RESOURCES ALLOCATION STRATEGIES OF DISEASE CONTROL IN MULTIPLEX NETWORKS

Bo Chen, Chun Yang[†]

School of Mathematical Science
University of Electronic Science and Technology of China
Chengdu 611731, China

Chuanji Fu, Yachun Gao, Hongchun Yang

School of Physics, University of Electronic Science and Technology of China
Chengdu 611731, China

(Received February 10, 2020; accepted June 3, 2020)

During the prevention and control of epidemics, both disease-related awareness diffusion and disease treatment require resources investment, so it is crucial to investigate the investment and allocation strategy of resources. Here, we propose an epidemiological model in the two-layer multiplex networks to study the interplay between disease and awareness under resource control. In this model, a part of the resources is used for disease treatment, and the other is used to facilitate the diffusion of awareness, with an adjustable parameter α setting to allocate the resources. First, we establish the evolutionary equations for different states and obtain the epidemic threshold of disease based on the microscopic Markov chain approach. Then, we conduct numerical simulations and find that stronger heterogeneity of the two-layer networks results in smaller epidemic threshold. Intriguingly, we find that there are optimal allocation coefficients in different multiplex networks structures and sizes. Finally, we find that the optimal allocation coefficient decreases with the increase of the immune degree.

DOI:10.5506/APhysPolB.51.1785

1. Introduction

Every large-scale outbreak of an epidemic, such as Ebola [1], SARS [2], H7N9 [3], HIV [4], *etc.*, severely threatens the lives of people in the outbreak

[†] Corresponding author: yangchun517@163.com

areas and causes a serious economic burden [5, 6]. Therefore, the underlying mechanisms and control strategies of epidemic spreading have become two main research topics in the field of spreading dynamics [7–14].

In the study of the underlying mechanisms of epidemic spreading, people have noticed the interplay between disease and disease-related awareness (disease-related awareness usually refers to some behavioral response of the disease, such as using vaccines, wearing masks, enhancing physique, *etc.* [15]). Funk *et al.* [15] first studied the impact of awareness diffusion on disease spreading, and found that the awareness diffusion can result in a lower size of epidemic outbreak, but does not affect the epidemic threshold in a well-mixed human population. Sahneh *et al.* [16] investigated how information dissemination can help to enhance the resilience of a population against epidemic spreading and found the optimal information dissemination network. Granell *et al.* [17] established a UAU (unaware–aware–unaware)–SIS (susceptible–infected–susceptible) model to mimic the interaction between disease and awareness. They analyzed the coupled dynamical process of awareness and disease, and found a metacritical point, at which the diffusion of awareness is able to control the onset of the epidemics. For a further research, they studied the impact of mass media on the final outcome of the epidemic incidence and found that the presence of mass media makes the metacritical point disappear [18].

In the research on the control strategies of epidemic spreading, some researchers have studied the effects of resources on disease spreading [19–21]. Chen *et al.* [22] explored the interaction between resource allocation in a social layer and disease spreading in the contact layer of a multiplex network. They found that when the edge overlap between two layers exceeds a critical value, the phase transition type transforms from hybrid to continuous. To investigate the impact of hybrid resource (local resource and global resource) allocation strategy on disease spreading dynamics, Chen *et al.* [23] did a further research. Their results showed that a global resource allocation has more advantages in suppressing epidemic spreading than local allocation. Jiang *et al.* [24] investigated the impact of resource amount on epidemic control in a two-layer network. They obtained the threshold of resource and discovered that the connection strength of networks will lead to the change of the phase transition type.

However, when investigating the interaction among resources, awareness, and disease spreading, these researchers only consider the effects of awareness or the impacts of resources on disease spreading. The effect of suppressing epidemics spreading by promoting the diffusion of disease-related awareness through investing resource has not been taken into account. To effectively suppress the spread of epidemic, we hold the opinion that public health resources can be divided into two parts, one for promoting the diffusion of

disease-related awareness and the other for disease treatment. Hence, to explore the effective investment of public health resources, we propose an epidemiological model that couples awareness diffusion and disease spreading under resources control in two-layer multiplex networks. In this model, a bias parameter α is introduced to regulate the allocation of resources between disease treatment and awareness diffusion. First, we use the microscopic Markov chain approach (MMCA) [25, 26] to analytically obtain the epidemic threshold of epidemic outbreak. Then we simulate the spread of epidemic in two-layer multiplex networks with different structures and find that the epidemic threshold decreases as the heterogeneity of the networks increases. More interestingly, optimal resource allocation coefficients are observed under different multiplex networks structures and sizes. On the other hand, the value of these coefficients suggests that to the maximum control of the outbreak of epidemic, most of the public health resources should be allocated to disease treatment. Finally, we find that the value of the optimal allocation coefficient decreases as the immune degree increases. Our work considers the impact of resources and awareness on disease spreading simultaneously, which may not only shed some light on future theoretical research on epidemiology, but also provides a reference for policymakers to formulate public resource allocation schemes.

2. Model description

In our model, the two-layer multiplex networks [17] are adopted to describe two competitive spreading process: the diffusion of disease-related awareness will inhibit the spread of disease, while the spread of disease may lead to the emergence of the new infected nodes (aware individuals) and then promotes the diffusion of disease-related awareness. Therefore, the two-layer multiplex networks can be built as follows (see Fig. 1): the upper layer (physical layer) denotes the network of physical contacts where the spread of disease happens, and the lower layer (virtual layer) represents the network of virtual contacts where the disease-related awareness diffusion happens. In the two-layer networks, each layer has N nodes and the set of nodes in the physical layer is the same as that in the virtual layer. While the two layers have different links, the information can be disseminated through the links in not only physical contacts network but also online social contacts network (*e.g.* Facebook, Twitter, Weibo, *etc.*). Therefore, the structure of virtual layer can be built by adding some new edges randomly (nonoverlapping with the original ones) on the basis of the structure of physical layer.

