the optimal allocation of patches is controlling the epidemic dynamics and stopping the virus propagation as quickly as possible [64]. If resources are not an issue, an intuitive solution to quickly restrain viruses is patching all network nodes, which, however, may be undesirable since the resources are often limited and patching every node typically incurs huge cost. Consequently, how to optimally allocate resources selecting the parameters of spreading model with fixed budget becomes a core issue. Many works have paid great attention to minimizing the threshold value of spreading model under various constraints. In [70] and [71], the problem of minimizing threshold was cast into a semidefinite program framework for undirected networks. In [70] and [72], this problem was solved for directed networks using geometric programming, where the solution can be obtained using standard off-the-shelf convex optimization software. Shakeri *et al.* [73] identified the optimal dynamical parameters on multilayer networks in order to achieve a dying-out epidemic. They coupled nonlinear Perron-Frobenius problem (NPF) with convex optimization problem, creating a general method that can be applied to solve a variety of optimization problems combined with NPF problems in various disciplines. Recently, Lokhov and Saad [65] proposed a general optimization framework based on scalable dynamic message-passing approach, which is principled, probabilistic, computationally efficient and incorporates the topological properties of the specific network. In this paper, this framework is extended to control the MMCA equations to derive the optimal solutions of multiple parameters of the virus-patch model.

III. System Model

In this section, we first introduce the network model and the mixed patch distribution strategy and the related virus-patch model. Then, a Markov chain is given to represent the evolution process of virus-patch model on the network model.

A. Network Model

Figure 1 shows the network model which contains a central node and a two-layers network. The upper layer represents the patch dissemination network layer (PL). The lower layer corresponds to the virus propagation network layer (VL). Nodes placed at each end of an inter-layer link actually represent the same nodes. That is, PL and VL share same set of network nodes yet different intra-layer connections. There are paths from the central node to network nodes since the network node could download the patch from the central node or the central node could send the patch to the network node. We use V to denote the set of nodes of the two-layers network, and $|V| = N$ is the number of network nodes. $A^p = \{a_{ij}^p\}_{N\times N}$ refers to the adjacency matrix of PL where $a_{ij}^p = 1$ indicates that there is a link between node i and node j in PL; otherwise $a_{ij}^p = 0$. Similar definition also applies to $A^v = \{a_{ij}^v\}_{N\times N}$ for VL. Besides, $\langle k^p\rangle$ and $\langle k^v\rangle$ are defined as the average degrees of PL and VL respectively.

B. Mixed Patch Distribution Strategy

The mixed patch distribution strategy runs on the network model. It distributes patches to network nodes directly via the central node and the patches can further disseminate across the patch layer. In this way, we can restrain the virus propagation on virus layer by taking advantages of the traditional centralized patch distribution strategy and decentralized patch distribution strategy at the same time. We now propose the

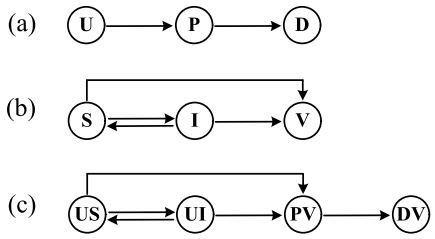

Fig. 2. State transition of nodes in (a) UPD model, (b) SISV model and (c) virus-patch model.

following models to simulate the ways of working of the mixed strategy and its evolution.

Based on the decentralized patch distribution strategy and the epidemiology [74], we propose an Unpatched-Patched-Disabled (UPD) model to define the patch dissemination on PL. The Unpatched nodes have no patches and could become patched after installing patches acquired from the central node or patched neighbors. For patched nodes, because they have been installed with patches, they are immunized against the viruses. They could disseminate patches to their unpatched neighbors. In particular, disabled nodes still have patches and are immune to the virus, but they no longer disseminate patches due to external control or loss of motivation, by which the network congestion during the patch dissemination can be avoided. The UPD model uses discrete time step for its evolution. At time step t, the unpatched node becomes patched with probability m if receiving patches from the central node, or β^p if receiving patches from a patched neighbor; while the patched node could become disabled with probability δ^p.

Furthermore, we utilize Susceptible-Infected-Susceptible-Vaccinated (SISV) model to characterize the virus propagation on VL. Susceptible nodes are free of viruses and could become infected after incurring viruses from infected neighbors. Infected nodes are assumed to carry the viruses and pass it towards susceptible neighbors. They could become susceptible again via system reinstallation. The vaccinated nodes are patched and immune to the viruses so that these nodes neither diffuse the virus nor get infected again. Both susceptible nodes and infected neighbors could be vaccinated directly if patches are installed on them. In particular, it is noted that the transition from susceptible or infected to vaccinated status is the same transition from unpatched to patched status in UPD. Therefore, we assume that at time step t of SISV, a susceptible node is infected by one of its infected neighbors with probability β^v; infected node becomes susceptible via system reinstallation with probability δ^v; a node in susceptible state or infected state becomes vaccinated with probability m if receiving patch from the central node, or β^p if receiving patch from patched neighbor.

In Figures 2 (a) and (b), we show the state transition of nodes in UPD and SISV. If we incorporate UPD and SISV together, the unpatched node in UPD could also be susceptible or infected state in SISV at the same time since it has not been patched. In addition, the patched node in UPD means that it is vaccinated in SISV. Although the disabled node no longer disseminates patches, it is also immune to viruses and is thus vaccinated in SISV. Totally, the nodes in the incorporated virus-patch model could be in one of following combination states: US (unpatched and susceptible), UI (unpatched and infected), PV (patched and vaccinated) and DV (disabled and vaccinated). The US node could become UI if it is infected by the virus, or PV if it has the patch. The UI node could become US state via system reinstallation, or PV state by installing patch. Finally, the PV node could become DV due to external control or loss of motivation. The state transition of nodes in virus-patch model is shown in Fig. 2(c).

In order to quantify the evolution of virus-patch model, we need to compute the marginal probability of each node i in the four states at time t, which are denoted as $p_i^{US}(t)$, $p_i^{UI}(t)$, $p_i^{PV}(t)$ and $p_i^{DV}(t)$ respectively. We propose the MMCA equations for the evolution of virus-patch model which read as

$$\begin{aligned} p_i^{US}(t+1) = {} & p_i^{US}(t)(1-m)r_i(t)q_i(t) \\ & + p_i^{UI}(t)(1-m)r_i(t)\delta^v, \end{aligned} \tag{1}$$

$$\begin{aligned} p_i^{UI}(t+1) = {} & p_i^{US}(t)(1-m)r_i(t)\big(1-q_i(t)\big) \\ & + p_i^{UI}(t)(1-m)r_i(t)(1-\delta^v), \end{aligned} \tag{2}$$

$$\begin{aligned} p_i^{PV}(t+1) = {} & p_i^{US}(t)\big(1-(1-m)r_i(t)\big) \\ & + p_i^{UI}(t)\big(1-(1-m)r_i(t)\big) \\ & + p_i^{PV}(t)(1-\delta^p), \end{aligned} \tag{3}$$

