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The impact of heterogeneous response on coupled spreading dynamics in multiplex networks

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HIGHLIGHTS

- We study the problem of epidemic spreading dynamics with the consideration that the alerting information of the disease may co-spread with the disease spreading process.
- Heterogeneous responses are considered when people learn the information of the disease.
- A heterogeneous mean-field theory is developed to described the behavior of coupled dynamics.

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ABSTRACT

Many recent studies have demonstrated that individual awareness of disease may significantly affect the spreading process of infectious disease. In the majority of these studies, the response of the awareness is generally treated homogeneously. Considering of diversity and heterogeneity in the human behavior which widely exist under different circumstances, in this paper we study heterogeneous response when people are aware of the prevalence of infectious diseases. Specifically, we consider that an individual with more neighbors may take more preventive measures as a reaction when he is aware of the disease. A suppression strength is introduced to describe such heterogeneity, and we find that a more evident heterogeneity may cause a more effective suppressing effect to the spreading of epidemics. A mean-field theory is developed to support the results which are verified on the multiplex networks with different interlayer degree correlation.

1. Introduction

Complex networks and dynamical processes on them have been of special concern in statistical physics and related disciplines [1–3]. As a typical dynamical process taking place on complex networks, epidemic spreading has received great attention along the development of complex network theory [4–9]. Existing works have considered various aspects on this issue, e.g. threshold models [10,11], immunization protocols [12,13], individual mobility patterns [14,15], duration of the disease [16].

Recently, instead of merely considering the direct contact between individuals, the effect of human responses with the awareness of disease spreading has been taken into consideration [17–22]. A natural consequence on the awareness of epidemics for the people is to take measures to prevent them from infection. By adopting multiplex networks model, where

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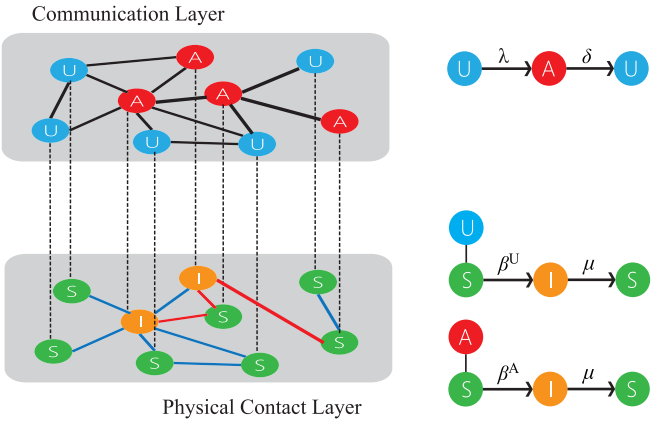

Fig. 1. Illustration of the coupled spreading process on a multiplex network with two layers. The information spreading process takes place on the upper layer where nodes have two possible states: unaware (U) or aware (A). The lower layer is supporting the spreading of epidemics, on which nodes are either susceptible (S) or infected (I). A pair of two nodes, one in the upper layer and the other in the lower layer, connected by a dashed link across the two layers represents the same individual.

nodes represent the individuals and connections in two different types represent the contact structure for the epidemic dynamics and information diffusion dynamics [23–25], respectively, Granell et al. [26] studied this effect of awareness and found the emergence of a metacritical point where the onset of epidemics is controlled by the diffusion of the information. Further studies show that this metacritical point may disappear after introducing a massive broadcast of awareness (mass media) [27]. Moreover, Wang et al. [28] found that the epidemic threshold increases when the communication spreads at a high rate, and the epidemic spreading could be suppressed through timely immunization of large-degree nodes.

While current studies have considered the effect of the awareness on the disease, the human reaction are treated homogeneously, i.e. the infection rate are changed uniformly when people learn the prevalence of the infectious disease. However, in reality, reactions of individuals may be diverse. For example, for those who have more friends may be more cautious to the epidemics, since they are more likely to contact with an infected neighbor. Consequently, they may adopt stricter preventive measures to protect themselves after acquiring information of the disease. In fact, similar degree-dependent infection effect has been considered on single layer networks [29] and metapopulation networks [30].