On the physical layer, we adopt the classic susceptible–infected–susceptible (SIS) model [27] to mimic the spread of disease. Every node in this layer can only exist in susceptible state (S) or infected state (I). At each time step, a susceptible node will be infected by its infected neighbor with a

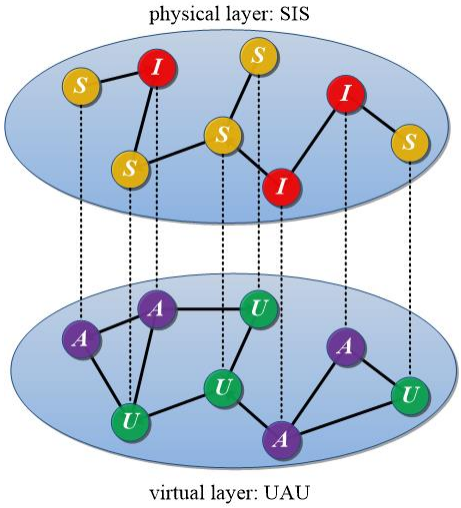

Fig. 1. (Color online) Schematic representation of the two-layer multiplex networks used in this work. The upper layer (physical layer) represents the network of disease spreading, in which each node can only exist one of two possible states: susceptible (S, orange nodes) or infected (I, red nodes). The lower layer (virtual layer) means the network of awareness diffusion, where nodes have two possible states: unaware (U, green nodes) or aware (A, purple nodes).

probability β. Meanwhile, an infected node will return to susceptible state with a probability μ. Since the recovery of infected nodes needs to consume resources (*e.g.* drug development, medical equipment purchases, *etc.*), we set the recovery rate μ as a function of the resource amount. On the virtual layer, an unaware–aware–unaware (UAU) process [28] is applied to model the awareness diffusion, which is similar to the SIS epidemiological model. In this layer, each node is in aware state (A) or unaware state (U). At each time step, an unaware node will become aware with a probability λ if it is connected to an aware neighbor. We assume that unaware individuals will not become aware spontaneously and must be informed by their aware neighbors before reacting to diseases (*e.g.* wearing protective mask, using vaccine, *etc.*). Since the behavioral response of disease depends on resources, we also set the awareness diffusion rate λ as a function of the resources amount. At the same time, we assume that an aware node will become unaware with a probability δ and denote δ as the awareness forgetting rate.

In this model, we set R as a total amount of resources on the epidemic control, which is divided into two parts according to an allocation coefficient α. One part $\alpha \times R$ of resources is used to enhance the rate of disease-related awareness diffusion in the virtual layer, and the remaining part $(1-\alpha)\times R$ of resources is used to improve the rate of disease recovery in

the physical layer. In the absence of public resources, nodes have an initial rate of disease recovery (μ_0) [29] and awareness diffusion (λ_0). To simplify the model, we assume $\mu_0 = 0$ and $\lambda_0 = 0$. Based on Refs. [20–23, 30], we set the disease recovery rate and the awareness diffusion rate as

$$\mu = 1 - \mathrm{e}^{-(1-\alpha)R}, \tag{1}$$

$$\lambda = 1 - \mathrm{e}^{-\alpha R}. \tag{2}$$

It should be noted that μ and λ denote the average disease recovery rate and the average awareness diffusion rate for all the individuals from the beginning to the end of the epidemic, respectively. The value of public resources R can increase the value of μ and λ. If the resources are used up before the end of epidemic, it means a small value of R. According to Eqs. (1) and (2), a small value of μ and λ will be for each time step.

Using Refs. [17, 18], we define the infection rate of susceptible node with and without disease-related awareness as β^{A} and β^{U} ($\beta^{\mathrm{U}} = \beta$), respectively. Moreover, they satisfy the following equation:

$$\beta^{\mathrm{A}} = \gamma\beta^{\mathrm{U}}, \tag{3}$$

where γ ($0 \leq \gamma < 1$) is called immune degree, which denotes the immunity of aware individuals to epidemic. In the particular case of $\gamma = 0$, the aware individuals are completely immune to the epidemic.

3. MMCA theoretical analysis

According to our model, each node i can be in one of the three states at time t: unaware and susceptible (US), aware and susceptible (AS), or aware and infected (AI), whose probabilities can be denoted by $p_i^{\mathrm{US}}(t)$, $p_i^{\mathrm{AS}}(t)$ and $p_i^{\mathrm{AI}}(t)$, respectively. Note that the state unaware and infected (UI) is not supposed to exist [17]. The reason is that an individual will be compelled to respond to epidemic after infected (*e.g.* receiving treatment, being quarantine, *etc.*), that is, the infected individual will be in an aware state immediately. In our model, the normalization condition $p_i^{\mathrm{US}}(t)+p_i^{\mathrm{AS}}(t)+p_i^{\mathrm{AI}}(t) \equiv 1$ holds for all time steps. Here, we denote elements in the adjacency matrix of physical layer (virtual layer) by a_{ij} (b_{ij}). If node i is connected to node j, corresponding element in the adjacency matrix is $a_{ij} = 1$ ($b_{ij} = 1$), otherwise $a_{ij} = 0$ ($b_{ij} = 0$). After that, on the virtual layer, we define the probability for an unaware node i not changing the state to aware at time t as $r_i(t)$. On the physical layer, $q_i^{\mathrm{U}}(t)$ and $q_i^{\mathrm{A}}(t)$ denote the probability for unaware and aware susceptible node i not being infected by any infected neighbors at time t respectively. Assuming the absence of dynamical correlations [31], we have the following formulae:

$$\begin{cases} r_i(t) = \prod_j \left[1 - b_{ji} p_j^{\mathrm{A}}(t)\lambda\right] , \\ q_i^{\mathrm{U}}(t) = \prod_j \left[1 - a_{ji} p_j^{\mathrm{AI}}(t)\beta^{\mathrm{U}}\right] , \\ q_i^{\mathrm{A}}(t) = \prod_j \left[1 - a_{ji} p_j^{\mathrm{AI}}(t)\beta^{\mathrm{A}}\right] , \end{cases} \tag{4}$$

where $p_j^{\mathrm{A}}(t)$ denotes the probability of a node j being in the aware state, which satisfies $p_j^{\mathrm{A}}(t) = p_j^{\mathrm{AS}}(t) + p_j^{\mathrm{AI}}(t)$.

According to Ref. [17], we provide the transition probability trees for the states of any node i from time t to $t+1$, see Fig. 2, where UI state is an intermediate state which is set in the analysis process of states transition.

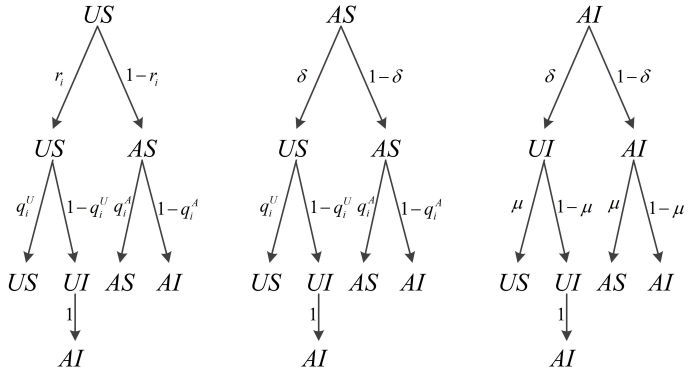

Fig. 2. Transition probability trees for different states of the UAU–SIS model per time step. The states include US (unaware and susceptible), AS (aware and susceptible), AI (aware and infected). In the transition probability trees, r_i represents the probability not varying from unaware to aware state, δ means the awareness forgotten (from aware to unaware state) probability, q_i^{U} and q_i^{A} denote the probability for unaware and aware susceptible node i not being infected by any infected neighbors, and μ denotes the probability for an infected node returns to susceptible state. We assume a node will go through the awareness diffusion process at first.

As shown in Fig. 2, the dynamically evolutionary equations of node i for each state can be described as follows by using the microscopic Markov chain approach (MMCA):

$$p_i^{\mathrm{US}}(t+1) = p_i^{\mathrm{US}}(t) r_i(t) q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AS}}(t)\delta q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AI}}(t)\delta\mu , \tag{5}$$

$$p_i^{\mathrm{AS}}(t+1) = p_i^{\mathrm{US}}(t)(1 - r_i(t)) q_i^{\mathrm{A}}(t) + p_i^{\mathrm{AS}}(t)(1-\delta) q_i^{\mathrm{A}}(t) + p_i^{\mathrm{AI}}(t)(1-\delta)\mu , \tag{6}$$

$$\begin{aligned} p_i^{\mathrm{AI}}(t+1) &= p_i^{\mathrm{US}}(t)\left[r_i(t)\left(1-q_i^{\mathrm{U}}(t)\right)+(1-r_i(t))\left(1-q_i^{\mathrm{A}}(t)\right)\right] \\ &\quad +p_i^{\mathrm{AS}}(t)\left[\delta\left(1-q_i^{\mathrm{U}}(t)\right)+(1-\delta)\left(1-q_i^{\mathrm{A}}(t)\right)\right] \\ &\quad +p_i^{\mathrm{AI}}(t)\left[\delta(1-\mu)+(1-\delta)(1-\mu)\right]. \end{aligned} \tag{7}$$

When $t \to +\infty$, *i.e.*, the epidemic spreading reaches a steady state, we have $p_i^{\mathrm{US}}(t+1) = p_i^{\mathrm{US}}(t) = p_i^{\mathrm{US}}$, $p_i^{\mathrm{AS}}(t+1) = p_i^{\mathrm{AS}}(t) = p_i^{\mathrm{AS}}$, and $p_i^{\mathrm{AI}}(t+1) = p_i^{\mathrm{AI}}(t) = p_i^{\mathrm{AI}}$. After that, we compute the epidemic threshold of disease β_c ($\beta_c = \beta_c^{\mathrm{U}}$).