$$p_i^{DV}(t+1) = p_i^{DV}(t) + p_i^{PV}(t)\delta^p, \tag{4}$$

where $q_i(t)$ and $r_i(t)$ indicate the probabilities that node is not infected by any infected neighbors and not patched by any patched neighbors respectively. They are given as

$$q_i(t) = \prod_j \left(1 - a_{ij}^v p_j^{UI}(t)\beta^v\right), \tag{5}$$

$$r_i(t) = \prod_j \left(1 - a_{ij}^p p_j^{PV}(t)\beta^p\right). \tag{6}$$

Solving iteratively the system of Eqs. (1)-(4), together with Eqs. (5) and (6), we can track the time evolution of viruses and patches for any initial condition. Although we use discrete-time model for the evolution of the virus-patch in present work, the continuous-time model is also suitable, which can be derived easily. The use of the discrete-time model here mainly due to the requirement for solving the optimal patches allocation problem discussed in section V. The MMCA equations derived under the assumption of discrete-time model is more easier controlled and handled by the forward-backward propagation algorithm to get the optimal patches allocation solutions.

IV. Analysis

In this section, we first analyze the evolution of virus-patch model based on Eqs. (1)-(6), then calculate the critical threshold of virus outbreak, finally analyze the impact of network topologies (intra-layer connections and inter-layer connections) and dynamic parameters of virus-patch model on the virus propagation. In this way, we can figure out how to better restrain virus propagation. Note that, in general we

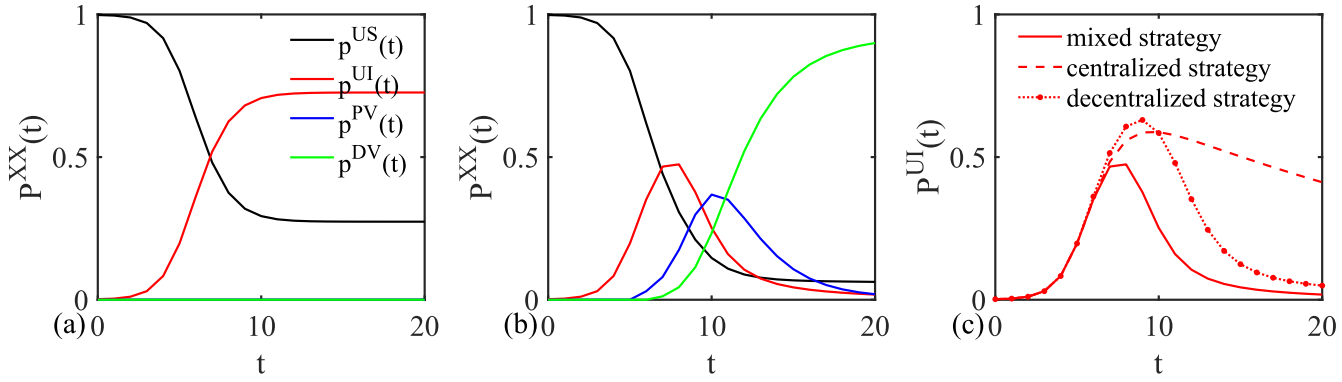

Fig. 3. The evolution of virus-patch model. $\beta^v = 0.3, \delta^v = 0.2$ for all panels (a), (b) and (c), $\beta^p = 0.3, \delta^p = 0.4, m = 0.03, t_c = 5$ for panel (b). In panel (c), for mixed strategy, we set $\beta^p = 0.3, \delta^p = 0.4, m = 0.03$ and $t_c = 5$; for centralized strategy, $\beta^p = 0.0, \delta^p = 0.0, m = 0.03$ and $t_c = 5$; for decentralized strategy, $\beta^p = 0.3, \delta^p = 0.4, m = 0$, and at $t_c = 5$ a random node is chosen to be patched. Both VL and PL are SF network with average degree $\langle k^p \rangle = \langle k^v \rangle = 4$.

detect certain viruses and then distribute patches into networks only after these viruses have spread out. Therefore, we define $t_c > 0$ (0 is the initial time of virus propagation) as the launch time when patches start to be distributed into the network.

Figures 3 (a) and (b) show the evolution of virus-patch model without and with the patch distribution respectively. Here, $p^{XX}(t)$ denotes the fraction of XX state nodes in the system at time t, which is given by $p^{XX}(t) = \frac{1}{N}\sum_i p_i^{XX}(t)$. When no patches are distributed into the PL, the virus propagation on VL follows the SISV model without any influence of patch. However, after inserting patches into the network ($t_c = 5$ in panel (b)), the increase in the number of infected nodes slows down and eventually disappears, which illustrates that patch distribution can effectively restrain the virus propagation. In Fig. 3 (c), we compare the efficiency of the mixed strategy with the traditional centralized patch distribution strategy and decentralized patch distribution strategy. For centralized patch strategy, only the central node distributes patches into the network, the patched nodes cannot disseminate patches to their unpatched neighbors. For decentralized strategy, one randomly chosen node is patched at time t_c, then the patches disseminate across the network following the UPD model while the central node cannot distribute any patch into the network at any time. It is clear that, compared with above two cases, the mixed strategy performs better. It could quickly and sharply restrain the virus propagation. In addition, in Section V of this paper, we will present an optimal allocation scheme of patches, which can further improve the mixed strategy.

Moreover, interestingly, since $t_c > 0$, we can solve analytically the stationary state of the virus-patch model, and determine the critical threshold β_c^v of the virus. Below the threshold, $\beta^v < \beta_c^v$, the network is virus free in the steady state, while for $\beta^v > \beta_c^v$, the network reaches an endemic state with a finite stationary density p^{UI}. The knowledge of the threshold β_c^v is an important practical tool to predict and control virus spreading in networks which also decides whether we should distribute the patch.

Theorem 1: When $t_c > 0$, the critical threshold of virus outbreak in virus-patch model is $\beta_c^v = \delta^v / \Lambda_{max}(A^v)$.