Here, we consider the situation that different individuals may adopt preventive measures as reactions to different extent according to the number of the neighbors they have. Specifically, a suppression strength is introduced to describe the heterogeneous extent of the reactions. We found that larger heterogeneity in the extent of such reaction may suppress the epidemic spreading more effectively. Moreover, the relation between the infection threshold and the suppression strength is obtained analytically, which reveals that the infection threshold increases with the increasing of the suppression strength and will be saturated when the suppression is large enough. Furthermore, our theoretical analysis also considers the networks with interlayer degree correlation, which is verified on the cases of full interlayer correlation and non-interlayer correlation.

2. Model

We start by considering a multiplex network which is composed of two layers (see Fig. 1), where one (the upper layer) is the communication layer where information spreads, and the other (the lower layer) is the physical contact layer where disease spreads. The two layers have exactly the same number of nodes N, where the degree of a node in the communication layer (physical contact layer) is denoted as k_1 (k_2). The joint degree distribution $P(k_1, k_2)$ represents the fraction of nodes whose number of neighbors is k_1 in communication layer and k_2 in physical contact layer, and the mean degrees of the communication layer and the physical contact layer are equal to $\langle k_1\rangle = \sum_{k_1,k_2} k_1 P(k_1, k_2)$ and $\langle k_2\rangle = \sum_{k_1,k_2} k_2 P(k_1, k_2)$, respectively.

In the communication layer, we use UAU model to describe the propagation of information between individuals. In this model, a node can be in one of the two states: A node in the awareness (A) state is the one who is aware of the infectious disease and may take protective measures to reduce the risk of being infected. Moreover, they may spread the information of the disease to those who are unaware of it. A node in the unawareness (U) state is the one who is unaware of the disease. They could become aware of the disease if they receive the information of the disease from their neighbors. Specifically, in each time step the information of disease could be transferred through an UA connection, i.e. a connection with a U state node and an A state node at its two ends, with probability λ. Besides, if a U state node is infected by the disease, he will automatically be aware of the disease. For a node in the A state he could recover to the U state with rate δ, which corresponds to the process that the related memory fades out.

On the physical contact layer, we use SIS model to describe the epidemic spreading process, in which each node is either susceptibility (S) or infected (I). If a susceptible node is in the U state, the infection rate for it through an infected neighbor

is β^{U}; but if he is in the A state, he will take preventive measures to protect himself resulting in a reduced infection rate β^{A} with $\beta^{\mathrm{A}} < \beta^{\mathrm{U}}$. In addition, each infected node may recover to be susceptible with rate μ. Distinct from a widely adopted scheme that β^{A} is varied uniformly with β^{U}, say $\beta^{\mathrm{A}} = \gamma\beta^{\mathrm{U}}$ with $\gamma \in [0, 1]$ [26], in this paper we consider that nodes with larger degree in the contact layer will be more precautious against the epidemics after being aware of the disease, as nodes with larger degree are in a more dangerous situation to get infected. Specifically, we consider that $\beta^{\mathrm{A}} = k_2^{-\alpha}\beta^{\mathrm{U}}$, where $\alpha \geq 0$ is the suppression strength and k_2 is the degree of the nodes in the physical contact layer. When $\alpha = 0$, it returns to the situation that the communication layer has no influence on the spreading of epidemics.

Combining the above, a node could be in four possible states, namely US (unaware and susceptible), AS (aware and susceptible), AI (aware and infected), and UI (unaware and infected). Since an infected node is deemed to be aware of the disease, the state UI actually does not exist. Therefore, we have the transition rules among the remaining three states as follows:

$$\mathrm{US} + \mathrm{AS} \xrightarrow{\lambda} \mathrm{AS} + \mathrm{AS}, \tag{1a}$$

$$\mathrm{US} + \mathrm{AI} \xrightarrow{\lambda} \mathrm{AS} + \mathrm{AI}, \tag{1b}$$

$$\mathrm{US} + \mathrm{AI} \xrightarrow{\beta^{\mathrm{U}}} \mathrm{AI} + \mathrm{AI}, \tag{1c}$$

$$\mathrm{AS} + \mathrm{AI} \xrightarrow{\beta^{\mathrm{A}}} \mathrm{AI} + \mathrm{AI}, \tag{1d}$$