If $\beta < \beta_c$, the disease will not be prevalent. Otherwise, the disease breaks out and persists in the population. When β approaches the epidemic threshold, the probability of nodes to be infected is close to zero, namely, $p_i^{\mathrm{AI}} = p_i^{\mathrm{I}} = \epsilon_i \ll 1$. Accordingly, q_i^{U} and q_i^{A} are approximated as

$$q_i^{\mathrm{U}} \approx 1-\beta^{\mathrm{U}}\sum_j a_{ji}\epsilon_j\,, \tag{8}$$

$$q_i^{\mathrm{A}} \approx 1-\beta^{\mathrm{A}}\sum_j a_{ji}\epsilon_j\,. \tag{9}$$

Furthermore, taking the above approximate equations into Eqs. (5)–(6) and omitting the $o(\epsilon_i)$ terms, we get

$$p_i^{\mathrm{US}} = p_i^{\mathrm{US}} r_i + p_i^{\mathrm{AS}}\delta\,, \tag{10}$$

$$p_i^{\mathrm{AS}} = p_i^{\mathrm{US}}(1-r_i) + p_i^{\mathrm{AS}}(1-\delta)\,. \tag{11}$$

Then, substituting Eqs. (8)–(11) into Eq. (7), a simple formula is obtained

$$\begin{aligned} \mu\epsilon_i &= p_i^{\mathrm{US}}\left[r_i\beta^{\mathrm{U}}\sum_j a_{ji}\epsilon_j + (1-r_i)\beta^{\mathrm{A}}\sum_j a_{ji}\epsilon_j\right] \\ &\quad +p_i^{\mathrm{AS}}\left[\delta\beta^{\mathrm{U}}\sum_j a_{ji}\epsilon_j + (1-\delta)\beta^{\mathrm{A}}\sum_j a_{ji}\epsilon_j\right] \\ &= \left(p_i^{\mathrm{AS}}\beta^{\mathrm{A}} + p_i^{\mathrm{US}}\beta^{\mathrm{U}}\right)\sum_j a_{ji}\epsilon_j\,. \end{aligned} \tag{12}$$

Note that $p_i^{\mathrm{AS}}+p_i^{\mathrm{AI}}+p_i^{\mathrm{US}} = 1$, where $p_i^{\mathrm{AS}}+p_i^{\mathrm{AI}} = p_i^{\mathrm{A}}$. Since $p_i^{\mathrm{AI}} = \epsilon_i \ll 1$, we can get $p_i^{\mathrm{AS}} \approx p_i^{\mathrm{A}}$ and $p_i^{\mathrm{US}} = 1-p_i^{\mathrm{AS}}-p_i^{\mathrm{AI}} = 1-p_i^{\mathrm{A}}$. Therefore, Eq. (12) can be further simplified as

$$\sum_j \left\{\left[1-(1-\gamma)p_i^{\mathrm{A}}\right]a_{ji} - \frac{\mu}{\beta^{\mathrm{U}}}\delta_{ji}\right\}\epsilon_j = 0\,, \tag{13}$$

where δ_{ji} is the element of the identity matrix I. We introduce a new matrix H whose elements fulfill

$$h_{ji} = \left[1 - (1-\gamma)p_i^{\mathrm{A}}\right] a_{ji}\,. \tag{14}$$

Thus, Eq. (13) can also be expressed as

$$\left(H - \frac{\mu}{\beta^{\mathrm{U}}} I\right) \epsilon = 0\,, \tag{15}$$

where $\epsilon = (\epsilon_1, \epsilon_2, \ldots, \epsilon_N)^T$.

The nontrivial solutions of Eq. (15) are eigenvectors of H, whose eigenvalues are equal to μ/β^{U}. Accordingly, the epidemic threshold is equal to the minimum value of β^{U} satisfying Eq. (15). Let $\Lambda_{\max}(H)$ denote the largest eigenvalue of H, and substitute Eq. (2) into Eq. (15). Then, the epidemic threshold is described as

$$\beta_c^{\mathrm{U}} = \frac{1 - \mathrm{e}^{-(1-\alpha)R}}{\Lambda_{\max}(H)}\,. \tag{16}$$

According to Eqs. (14) and (16), β_c^{U} depends on the allocation coefficient (α), the amount of resources (R), the structure of physical layer (a_{ij}), the immune degree (γ), and the spreading dynamics on the virtual layer (p_i^{A}). The value of p_i^{A} is further determined by the structure of virtual layer and the parameter δ.

4. Numerical results

To examine how resource allocation strategies affect epidemic spreading, we perform extensive numerical simulations in the two-layer multiplex networks. Due to the limitation of the computer storage level, in our experiments, the network sizes range from $N = 1000$ to $N = 10\,000$. To build the Erdős–Rényi random network (ER network) of two-layer multiplex networks, we adopt the method described in Ref. [32], that is, the edges among nodes are added with probability $p = \frac{\langle k \rangle}{N-1}$. Among these, $\langle k \rangle$ is the average degree of ER network which is set artificial. To build the scale-free network (SF network) of two-layer multiplex networks, we use an uncorrelated configuration model (UCM) [33] with a given degree distribution $P(k) \sim k^{-\gamma_s}$, in which γ_s is the degree exponent. The degree exponent γ_s here is usually set between 2 and 3, and a smaller γ_s implies a stronger degree heterogeneity of network [34]. In the construction of scale-free network, the maximum degree is determined by the structural cut-off $k_{\max} \sim \sqrt{N}$ [35] and the minimum degree is set as $k_{\min} = 3$. In our experiments, the total amount of resources

is set as 1 unit, which indicates the overall resources invested by the government on disease prevention and control. The initial density of infected nodes in the physical layer is set as $\rho_0^{\mathrm{I}} = 0.01$, and the initial density of aware nodes in the virtual layer is set as $\rho_0^{\mathrm{A}} = 0.01$. To ensure the reliability of the experimental results, each final experimental data is obtained from the average result of 50 experiments under the same conditions.