Proof: When $t_c > 0$, no patches are allocated into the networks at the time $t < t_c$, therefore we have $m = \beta^p = \delta^p = 0$ for $t < t_c$. In this sense, the Markov chains of virus-patch model can be simplified as

$$p_i^{US}(t+1) = p_i^{US}(t)q_i(t) + p_i^{UI}(t)\delta^v, \tag{7}$$

$$p_i^{UI}(t+1) = p_i^{US}(t)\big(1 - q_i(t)\big) + p_i^{UI}(t)(1-\delta^v), \tag{8}$$

where

$$q_i(t) = \prod_j [1 - a_{ij}^v p_j^{UI}(t)\beta^v]. \tag{9}$$

At the early stage of the model, we can assume that the infected nodes account for only a very small fraction of the entire network, i.e., $p_i^{UI} = \sigma_i \ll 1$. In such a regime, items like $\prod_{k=1}^{m} \sigma_{j_k}$ $(m \geq 2)$ shall be approximated as being equal to 0. Therefore, the Eq. (9) could be approximated as

$$q_i = \prod_j [1 - a_{ij}^v \sigma_j \beta^v] \approx 1 - \beta^v \sum_j a_{ij}^v \sigma_j. \tag{10}$$

Eq. (8) then becomes

$$\sigma_i = (1-\sigma_i)\beta^v \sum_j a_{ij}^v \sigma_j + \sigma_i(1-\delta^v). \tag{11}$$

By neglecting all $\sigma_i\sigma_j$ terms in Eq. (11), we obtain

$$\sum_j [\beta^v a_{ij}^v - \delta^v e_{ij}]\sigma_j = 0, \tag{12}$$

where e_{ij} is the element of the identity matrix E. Together with all $i \in V$, we have

$$(A^v - \frac{\delta^v}{\beta^v}E)\sigma = 0, \tag{13}$$

where $\sigma^T = (\sigma_1, \sigma_2, \ldots, \sigma_N)$ is the transpose of σ. The non-trivial solution of Eq. (13) is eigenvectors of A^v, whose largest real eigenvalues are equal to δ^v/β^v. Therefore, the onset of the virus is given by the largest real eigenvalue of A^v. That is

$$\beta_c^v = \frac{\delta^v}{\Lambda_{max}(A^v)}. \tag{14}$$

□

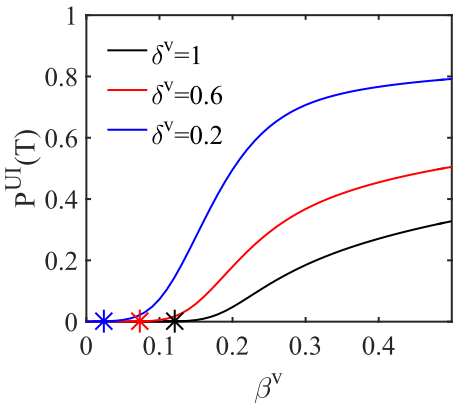

Fig. 4. The positions of asterisk on X-axis represent the corresponding values of β_c^v. $\beta^p = 0.3, \delta^p = 0.4, m = 0.03, t_c = 10$ and $T = 10$ for the virus-patch model. Both VL and PL are SF network with average degree $\langle k^p \rangle = \langle k^v \rangle = 4$.

We verify the accuracy of Eq. (14) in Fig. 4, where the positions of asterisk on X-axis represent the corresponding values of β_c^v. It is clear that the theoretical thresholds exactly confirm the existence of virus regime. When $\beta^v < \beta_c^v$, one can see that $p^{UI} \approx 0$. For $\beta^v > \beta_c^v$, we have $p^{UI} > 0$.

Figure 5 shows the influences of t_c and dynamical parameters of virus-patch model on virus propagation. From panel (a), one sees that the smaller t_c is, the fewer nodes will be infected. This reminds us that we should detect the virus and distribute patches as early as possible. In panel (b), it can be found that m has a significant inverse relationship with the number of infected nodes. The more patches the central node distributes, the fewer nodes are infected by the virus. Therefore, the central node should distribute as many patches as possible within the capacity limitation. Furthermore, we consider the impact of effective spreading rate which is defined as the ratio of spreading rate to recovery rate. Specifically, two effective spreading rates exist in virus-patch mode, which are given by $\eta^p = \beta^p/\delta^p$ and $\eta^v = \beta^v/\delta^v$ respectively. The results shown in panel (c) are in line with our general cognition. For a given η^v, larger η^p could restrain the virus propagation more efficiently. If β^p is assumed to be unchanged, we should decrease δ^p as much as possible to increase the value of η^p, thus to restrain the virus. That is, we should control the transition of nodes from Patched state to Disabled state in virus-patch model.

In Fig. 6, we investigate the influence of topologies of multiplex network, including intra-layer topology and inter-layer topology, on virus propagation. From panel (a), we find the average degree of PL has dramatic impact on the outbreak scale of viruses. The larger $\langle k^p \rangle$ is, the fewer nodes will be infected. This illustrates we should find the networks with large average degree as the PL. The inter-layer correlation between nodes in different layers is another major topology property of multiplex networks. Here, we consider two well known types of correlations, degree-degree correlations (DDC) and average similarity of neighbors (ASN) [52]. The DDC of our proposed multiplex networks can be defined as

$$r = \frac{\langle k_i^p k_i^v \rangle - \langle k_i^p \rangle \langle k_i^v \rangle}{\tau^p \tau^v}, \tag{15}$$

where $\tau^p = \sqrt{\langle (k_i^p)^2 \rangle - \langle k_i^p \rangle^2}$, and k_i^p is the degree of node i in PL. The range of values for r is -1 to 1. $r > 0$ is called positive correlation, i.e. large (small) degree nodes in one layer often couple with large (small) degree nodes in the other layer. While $r < 0$ indicates negative correlation, i.e. large (small) degree nodes in one layer often connect with small (large) degree nodes in the other layer. The ASN is given by

$$\alpha = \frac{\sum_i |\Gamma_i^p \cap \Gamma_i^v|}{\sum_i |\Gamma_i^p \cup \Gamma_i^v|}, \tag{16}$$

where Γ_i^p refers to the set of neighbors of node i in PL. For an increasing value of α, more neighbors of a node in one layer are also its neighbors in another layer, i.e. two layers become more similar. For $\alpha = 1$, these two layers will be identical.

From Fig. 6 (b), $p^{UI}(T)$ decreases with the increase of r, which illustrates that positive correlation between PL and VL could restrain virus propagation more effectively than negative correlation. Since large degree nodes are more likely to be patched when patches are disseminated in PL. The positive correlation between PL and VL makes larger degree nodes of VL to be patched more quickly and easily, which is thus more favorable to restrict virus propagation. In addition, from Fig. 6 (c), one sees that ASN has no obvious impact on the virus propagation. Therefore, when finding or constructing the PL, we do not need to consider ASN between PL and VL.

All above analyses tell us that virus-patch model with small t_c and large m and η_p, and PL with large $\langle k^p \rangle$ and positive correlated with VL, are more favorable to restrain the virus propagation.

V. Optimal Patch Allocation

From above section, we got the knowledge: what dynamical parameters of virus-patch model and topology structures of network model can better restrain the virus propagation. However, for a given network model, the capacity of the central node and the bandwidth of network links are limited, which make the dynamical parameters related with patch distribution m and η^p ($\eta^p = \beta^p/\delta^p$) not be infinitely large. In addition, the setup of the same values of m, β^p and δ^p for each network nodes at any time is apparently not the optimum configuration, since if nodes with highest risk of infection or with highest probability to infect others could have highest priority for patching, the virus may be restrained more effectively. In this sense, an interesting question naturally poses itself, which we aim to address in this section. Namely, how to optimally invest patches to best hinder the virus propagation under fixed budget, which can be primarily described by the following problem definition.