$$\mathrm{AS} \xrightarrow{\delta} \mathrm{US}, \tag{1e}$$

$$\mathrm{AI} \xrightarrow{\mu} \mathrm{AS}, \tag{1f}$$

where conditions (1a) and (1b) describe the communication process that a node in the U state become an A state node with rate λ when he contacts with an A state neighbor, conditions (1c) and (1d) describe the process that a susceptible node is infected with infection rate β^{U} or β^{A} depending on whether he is aware of the disease or not, and conditions (1e) and (1f) describe the process that an A state node recovers to U state with rate δ and an infected node recovers to be susceptible with rate μ, respectively. In this paper, for the convenience of analysis, we consider small transmission rates β and λ. Thus, the possibility of the case that a node is infected in the physical contact layer and informed in the communication layer in the same time step could be very small, and is neglected in this paper.

Let $\rho^{\mathrm{US}}_{k_1,k_2}(t)$, $\rho^{\mathrm{AS}}_{k_1,k_2}(t)$, $\rho^{\mathrm{AI}}_{k_1,k_2}(t)$ be the fractions of nodes within degree compartment (k_1, k_2) in the states US, AS, AI, respectively. They satisfy the normalization condition $\rho^{\mathrm{US}}_{k_1,k_2}(t) + \rho^{\mathrm{AS}}_{k_1,k_2}(t) + \rho^{\mathrm{AI}}_{k_1,k_2}(t) = 1$, for each set of (k_1, k_2). A heterogeneous mean-field (HMF) theory could be developed for the evolution of these fractions as follows:

$$\frac{\mathrm{d}\rho^{\mathrm{US}}_{k_1,k_2}}{\mathrm{d}t} = -\lambda k_1\theta_1^{\mathrm{A}}\rho^{\mathrm{US}}_{k_1,k_2} - \beta^{\mathrm{U}}k_2\theta_2^{\mathrm{I}}\rho^{\mathrm{US}}_{k_1,k_2} + \delta\rho^{\mathrm{AS}}_{k_1,k_2}, \tag{2a}$$

$$\frac{\mathrm{d}\rho^{\mathrm{AS}}_{k_1,k_2}}{\mathrm{d}t} = -\beta^{\mathrm{A}}k_2\theta_2^{\mathrm{I}}\rho^{\mathrm{AS}}_{k_1,k_2} - \delta\rho^{\mathrm{AS}}_{k_1,k_2} + \lambda k_1\theta_1^{\mathrm{A}}\rho^{\mathrm{US}}_{k_1,k_2} + \mu\rho^{\mathrm{AI}}_{k_1,k_2}, \tag{2b}$$

$$\frac{\mathrm{d}\rho^{\mathrm{AI}}_{k_1,k_2}}{\mathrm{d}t} = -\mu\rho^{\mathrm{AI}}_{k_1,k_2} + \beta^{\mathrm{U}}k_2\theta_2^{\mathrm{I}}\rho^{\mathrm{US}}_{k_1,k_2} + \beta^{\mathrm{A}}k_2\theta_2^{\mathrm{AI}}\rho^{\mathrm{AS}}_{k_1,k_2}, \tag{2c}$$

where $\theta_1^{\mathrm{A}} = \frac{1}{\langle k_1\rangle}\sum_{k_1,k_2} k_1 P(k_1,k_2)\left[\rho^{\mathrm{AS}}_{k_1,k_2} + \rho^{\mathrm{AI}}_{k_1,k_2}\right]$ denoting the possibility that a randomly chosen link in the communication layer will reach an A state node, and $\theta_2^{\mathrm{I}} = \frac{1}{\langle k_2\rangle}\sum_{k_1,k_2} k_2 P(k_1,k_2)\rho^{\mathrm{AI}}_{k_1,k_2}$ denoting the possibility that a randomly chosen link in the physical contact layer will reach an I state node.