4.1. Comparisons between MMCA analytical results and MC simulations

To verify the validity of theoretical analysis based on MMCA, we conduct Monte Carlo (MC) simulations and numerical iterations in the two-layer ER networks and the two-layer scale-free networks, respectively. The sizes of multiplex networks are both set as $N = 5000$. For ER networks, we use $\langle k \rangle = 7$ and for SF networks, $\gamma_s = 2.4$. To construct the virtual layer, we first set that this layer has the same adjacency matrix as the physical layer, and then we add randomly 2000 links in the virtual layer. We assume that the resource allocation coefficient $\alpha = 0.5$. Using the research method of Ref. [17], it is assumed that $\gamma = 0$ for the simplicity of study and then obtained $\beta^{\mathrm{A}} = 0$ according to Eq. (3). Under these assumptions, p_i^{A} and p_i^{I} are obtained at the stationary state by iterating Eqs. (5) to (7), and $\rho^{\mathrm{A}} = \frac{1}{N}\sum_i p_i^{\mathrm{A}}$ and $\rho^{\mathrm{I}} = \frac{1}{N}\sum_i p_i^{\mathrm{I}}$ represent the density of infected nodes and the density of aware nodes in MMCA, respectively.

Figure 3 (a) and (b) shows the results of disease spread and awareness diffusion in multiplex networks, respectively. Comparing the curves obtained by the two methods, we find good agreement between MMCA iterations and MC simulations in calculating the epidemic and awareness prevalence, no matter in the two-layer ER networks or the two-layer scale-free networks.

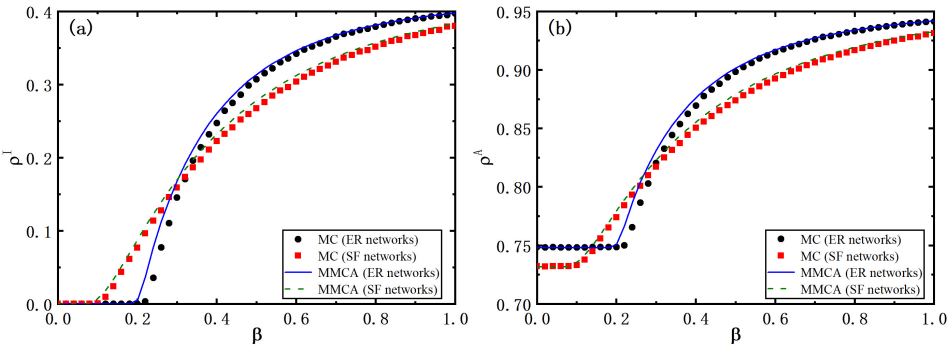

Fig. 3. Comparisons between MMCA analytical results and MC simulations. (a) and (b) represent ρ^{I} as a function of β, and ρ^{A} as a function of β in multiplex networks, respectively. The rest of the values of parameters are $\delta = 0.3$, $\gamma = 0$, $\alpha = 0.5$, $R = 1$.

We also conduct corresponding numerical simulations for different values of α (not shown in the paper), and find that the MMCA iterative method can well agree with MC simulations whatever α is. Our results show that the MMCA method has good accuracy in solving the proposed coupling dynamics problem. In addition, a slight discrepancy between MMCA iterations and MC simulations is observed, which may be caused by the hypothesis of the absence of dynamical correlations in the MMCA method.

4.2. Effects of network topology

To investigate the effects of network topology on epidemic spreading dynamics under our model, we explore the density of infected individuals ρ^{I} as a function of disease transmission rate β in different topological structures. When we investigate the impact of topological structure on the disease spreading, without loss of generality, the resource allocation coefficient is set as $\alpha = 0.5$, and the forgetting rate of awareness is set as $\delta = 0.3$. At the same time, for the sake of simplicity, if a node is aware of the disease, we assume that the node will not get infected, that is, $\gamma = 0$, and such a setting will not affect our exploration of the essence of the problem.

Figure 4 shows the disease spreading results in the two-layer ER networks with average degree $\langle k \rangle = 7$ (black squares) and the two-layer SF networks with different degree exponents using MC simulations. Observing Fig. 4, we see that the epidemic thresholds of disease in the two-layer scale-free networks are smaller than that in the two-layer ER networks under our model. On the other hand, note that the smaller the degree exponent of the two-layer scale-free networks is, the lower the epidemic threshold is, which

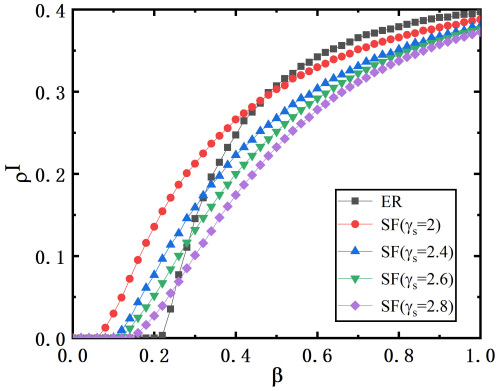

Fig. 4. The effects of network topology on epidemic spreading. The curves show the density of infected nodes ρ^{I} as a function of β in the two-layer ER networks and the two-layer SF networks with different degree exponents. Other parameters are set to be $\delta = 0.3$, $\gamma = 0$, $\alpha = 0.5$, $R = 1$.

is qualitatively the same as in single networks [8, 36]. This may be due to the fact that the scale-free network is heterogeneous, and when the degree exponent of the network decreases, the heterogeneity of network increases, and more hub nodes emerges. In this model, each susceptible individual will be in full contact with his or her infected neighbors, which means that the hub nodes will get infected with a greater probability than other small-degree nodes. If a hub node is infected, the infection risk of the susceptible nodes around it will increase. More hub nodes will further increase the infection probabilities of other nodes. Therefore, in our model, disease spreads more easily in a network with strong degree heterogeneity. This indicates that epidemic spreading in a strong heterogeneity of scale-free network requires more control.