Problem Definition: Given fixed budgets of capacity of central node and network bandwidth, find the optimal probability that unpatched node i becomes patched state when receiving patches from the central node at time t, and the optimal probability that patched node j becomes disabled state at time t, to best restrain the virus.

Noted that the effective spreading rate of patches on PL is determined by its real spreading rate and recovery rate, changing one of them is equivalent to changing the effective

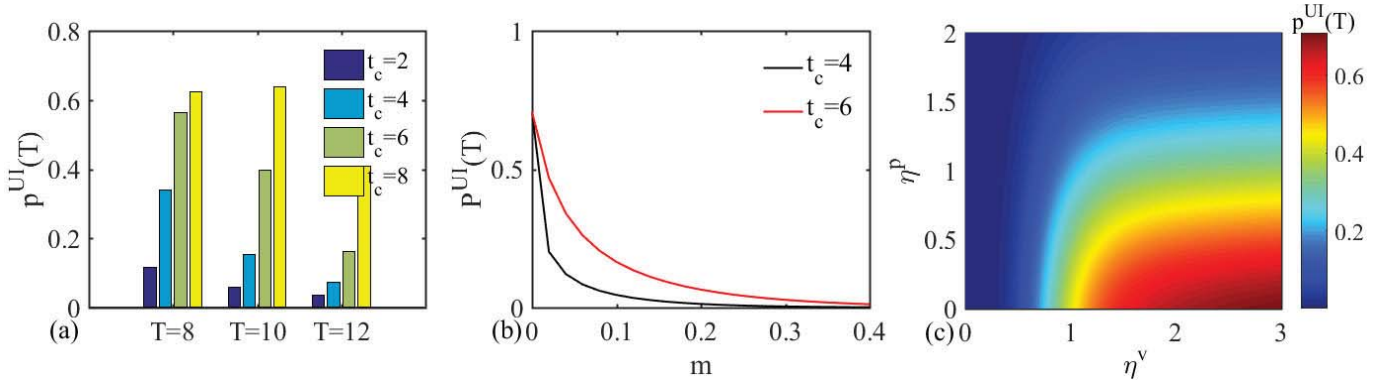

Fig. 5. (a) Influence of t_c, $\beta^v = 0.3, \beta^p = 0.3, \delta^v = 0.2, \delta^p = 0.4, m = 0.03$. (b) Influence of m, $\beta^v = 0.3, \beta^p = 0.3, \delta^v = 0.2, \delta^p = 0.4, T = 10$. (c) Influence of effective spreading rates, $t_c = 6, T = 10$. For (a-c), both PL and VL are SF network with average degree $\langle k^p \rangle = \langle k^v \rangle = 4$.

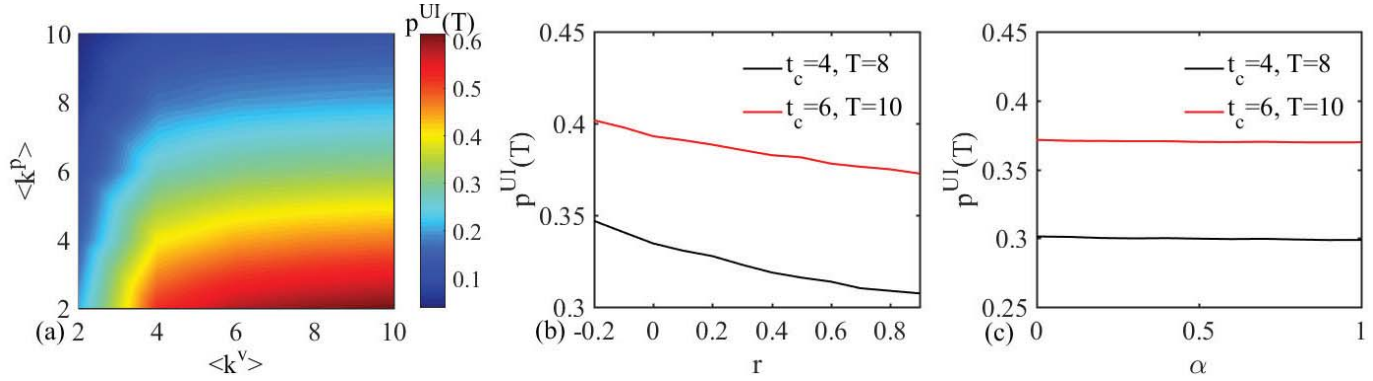

Fig. 6. (a) Influence of average degree, $t_c = 6, T = 10$. (b) Influence of DDC. (c) Influence of ANS. For (a-c), we set $\beta^v = 0.3, \beta^p = 0.3, \delta^v = 0.2, \delta^p = 0.4, m = 0.03$. For (b) and (c), both PL and VL are SF network with average degree $\langle k^p \rangle = \langle k^v \rangle = 4$.

spreading rate. Based on real scenario of control, manipulating the recovery rate is more reasonable and practical than manipulating the spreading rate. Therefore, in *Problem definition* and in what follows, we optimize the effective spreading rate via optimizing the recovery rate of patched nodes to disabled state.

By simultaneously optimizing these two type of dynamical parameters, an optimal mixed strategy of patch distribution is obtained. The basic idea of optimal allocation of patches is first to replace m and δ^p with $m_i(t)$ and $\delta_i^p(t)$ in the dynamical equations of virus-patch model, which are given by

$$\begin{aligned} p_i^{US}(t+1) &= p_i^{US}(t)(1-m_i(t))r_i(t)q_i(t) \\ &\quad + p_i^{UI}(t)(1-m_i(t))r_i(t)\delta^v, \end{aligned} \tag{17}$$

$$\begin{aligned} p_i^{UI}(t+1) &= p_i^{US}(t)(1-m_i(t))r_i(t)\big(1-q_i(t)\big) \\ &\quad + p_i^{UI}(t)(1-m_i(t))r_i(t)(1-\delta^v), \end{aligned} \tag{18}$$

$$\begin{aligned} p_i^{PV}(t+1) &= p_i^{US}(t)\big(1-(1-m_i(t))r_i(t)\big) \\ &\quad + p_i^{UI}(t)\big(1-(1-m_i(t))r_i(t)\big) \\ &\quad + p_i^{PV}(t)(1-\delta_i^p(t)), \end{aligned} \tag{19}$$

$$p_i^{DV}(t+1) = p_i^{DV}(t) + p_i^{PV}(t)\delta_i^p(t), \tag{20}$$

where

$$q_i(t) = \prod_j \Big(1 - a_{ij}^v p_j^{UI}(t)\beta^v\Big), \tag{21}$$

$$r_i(t) = \prod_j \Big(1 - a_{ij}^p p_j^{PV}(t)\beta^p\Big). \tag{22}$$

That is, the probability that an unpatched node goes into the patched state when receiving patches from the central node, and the probability that one patched node becomes disabled, could be different for different nodes and vary with time. $m_i(t)$ and $\delta_i^p(t)$ are the parameters that need to be optimized under given fixed budgets.