With the normalization condition, there are only two independent equations in Eqs. (2). Therefore, we only need to analyze $\rho^{\mathrm{AS}}_{k_1,k_2}$, $\rho^{\mathrm{AI}}_{k_1,k_2}$ for the evolution of dynamics. It is easy to see that $\rho^{\mathrm{AS}}_{k_1,k_2} = 0$ and $\rho^{\mathrm{AI}}_{k_1,k_2} = 0$, for $\forall(k_1,k_2)$ is a trivial solution where all nodes are in US state. To obtain a non-trivial solution, i.e. $\rho^{\mathrm{AS}}_{k_1,k_2} \neq 0$, $\rho^{\mathrm{AI}}_{k_1,k_2} \neq 0$, we first represent $\rho^{\mathrm{AS}}_{k_1,k_2}$ and $\rho^{\mathrm{AI}}_{k_1,k_2}$ as functions of θ_1^{A} and θ_2^{I}, i.e. $\rho^{\mathrm{AS}}_{k_1,k_2} = \rho^{\mathrm{AS}}_{k_1,k_2}\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right)$, $\rho^{\mathrm{AI}}_{k_1,k_2} = \rho^{\mathrm{AI}}_{k_1,k_2}\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right)$. After some arrangements of Eqs. (2), we obtain a set of self-consistent equations of θ_1^{A} and θ_2^{I} as follows

$$\theta_1^{\mathrm{A}} = \frac{1}{\langle k_1\rangle}\sum_{k_1,k_2} k_1 P(k_1,k_2)\left[\rho^{\mathrm{AS}}_{k_1,k_2}\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right) + \rho^{\mathrm{AI}}_{k_1,k_2}\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right)\right] = f_1\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right),$$

$$\theta_2^{\mathrm{I}} = \frac{1}{\langle k_2\rangle}\sum_{k_1,k_2} k_2 P(k_1,k_2)\rho^{\mathrm{AI}}_{k_1,k_2}\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right) = f_2\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right).$$

Since θ_2^{I} denotes the possibility that a randomly chosen link in the physical contact layer reaches an infected node, when $\theta_2^{\mathrm{I}} = 0$, it means all nodes are literally susceptible. Thus, the prevalence of the disease requires the condition $\theta_2^{\mathrm{I}} > 0$. With the graphical method [5,31] for solving the self-consistent equation $\theta_2^{\mathrm{I}} = f_2\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right)$, the emergence of a nontrivial solution

of θ_2^{I}, i.e. $\theta_2^{\mathrm{I}} > 0$, should satisfy the following condition

$$\left[\frac{\partial f_2\left(\theta_1^{\mathrm{A}}, \theta_2^{\mathrm{I}}\right)}{\partial \theta_2^{\mathrm{I}}}\right]_{\theta_2^{\mathrm{I}}=0} \geq 1. \tag{3}$$

This condition gives the threshold of the infection rate β^{U}, denoted as $\beta_{\mathrm{c}}^{\mathrm{U}}$, where the equality is taken in Eq. (3) as follows

$$\beta_{\mathrm{c}}^{\mathrm{U}} = \frac{\langle k_2 \rangle}{\sum\limits_{k_1,k_2} k_2^{-\alpha+2} P\left(k_1, k_2\right) \frac{\lambda k_1 \theta_1^{\mathrm{A}}}{\mu\lambda k_1 \theta_1^{\mathrm{A}} + \mu\delta} + \sum\limits_{k_1,k_2} k_2^2 P\left(k_1, k_2\right) \frac{\delta}{\mu\lambda k_1 \theta_1^{\mathrm{A}} + \mu\delta}}, \tag{4}$$

which is determined by θ_1^{A}. Since the number of infected nodes is almost zero when β^{U} approaches the threshold $\beta_{\mathrm{c}}^{\mathrm{U}}$, in the communication layer the number of nodes who becomes aware due to the infection, i.e. the process of Eq. (1c), could be ignored. Therefore, the number of A state nodes in the communication layer mainly accounts for the spreading process of the disease information. Thus, when β^{U} is close to the threshold $\beta_{\mathrm{c}}^{\mathrm{U}}$, θ_1^{A} is irrelevant to the epidemic spreading process. Obviously, when $\theta_1^{\mathrm{A}} = 0$ the information is suppressed to be vanished, and the situation in the physical contact layer returns to the single layer. Since in this paper, our interest is mainly on the non-trivial impact of the communication layer on the physical contact layer, we shall focus on the case of $\theta_1^{\mathrm{A}} > 0$. With the above arguments, i.e. when the infection rate β^{U} approaches the threshold $\beta_{\mathrm{c}}^{\mathrm{U}}$, since the process of Eq. (1c) is rare, the communication layer could be treated as being independent of the physical contact layer, the highly accurate effective degree method [32] could be applied to analytically evaluate θ_1^{A}. Further, with the result of θ_1^{A}, one can obtain the value of $\beta_{\mathrm{c}}^{\mathrm{U}}$ from Eq. (4).