4.3. Effects of resource allocation coefficient

In this section, we investigate the impact of resource allocation coefficient α on disease spreading. In the numerical experiments, we adopt the network setup and parameter settings (except for α) of Fig. 3 to examine the density of infected individuals ρ^{I} as a function of β and α in two-layer ER networks and two-layer scale-free networks. Figure 5 shows the phase diagrams of the relation among them.

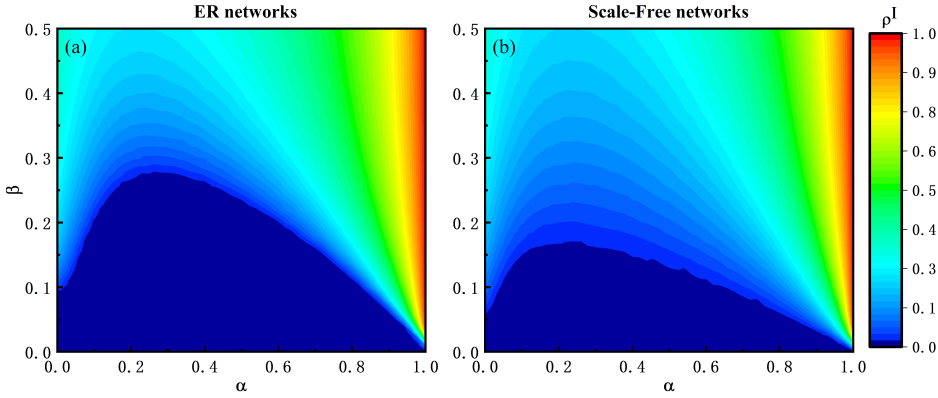

Fig. 5. Dependence of infection density ρ^{I} on parameters β and α. (a) and (b) represent the experiments in the two-layer ER networks and two-layer SF networks, respectively. Other parameters are set to be $\delta = 0.3$, $\gamma = 0$, $R = 1$.

Figure 5 (a) and (b) shows that in our model, there is a peak in the phase diagram of both the two-layer ER networks and the two-layer scale-free networks. Interestingly, the peak is between 0.2 and 0.3 in both multiplex networks. We define the resource allocation coefficient corresponding to the peak position as the optimal resource allocation coefficient α_{opt}. When $\alpha = \alpha_{\mathrm{opt}}$, the epidemic threshold of disease reaches the maximum

value, which means that such a resource allocation strategy can suppress the spread of epidemics to the greatest extent. By further observation in Fig. 5 (a) and (b), we see that when $\alpha < \alpha_{\text{opt}}$, the density of infected individuals ρ^{I} is significantly lower than that of $\alpha > \alpha_{\text{opt}}$. Such experimental results indicate that when the resource allocation coefficient cannot reach α_{opt}, it is a better allocation strategy to suppress the spread of epidemics by investing most of the resources for patients' recovery while a small amount of resources for improvement of susceptible individuals' awareness. It can also be seen from the figure that when $\alpha > \alpha_{\text{opt}}$, the larger α is, the more likely disease outbreaks. Especially, when $\alpha \to 1$, a small disease transmission rate β will lead to a large-scale outbreak of disease on the network. This phenomenon shows that, if the public resources completely focus on raising awareness, it cannot achieve the goal of suppressing epidemics spreading and may even cause a waste of resources in disease prevention and control. A possible reason for this phenomenon is that when the value of α is too large, almost all resources are devoted to improve disease-related awareness, resulting in the public resources on the treatment of disease not being able to meet the recovery needs of patients. Therefore, even a small disease transmission rate can lead to a large-scale outbreak of disease on the network. Meanwhile, we also notice a certain difference of the optimal resource allocation coefficient between the two-layer ER networks and the two-layer scale-free networks. However, the difference is unobvious, and both of optimal allocation coefficients are relatively small. This indicates that for no matter what kind of network structure, in the disease control, we cannot neglect to improve individuals' awareness of disease as has been proved in the previous study [15, 17] that awareness has an important influence on epidemic spreading. Therefore, it is necessary to invest a certain amount of resources to facilitate awareness diffusion. On the other hand, to suppress the outbreak of disease as a whole, we must focus on the treatment of patients and increase the investment in disease treatment.

To prove the robustness of the above results, we first explore how network structure affects the resource allocation strategy. In the numerical experiments, we adopt the network construction mode and parameter settings (except for α) of Fig. 3 (b) to explore the epidemic threshold β_c as a function of α in the two-layer scale-free networks with different degree exponents. The epidemic thresholds β_c are calculated by using Eq. (16), and Fig. 6 shows the relation curves between β_c and α. As can be seen from the figure, there is always a peak in the two-layer scale-free networks with different degree exponents, which means that optimal resource allocation coefficient exists in different network structures under our model. Moreover, we notice that in Fig. 6 the stronger the heterogeneity of the two-layer scale-free networks, the smaller the value of the optimal resource allocation coefficient, which indicates that we should allocate more public resources