We then quantify the optimal objective via function O and the fixed budgets via available resources B.

O- The ultimate goal of restraining virus is to minimize the outbreak size of infection. Furthermore, minimizing the outbreak size of infection at particular time i.e. minimizing $\sum_{i\in V} p_i^{UI}(T)$, may be more valuable in practical applications. And minimizing $\sum_{i\in V} p_i^{UI}(T)$ is equivalent to maximizing $\sum_{i\in V}(1-p_i^{UI}(T))$. Therefore, we define the objective function of optimal allocation of patches as

$$O = \sum_{i\in V} \big(1 - p_i^{UI}(T)\big). \tag{23}$$

B- The constraint is of two types, the capacity of central node and the bandwidth of network, which correspond to the dynamical parameters $m_i(t)$ and $\delta_i^p(t)$ respectively. In addition, the constraints may vary with time. Therefore, the constraints are presented by

$$\sum_{i\in V} m_i(t) = B_m(t), \tag{24}$$

$$\begin{aligned}\mathcal{L} = &\sum_{i\in V}\left(1-p_i^{UI}(T)\right)+\sum_{t=0}^{T-1}\lambda_m(t)\Big(\sum_{i\in V}m_i(t)-B_m(t)\Big)+\sum_{t=0}^{T-1}\lambda_{\delta^p}(t)\Big(\sum_{i\in V}\delta_i^p(t)-B_{\delta^p}(t)\Big)\\
&+\varepsilon\sum_{t=0}^{T-1}\sum_{i\in V}\Big(\log\Big(m_i(t)-\underline{m_i(t)}\Big)+\log\Big(\overline{m_i(t)}-m_i(t)\Big)\Big)+\varepsilon\sum_{t=0}^{T-1}\sum_{i\in V}\Big(\log\Big(\delta_i^p(t)-\underline{\delta_i^p(t)}\Big)+\log\Big(\overline{\delta_i^p(t)}-\delta_i^p(t)\Big)\Big)\\
&+\sum_{t=0}^{T-1}\sum_{i\in V}\lambda_i^{US}(t+1)\Big(p_i^{US}(t+1)-p_i^{US}(t)\big(1-m_i(t)\big)r_i(t)q_i(t)-p_i^{UI}(t)\big(1-m_i(t)\big)r_i(t)\delta^v\Big)\\
&+\sum_{t=0}^{T-1}\sum_{i\in V}\lambda_i^{UI}(t+1)\Big(p_i^{UI}(t+1)-p_i^{US}(t)\big(1-m_i(t)\big)r_i(t)\big(1-q_i(t)\big)-p_i^{UI}(t)\big(1-m_i(t)\big)r_i(t)\big(1-\delta^v\big)\Big)\\
&+\sum_{t=0}^{T-1}\sum_{i\in V}\lambda_i^{PV}(t+1)\Big(p_i^{PV}(t+1)-p_i^{US}(t)\Big(1-\big(1-m_i(t)\big)r_i(t)\Big)-p_i^{UI}(t)\Big(1-\big(1-m_i(t)\big)r_i(t)\Big)-p_i^{PV}(t)\big(1-\delta_i^p(t)\big)\Big)\\
&+\sum_{t=0}^{T-1}\sum_{i\in V}\lambda_i^{DV}(t+1)\Big(p_i^{DV}(t+1)-p_i^{DV}(t)-p_i^{PV}(t)\delta_i^p(t)\Big)\\
&+\sum_{t=0}^{T}\sum_{i\in V}\lambda_i^{q}(t)\Big(q_i(t)-\prod_j\Big(1-a_{ij}^v p_j^{UI}(t)\beta^v\Big)\Big)\\
&+\sum_{t=0}^{T}\sum_{i\in V}\lambda_i^{r}(t)\Big(r_i(t)-\prod_j\Big(1-a_{ij}^p p_j^{PV}(t)\beta^p\Big)\Big)\end{aligned}\tag{28}$$

and

$$\sum_{i\in V}\delta_i^p(t)=B_{\delta^p}(t).\tag{25}$$

Moreover, $m_i(t)$ and $\delta_i^p(t)$ may have their own variation ranges that we should not exceed. That is, $m_i(t)$ and $\delta_i^p(t)$ can increase up to a certain maximum or decrease down to a certain minimum, i.e.,

$$\underline{m_i(t)}\le m_i(t)\le\overline{m_i(t)}.\tag{26}$$

and

$$\underline{\delta_i^p(t)}\le\delta_i^p(t)\le\overline{\delta_i^p(t)}.\tag{27}$$

Based on the objective function O and the constraints B, a Lagrangian formulation of constrained optimization problem is employed. As shown in Eq. (28), as shown at the top of this page, $\lambda_m(t)$, $\lambda_{\delta^p}(t)$, $\lambda_i^{US}(t)$, $\lambda_i^{UI}(t)$, $\lambda_i^{PV}(t)$, $\lambda_i^{DV}(t)$, $\lambda_i^q(t)$ and $\lambda_i^r(t)$ are defined as the Lagrange multipliers. The first term of r.h.s. is the objective functions and the second and third terms are the resource constraints. The forth and fifth terms are the barrier functions where ε is a small regularization parameter chosen to minimize the impact on the objective functions in the regime of allowed values of $m_i(t)$ and $\delta_i^p(t)$, away from their borders. And the rest terms are the dynamical model constraints. Setting the derivatives of $\mathcal{L}$ with respect to $p_i^{US}(t)$, $p_i^{UI}(t)$, $p_i^{PV}(t)$, $p_i^{DV}(t)$, $q_i(t)$, $r_i(t)$, $m_i(t)$ and $\delta_i^p(t)$ to zero, we obtain Eqs. (29a)-(29h), as shown at the top of the next page.

Based on the above work, we extend the DMP-based optimization algorithm proposed in [65] to control our MMCA equations and to simultaneously optimize the $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ for best restraining the virus. Here, we call our optimization method the MMCA-based optimization scheme. The MMCA-based optimization is initialized by randomly assigning values to $m_i(t)$ and $\delta_i^p(t)$ for all i and $t\in[0,T-1]$. Then we repeat the following steps for a fixed number of iterations or until convergence:

(1) Starting from the initial values for $p_i^{US}(0)$, $p_i^{UI}(0)$, $p_i^{PV}(0)$ and $p_i^{DV}(0)$, propagate the MMCA Eqs. (17)-(22) forward, up to the horizon T. We obtain the values of $p_i^{US}(t)$, $p_i^{UI}(t)$, $p_i^{PV}(t)$, $p_i^{DV}(t)$, $q_i(t)$ and $r_i(t)$ for all i and $t\in[0,T]$.

(2) Use Eqs. (29a)-(29h) for fixing the boundary values of Lagrange multipliers at time T:

- Eq. (29a) assigns $\lambda_i^{US}(T)=0$;
- Eq. (29d) gives $\lambda_i^{DV}(T)=0$;
- Eq. (29e) gives $\lambda_i^q(T)=0$;
- Eq. (29f) assigns $\lambda_i^r(T)=0$;
- Eq. (29b) assigns $\lambda_i^{UI}(T)=1$ through $\lambda_i^q(T)$;
- Eq. (29c) assigns $\lambda_i^{PV}(T)=0$ through $\lambda_i^r(T)$;
- Eq. (29g) sets $\lambda_m(T-1)$ and $m_i(T-1)$ through $\lambda_i^{US}(T)$, $\lambda_i^{UI}(T)$ and $\lambda_i^{PV}(T)$.
- Eq. (29h) sets $\lambda_{\delta^p}(T-1)$ and $\delta_i^p(T-1)$ through $\lambda_i^{PV}(T)$ and $\lambda_i^{DV}(T)$.

When the control parameter $m_i(t)$ can take all possible values between 0 and 1, means that $\underline{m_i(t)}=0$ and $\overline{m_i(t)}=1$ for all i and $t\in[0,T]$. Together with Eq. (29g), we have

$$m_i(t)=\frac{\lambda_m(t)+x_i(t)-2\varepsilon\pm\sqrt{(\lambda_m(t)+x_i(t))^2+4\varepsilon^2}}{2(\lambda_m(t)+x_i(t))},\tag{30}$$

$$\partial\mathcal{L}/\partial p_i^{US}(t) = \lambda_i^{US}(t) + \Big[-\lambda_i^{US}(t+1)\big(1-m_i(t)\big)r_i(t)q_i(t) - \lambda_i^{UI}(t+1)\big(1-m_i(t)\big)r_i(t)\big(1-q_i(t)\big) - \lambda_i^{PV}(t+1)\Big(1-\big(1-m_i(t)\big)r_i(t)\Big)\Big][t\neq T] = 0 \tag{29a}$$

$$\partial\mathcal{L}/\partial p_i^{UI}(t) = -1[t=T] + \lambda_i^{UI}(t) + \Big[-\lambda_i^{US}(t+1)\big(1-m_i(t)\big)r_i(t)\delta^v - \lambda_i^{UI}(t+1)\big(1-m_i(t)\big)r_i(t)\big(1-\delta^v\big) - \lambda_i^{PV}(t+1)\Big(1-\big(1-m_i(t)\big)r_i(t)\Big)\Big][t\neq T] + \sum_{j\in\Gamma_i^v}\lambda_j^q(t)a_{ji}^v\beta^v\prod_{k\in\Gamma_j^v\backslash i}\Big(1-a_{jk}^v p_k^{UI}(t)\beta^v\Big) = 0 \tag{29b}$$

$$\partial\mathcal{L}/\partial P_i^{PV}(t) = \lambda_i^{PV}(t) + \Big[-\lambda_i^{PV}(t+1)\big(1-\delta_i^p(t)\big) - \lambda_i^{DV}(t+1)\delta_i^p(t)\Big][t\neq T] + \sum_{j\in\Gamma_i^p}\lambda_j^r(t)a_{ji}^p\beta^p\prod_{k\in\Gamma_j^p\backslash i}\Big(1-a_{jk}^p p_k^{PV}(t)\beta^p\Big) = 0 \tag{29c}$$

$$\partial\mathcal{L}/\partial p_i^{DV}(t) = \lambda_i^{DV}(t) - \lambda_i^{DV}(t+1)[t\neq T] = 0 \tag{29d}$$

$$\partial\mathcal{L}/\partial q_i(t) = \Big[-\lambda_i^{US}(t+1)p_i^{US}(t)\big(1-m_i(t)\big)r_i(t) + \lambda_i^{UI}(t+1)p_i^{US}(t)\big(1-m_i(t)\big)r_i(t)\Big][t\neq T] + \lambda_i^q(t) = 0 \tag{29e}$$

$$\partial\mathcal{L}/\partial r_i(t) = \Big[-\lambda_i^{US}(t+1)p_i^{US}(t)\big(1-m_i(t)\big)q_i(t) - \lambda_i^{US}(t+1)p_i^{UI}(t)\big(1-m_i(t)\big)\delta^v - \lambda_i^{UI}(t+1)p_i^{US}(t)\big(1-m_i(t)\big)\big(1-q_i(t)\big) - \lambda_i^{UI}(t+1)p_i^{UI}(t)\big(1-m_i(t)\big)\big(1-\delta^v\big) + \lambda_i^{PV}(t+1)p_i^{US}(t)\big(1-m_i(t)\big) + \lambda_i^{PV}(t+1)p_i^{UI}(t)\big(1-m_i(t)\big)\Big][t\neq T] + \lambda_i^r(t) = 0 \tag{29f}$$

$$\partial\mathcal{L}/\partial m_i(t) = \lambda_m(t) + \frac{\varepsilon}{m_i(t)-\underline{m_i(t)}} - \frac{\varepsilon}{\overline{m_i(t)}-m_i(t)} + \Big[\lambda_i^{US}(t+1)p_i^{US}(t)r_i(t)q_i(t) + \lambda_i^{US}(t+1)p_i^{UI}(t)r_i(t)\delta^v + \lambda_i^{UI}(t+1)p_i^{US}(t)r_i(t)\big(1-q_i(t)\big) + \lambda_i^{UI}(t+1)p_i^{UI}(t)r_i(t)\big(1-\delta^v\big) - \lambda_i^{PV}(t+1)p_i^{US}(t)r_i(t) - \lambda_i^{PV}(t+1)p_i^{UI}(t)r_i(t)\Big][t\neq T] = 0 \tag{29g}$$

$$\partial\mathcal{L}/\partial\delta_i^p(t) = \lambda_{\delta^p}(t) + \frac{\varepsilon}{\delta_i^p(t)-\underline{\delta_i^p(t)}} - \frac{\varepsilon}{\overline{\delta_i^p(t)}-\delta_i^p(t)} + \Big[\lambda_i^{PV}(t+1)p_i^{PV}(t) - \lambda_i^{DV}(t+1)p_i^{PV}(t)\Big][t\neq T] = 0 \tag{29h}$$

where

$$x_i(t) = \lambda_i^{US}(t+1)p_i^{US}(t)r_i(t)q_i(t) + \lambda_i^{US}(t+1)p_i^{UI}(t)r_i(t)\delta^v + \lambda_i^{UI}(t+1)p_i^{US}(t)r_i(t)\big(1-q_i(t)\big) + \lambda_i^{UI}(t+1)p_i^{UI}(t)r_i(t)\big(1-\delta^v\big) - \lambda_i^{PV}(t+1)p_i^{US}(t)r_i(t) - \lambda_i^{PV}(t+1)p_i^{UI}(t)r_i(t).$$

Since ε is a very small positive number, $m_i(t)$ has positive sign in front of the square root being always in the range of (0 1), that is, $0 < m_i(t) < 1$. Together with the constraint shown in Eq. (24), we could numerically obtain $\lambda_m(T-1)$ and hence $m_i(T-1)$ for all i.