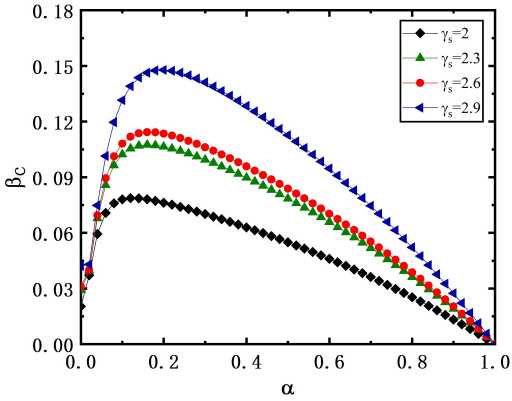

Fig. 6. The impact of network structure on the resource allocation strategy. The curves show the epidemic threshold β_c as a function of α in two-layer scale-free networks with different degree exponents. Other parameters are set to be $\delta = 0.3$, $\gamma = 0$, $R = 1$.

on disease treatment in a more heterogeneous network. A possible reason for this phenomenon is that the stronger the heterogeneity of network, the more hub nodes will emerge. In our model, the hub nodes are more likely to be infected or gain disease-related awareness than other small-degree nodes, thus the increase of hub nodes further increases the probabilities of other nodes getting infected and gaining disease-related awareness. Therefore, the density of infected individuals and the density of aware individuals are relatively large in the network with strong heterogeneity. As a result, to hold back the spread of epidemics, we should allocate more resources on disease treatment to reduce the scale of disease infection. At the same time, by comparing Fig. 5 and Fig. 6, we find that the values of optimal resource allocation coefficients in different network structures are all relatively small, which leads to the same conclusion as above.

We next investigate how the size of network influences the resource allocation strategy. We perform numerical experiments in multiplex networks with network sizes $N = 1000$, $N = 2000$, $N = 5000$, and $N = 10\,000$, respectively. In our experiments, the average degree of physical layer in two-layer ER networks are set as $\langle k \rangle = 7$, the degree exponents of physical layer in two-layer scale-free networks are set as $\gamma_s = 2.4$, and the structures of virtual layer are set to randomly add 400, 800, 2000, and 4000 new edges on the basis of the structure of physical layer. The parameter settings (except for α) are the same as in Fig. 3. Figure 7 shows the curves of epidemic threshold β_c as a function of α, and the epidemic threshold β_c is calculated by using Eq. (16). From Fig. 7 (a) and (b), there are peaks in curves of different network sizes, which indicates that the optimal resource allocation coefficient exists in different sizes of network in our model. In addition, we

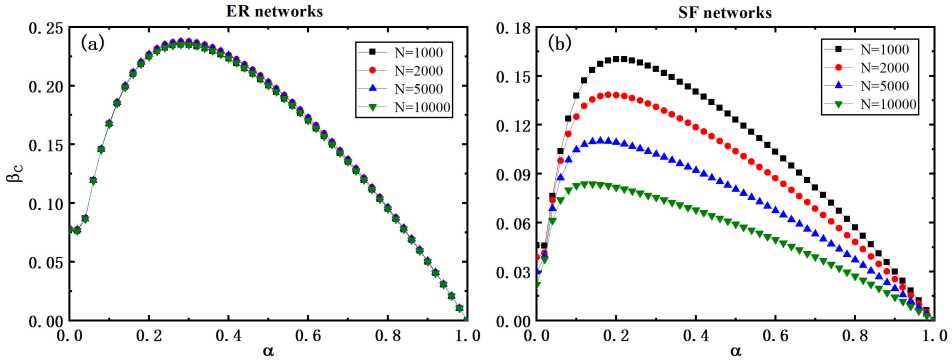

Fig. 7. The impact of network size on the resource allocation strategy in different multiplex networks. The curves of (a) and (b) show the epidemic threshold β_c as a function of α in two-layer ER networks and two-layer scale-free networks with different network size, respectively. Other parameters are set to be $\delta = 0.3$, $\gamma = 0$, $R = 1$.

find that the values of the optimal resource allocation coefficients in both the two-layer ER networks and the two-layer SF networks are close to the above experiments, all of which are relatively small. Such results show that our conclusion is valid at different network sizes. Moreover, we investigate the impact of resource allocation coefficient on disease spreading when values of δ are different, see Fig. 8. The theoretical result shows that the optimal resource allocation coefficient exists in different values of δ. It should be pointed out that when $\delta = 0$, all individuals will become aware as time goes on. According to our model and parameter settings, we obtained $\beta^{\mathrm{A}} = 0$, which means all individuals with awareness will not infect. Therefore, the optimal resource allocation coefficient does not exist.

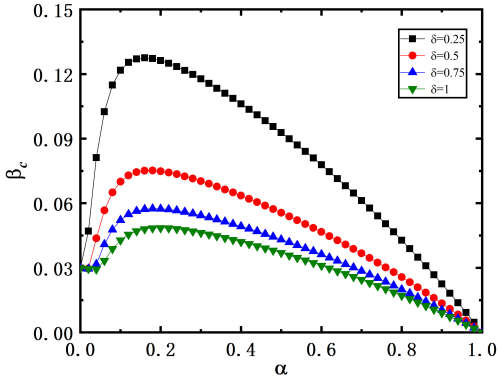

Fig. 8. The impact of awareness forgetting rate on the resource allocation strategy. The curves show the epidemic threshold β_c as a function of α in two-layer scale-free networks with different δ. Other parameters are set to be $\gamma = 0$, $R = 1$.

4.4. Effects of immune degree γ

It is worth mentioning that the above experiments have always set the individuals with awareness completely immune to the epidemic (*i.e.* $\gamma = 0$), which is obviously a very idealized setting. However, in real life, for almost all diseases, the individual can still get infected even if he or she has disease-related awareness. Therefore, in this section, we further investigate the spread of disease at different immune degrees. In our experiments of this part, the network setup and parameter settings (except for γ) are consistent with Fig. 3 (b).