Similar to the above operation, when $\underline{\delta_i^p(t)} = 0$ and $\overline{\delta_i^p(t)} = 1$ for all i and $t \in [0, T]$, we have

$$\delta_i^p(t) = \frac{\lambda_{\delta^p}(t) + y_i(t) - 2\varepsilon + \sqrt{(\lambda_{\delta^p}(t)+y_i(t))^2 + 4\varepsilon^2}}{2(\lambda_{\delta^p}(t)+y_i(t))}, \tag{31}$$

where

$$y_i(t) = \lambda_i^{PV}(t+1)p_i^{PV}(t) - \lambda_i^{DV}(t+1)p_i^{PV}(t).$$

Through Eq. (31), Eq. (25) and $\lambda_i^{PV}(T)$ and $\lambda_i^{DV}(T)$, we could obtain $\lambda_{\delta^p}(T-1)$ and $\delta_i^p(T-1)$.

(3) Using above computed boundary values of Lagrange multipliers and $m_i(T-1)$ and $\delta_i^p(T-1)$, propagate Eqs. (29a)-(29h) backward to compute the values of Lagrange multipliers and $m_i(t)$ and $\delta_i^p(t)$ at all time.

- Eqs. (29a), (29d), (29e), (29f), (29b) and (29c) in turn assign $\lambda_i^{US}(t)$, $\lambda_i^{DV}(t)$, $\lambda_i^q(t)$, $\lambda_i^r(t)$, $\lambda_i^{UI}(t)$ and $\lambda_i^{PV}(t)$ respectively;
- Eq. (29g) gives $\lambda_m(t-1)$ and $m_i(t-1)$ based on Eq. (30), Eq. (24), $\lambda_i^{US}(t)$, $\lambda_i^{UI}(t)$ and $\lambda_i^{PV}(t)$;
- Eq. (29h) gives $\lambda_{\delta^p}(t-1)$ and $\delta_i^p(t-1)$ based on Eq. (31) and Eq. (25), $\lambda_i^{PV}(t)$ and $\lambda_i^{DV}(t)$;.

(4) Update the values of $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ and then go back to Step (1).

The details of the MMCA-based optimization is also described in Algorithm 1.

It can be found that the forward-backward propagation algorithm is simple to be implemented. Both the forward propagation of Eqs. (17)-(22) and the backward propagation of Eq. (29) have a linear complexity $O(NT)$, and the number of

Algorithm 1 MMCA-Based Optimization Algorithm

Initialize

$\{m_i(t)\}_{i,t} \leftarrow$ random value, $\{\delta_i^p(t)\}_{i,t} \leftarrow$ random value, counter$\leftarrow$0;

do

for $t = 0$ to T

compute $\{p_i^{US}(t)\}_i$, $\{p_i^{UI}(t)\}_i$, $\{p_i^{PV}(t)\}_i$, $\{p_i^{DV}(t)\}_i$, $\{q_i(t)\}_i$ and $\{r_i(t)\}_i$ based on Eqs. (17)-(22) in turn;

for $t = T$ to 0

compute $\{\lambda_i^{US}(t)\}_i$, $\{\lambda_i^{DV}(t)\}_i$, $\{\lambda_i^{q}(t)\}_i$, $\{\lambda_i^{r}(t)\}_i$, $\{\lambda_i^{UI}(t)\}_i$ and $\{\lambda_i^{PV}(t)\}_i$ based on Eqs. (29a), (29d), (29e), (29f), (29b) and (29c) in turn;

compute $\lambda_m(t-1)$ and $\{m_i(t-1)\}_i$ based on Eqs. (29g) and (30);

compute $\lambda_{\delta^p}(t-1)$ and $\{\delta_i^p(t-1)\}_i$ based on Eqs. (29h) and (31);

counter$\leftarrow$counter+1;

while $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ are not convergence && counter$<$*Step*;

return $\{m_i(t)\}_{i,t}$, $\{\delta_i^p(t)\}_{i,t}$.

iterations is typically small and can be controlled. Therefore, the proposed algorithm is of modest computational complexity and can be applied to large scale networks. In addition, through a large number of experiments which are performed in the next section, we find the MMCA-based optimization algorithm often quickly converges to a unique optimal solution within a few forward-backward iterations for small scale networks. While for large scale networks, the optimization algorithm no longer always converges to a unique optimal solution, but jumps between several local optimal solutions after a few iterations. In this case, we can get the patch allocation solutions by repeating the optimization algorithm a fixed number of iterations (we limit the number of iterations to be 8 in present work).

VI. Simulation Results

In Fig. 7, we verify the efficiency of simultaneously optimizing $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ in MMCA-based optimization algorithm. We compare the results by simultaneous optimization versus those by random allocation and single-parameter optimization. More specifically, random allocation refers to assign random values for $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ under the constrains shown in Eqs. (24)-(27). The single-parameter optimization indicates that only a single set of parameters, $\{m_i(t)\}_{i,t}$ or $\{\delta_i^p(t)\}_{i,t}$, is optimized which could be deduced from the MMCA-based optimization procedure easily. When optimizing a single parameter, we let the other one follows a random allocation. In panel (a), we consider constrains of $B_m(t) = \sum_i m_i(t) = 0.03N$ and $B_{\delta^p}(t) = \sum_i \delta_i^p(t) = 0.4N$ for time $t \geq t_c$, both of which equal 0 for time $t < t_c$. In this sense, one sees that the simultaneous optimization performs the best. The single-parameter optimization gives the secondary best performance, but we cannot deduce which one is obviously better between optimizing $\{m_i(t)\}_{i,t}$ or $\{\delta_i^p(t)\}_{i,t}$. The random allocation has the weakest performance.