Figure 9 (a)–(d) shows the phase diagram of the infection density ρ^{I} as a function of the two-parameter (β, α) space using MC simulations when $\gamma = 0.25$, $\gamma = 0.5$, $\gamma = 0.75$, and $\gamma = 0.9$, respectively. Note that there are peaks of the phase diagrams in Fig. 9 (a)–(d) that is similar to Fig. 5, and the resource allocation coefficient corresponding to the peak position decreases with the increase of immune degree γ. The results show that the

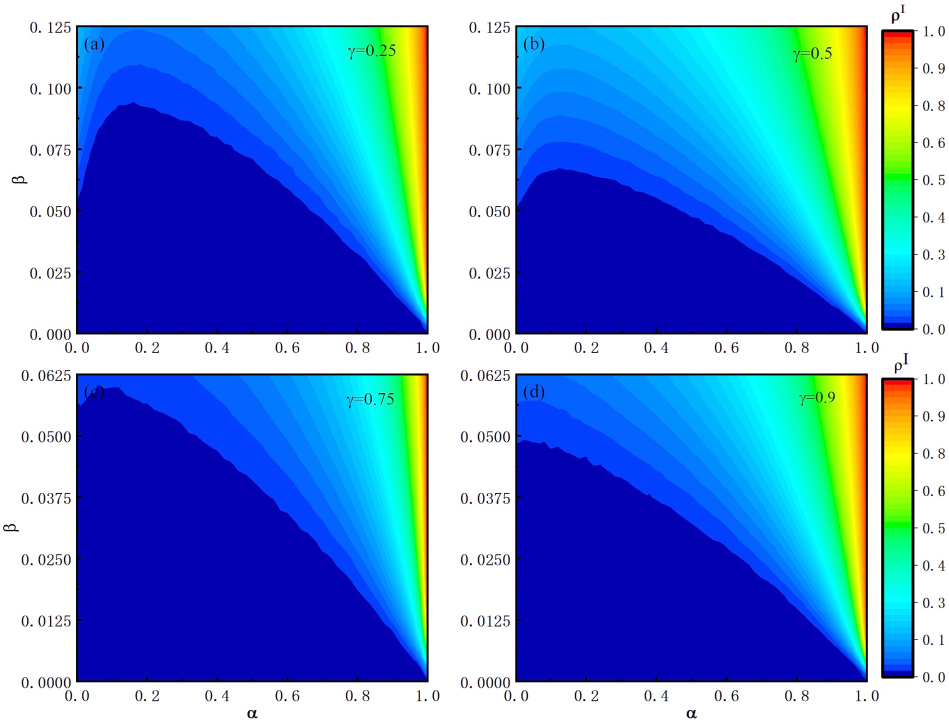

Fig. 9. Dependence of infection density ρ^{I} on parameters β and α. (a), (b), (c), and (d) represent the phase diagrams of $\gamma = 0.25$, $\gamma = 0.5$, $\gamma = 0.75$, and $\gamma = 0.9$ in the two-layer SF networks, respectively. Other parameters are set to be $\delta = 0.3$, $R = 1$.

optimal allocation coefficient of resources still exists in the case of $\gamma \neq 0$, and the value of the coefficient is still relatively small. On the other hand, the weaker the immunity of disease-related awareness to the epidemic is, the more resources we should allocate to the disease treatment. In particular, when $\gamma \approx 1$ in Fig. 9 (d), *i.e.*, there is no effect of disease-related awareness on disease spreading, the optimal allocation coefficient of resources is zero.

5. Discussion and conclusions

In this paper, we propose a UAU–SIS model in the two-layer multiplex networks to simulate the coupled dynamical process of disease and awareness under resource control. In our model, both the recovery rate of disease and the diffusion rate of disease-related awareness are related to the amount of public resources, and a parameter α is introduced to adjust the allocation of resources between them. Based on the microscopic Markov chain approach, we have established the dynamically evolutionary equations for different states and analytically derived the expression of the epidemic threshold of disease. The results of theoretical iterations are in good agreement with the results of Monte Carlo simulations when performing verification in network with different structures. From extensive numerical simulations, the epidemic threshold decreases gradually with the increasing of the heterogeneity of the two-layer networks. In particular, we have obtained optimal allocation coefficients α_{opt} in the two-layer ER networks and the two-layer scale-free networks. Besides, the values of α_{opt} are relatively small, which means that we should allocate more resources on disease treatment. Moreover, we have demonstrated the robustness of α_{opt} in two-layer scale-free networks with different heterogeneities and sizes. Finally, in our model, the larger the value of immune degree γ is, the smaller the value of the optimal allocation coefficient is.

Our findings reveal that the allocation of public resources between disease treatment and awareness diffusion plays a crucial role in suppressing the spread of epidemics. Therefore, when formulating disease prevention and control strategies, policymakers must consider the important influence of disease-related awareness on epidemic spreading, and a part of the resources should be invested to promote awareness diffusion. In addition, to suppress the outbreak of disease as a whole, it is necessary to give priority to the disease treatment, that is, most of the resources should be used to help patients recover.

In our study, the effects of public resources on disease treatment and awareness diffusion are considered, but in reality, social support from family and friends [22] also affects patients' recovery and awareness diffusion. Therefore, it will be an interesting topic to study the allocation strategies of

a hybrid resource between disease and awareness. In addition, our research is based on the SIS model, and the SIR (susceptible–infected–recovered) model [37] and other epidemiological models can be considered in future work.

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