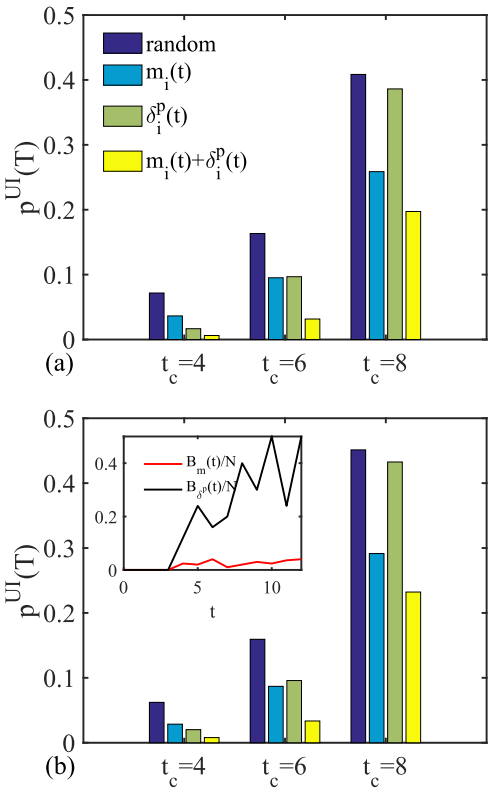

Fig. 7. The comparison of different optimization schemes. "random" refers to random allocation, that is, no optimization is performed. "$m_i(t)$" and "$\delta_i^p(t)$" indicate the optimization results of single-parameter optimization for $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$ respectively. "$m_i(t) + \delta_i^p(t)$" represents the simultaneous optimization of $\{m_i(t)\}_{i,t}$ and $\{\delta_i^p(t)\}_{i,t}$. In panel (a), the constraints $B_m(t) = 0.03N$ and $B_{\delta^p}(t) = 0.4N$ are constant for all time $t \geq t_c$. Panel (b) uses variable constraints which are shown in insets. For (a) and (b), we use $\beta^v = 0.3$, $\beta^p = 0.3$, $\delta^v = 0.2$ and $T = 12$. Both VL and PL are SF network with average degree $\langle k^p \rangle = \langle k^v \rangle = 4$.

Panel (b) considers the case where the constrains could vary with time. The simultaneous optimization is still the best which further illustrates the superiority of the proposed MMCA-based optimization algorithm.

We also verify the effectiveness of MMCA-based optimization algorithm by comparing with other heuristic strategies including degree centrality-based algorithm and two kinds of greedy strategies ((t + 1)-greedy and T-greedy).

(1) The degree centrality-based algorithm does not take into account the process of virus propagation and patch distribution. It just exploits the power of centrality of network in influencing the spreading dynamics. Specifically, at each time step $t \geq t_c$, the central node distributes patches to the first $B_m(t)$ largest degree nodes that have not been immunized and let the first $B_{\delta_p}(t)$ smallest degree nodes be the disabled nodes.

(2) The (t+1)-greedy algorithm aims to minimize the virus propagation at the next time step only by allocating the patches at the current time. At each time step $t \geq t_c$, it first iteratively chooses the node, patched by the central nodes, which could lead to the smallest $P^{UI}(t+1)$ until the constraint $B_m(t)$ is satisfied, then iteratively chooses the disabled nodes, which

TABLE I

COMPARATIVE RESULTS OF THE MMCA-BASED OPTIMIZATION ALGORITHM AND OTHER HEURISTIC STRATEGIES. N AND M ARE THE NUMBER OF NODES AND LINKS OF EACH NETWORK, RESPECTIVELY. COLUMNS 4-8 SHOW THE CORRESPONDING VALUE OF $P^{UI}(T)$ OF THE STRATEGY ON DIFFERENT NETWORK INSTANCES. THE PARAMETERS OF THE EXPERIMENT ON ALL NETWORK INSTANCES ARE SAME, WHERE $\beta^v = 0.3$, $\beta^p = 0.2$, $\delta^v = 0.1$, $t_c = 3$ AND $T = 7$, $B_m(t) = 0.03N$ AND $B_{\delta^p}(t) = 0.3N$ FOR ALL TIME $t \geq t_c$. THE SYMBOL '-' INDICATES THAT WE CAN NOT GET VALID RESULTS WITHIN THE ACCEPTABLE COMPUTATION TIME

Networks	N	M	random	degree	(t+1)-greedy	T-greedy	MMCA
AS (PL) SF_1 (VL)	767	1734 2297	0.4346	0.2695	0.3571	0.1795	0.1740
Email (PL) SF_2 (VL)	1133	5451 2266	0.1680	0.0845	0.0716	0.0368	0.0355
α-RoadEU (PL) $\beta - SF_3$ (VL)	1174	1417 2343	0.4124	0.3089	0.1464	0.1383	0.1342
α-PPI (PL) $\beta - SF_4$ (VL)	2361	6646 4721	0.2577	0.1768	0.0764	0.0485	0.0441
α-HepTh (PL) $\beta - SF_5$ (VL)	9877	25998 78957	0.5681	0.5255	-	-	0.4536
$\beta - SF_6$ (PL) α-PGP (VL)	10680	10696 24316	0.2268	0.1268	-	-	0.0354
α-Author (PL) $\beta - SF_7$ (VL)	23133	198110 323752	0.4601	0.4116	-	-	0.3657
P2P (PL) $\beta - SF_8$ (VL)	62586	147892 250330	0.4447	0.2925	-	-	0.1069

could lead to the smallest $P^{UI}(t+1)$ until the constraint $B_{\delta_p}(t)$ is satisfied.

(3) The whole operation of the T-greedy algorithm is very similar to the (t + 1)-greedy, only with different time horizon of optimization. The patch allocation at each time step of the T-greedy strategy aims to minimize $P^{UI}(T)$.

The experiments are performed on a series of different multiplex networks, each of which is formed by randomly connecting an artificial SF network (generated by BA scale free model [75]) and a real-world network (including Autonomous Systems (AS [76]), email communication network (Email [77]), European express road network (RoadEU [78]), protein-protein interaction network (PPI [79]), collaboration network of high energy physics authors (HepTh [76]), interaction network of users of the Pretty Good Privacy (PGP [80]), collaboration network of condensed-matter authors (Author [76]), peer-to-peer interaction network (P2P [81]).

From the experiment results shown in Table 1, our MMCA algorithm performs the best in all network instances. The T-greedy algorithm performs well and is just slightly weaker than MMCA algorithm in small scale networks, while it is computationally expensive such that we can not get valid results within the acceptable computation time for large scale networks. Similarly, the (t + 1)-greedy algorithm can not be applied to large scale networks. Though the degree-based algorithm is easy to operate and can be applied to large networks, its performance is relatively poor. The random allocation has the weakest performance.

VII. CONCLUSION

In this paper, for effectively restraining the virus propagation, we presented a mixed strategy of patch distribution which is formalized by a network and virus-patch model. The model contains a central node distributing patches to network nodes directly and a two-layer networks formed by virus propagation layer and patch dissemination layer to support the virus propagation and patch dissemination respectively. Based on proposed model, we analyzed the impacts of patch distribution on virus propagation, revealing some interesting insights of how to better restrain the virus propagation via patch distribution. It was observed that virus-patch model with small t_c and large m and η_p, and PL with large $\langle k^p \rangle$ and positive correlated with VL are more efficient in restraining the virus propagation. Furthermore, considering the constrains of capacity of central node and the bandwidth of networks, we proposed an optimal allocation scheme which could simultaneously optimize the values of multiple dynamical parameters related with patch distribution. The scheme is simple to be implemented and is of a modest computational complexity. By comparing the proposed method versus other strategies, we verify the efficacy of the proposed optimization scheme.

As for our future work, we will investigate discrete optimal allocation scheme of patches. Specifically, we should identify the concrete nodes that need to be patched at each time step under the given budgets, which is more practical and easier to be carried out.

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