3. Results

In our simulation, the synchronous updating scheme is adopted. At the beginning, 20 percentage of nodes in the physical contact layer are randomly chosen as the initial infected, so their counterpart nodes in the communication layer are automatically being aware of the disease. At each time step Δt, each unaware node in the communication layer becomes aware of the disease with probability $1-(1-\lambda\Delta t)^{n_1}$, where λ is the information spreading rate and n_1 is the number of its aware neighbors. Each susceptible node in the physical contact layer becomes infected with probability $1-(1-\beta\Delta t)^{n_2}$, where β is the infection rate and n_2 is the number of its infected neighbors. At the same time, each aware node and infected node recovers with recovery rates δ and μ, respectively. The updating process terminates when the infected density ρ does not change with time. We apply the standard configuration model [33] to construct the networks for both layers. We first consider the case that the degree of two the layers are fully correlated, i.e. $P\left(k_1, k_2\right) = P\left(k_1\right)\delta_{k_1,k_2}$, where $P\left(k_1\right) \sim k_1^{-\gamma}$ is the power-law degree distribution of the communication layer with γ being the exponent. In this case, a node in the physical contact layer has the same number of neighbors as they have in the communication layer. The results of disease spreading process of this type of network is presented in Fig. 2, where the behavior of stationary fraction of infected nodes ρ is a function of infection rate β^{U} for different suppression strength α. We can see that the suppression strength α clearly has the effect in suppressing the spreading of infectious disease. On one hand, the threshold of infection rate $\beta_{\mathrm{c}}^{\mathrm{U}}$ (when $\beta^{\mathrm{U}} > \beta_{\mathrm{c}}^{\mathrm{U}}$ the network could be in endemic state) increases with the increasing of α. On the other hand, with a given infection rate β^{U}, the infected fraction ρ decreases with the increasing of α, therefore less infected nodes remain in the network for larger α. In fact, such phenomenon roots in the mechanism of the degree-dependent infection rate $\beta^{\mathrm{A}} = k_2^{-\alpha}\beta^{\mathrm{U}}$. We can see that a larger α causes a smaller infection rate for an aware node, leading to a stronger suppression of the disease transmission. As shown in Fig. 2, this result is robust to different average degrees, where simulation results and analytic results are in good accordance.

We now study how the individual suppression effect influences the global epidemic behavior. As mentioned above, since in this work we are interested in how the information spreading process influences the behavior of epidemic spreading, we consider the information spreading process goes faster than the epidemic spreading. Therefore, when the infection rate is around the threshold $\beta_{\mathrm{c}}^{\mathrm{U}}$, so that $\rho \simeq 0$, the information may have been prevalent in the communication layer. Note that the only way that the disease spreading process influences the information state of a node is through the process that a U state node will immediately become an A state node once he is infected. Therefore, when the number of infected node is very small, this influence could be ignored. Thus, in this situation the information spreading process could be treated as a single network, and the effective degree theory [32] which has high accuracy could then be applied to evaluate the value of θ_1^{A}, i.e. the possibility of reaching an A state node, and further the solution of $\beta_{\mathrm{c}}^{\mathrm{U}}$. Fig. 3 shows the gray-scale plot of ρ on the plane of α–β^{U}, where the red curve is obtained from Eq. (4). We can see that the plane is well separated into two regions: endemic region ($\rho > 0$) and non-endemic region ($\rho = 0$). The behavior of $\beta_{\mathrm{c}}^{\mathrm{U}}$ shows that when α is small, $\beta_{\mathrm{c}}^{\mathrm{U}}$ increases quickly with α, but when α is large enough, $\beta_{\mathrm{c}}^{\mathrm{U}}$ is relatively stable. This phenomenon indicates that when α begins to increase it has a prominent effect in suppressing the epidemic spreading and this effect tends to be saturated when α is large enough.

Now, we turn to the case that the degree of the two layers are uncorrelated, where the joint degree distribution can be expressed as $P\left(k_1, k_2\right) = P\left(k_1\right)P\left(k_2\right)$ with $P\left(k_1\right) \sim k_1^{-\gamma}$. Fig. 4(a) shows the relation between β^{U} and ρ for different α,

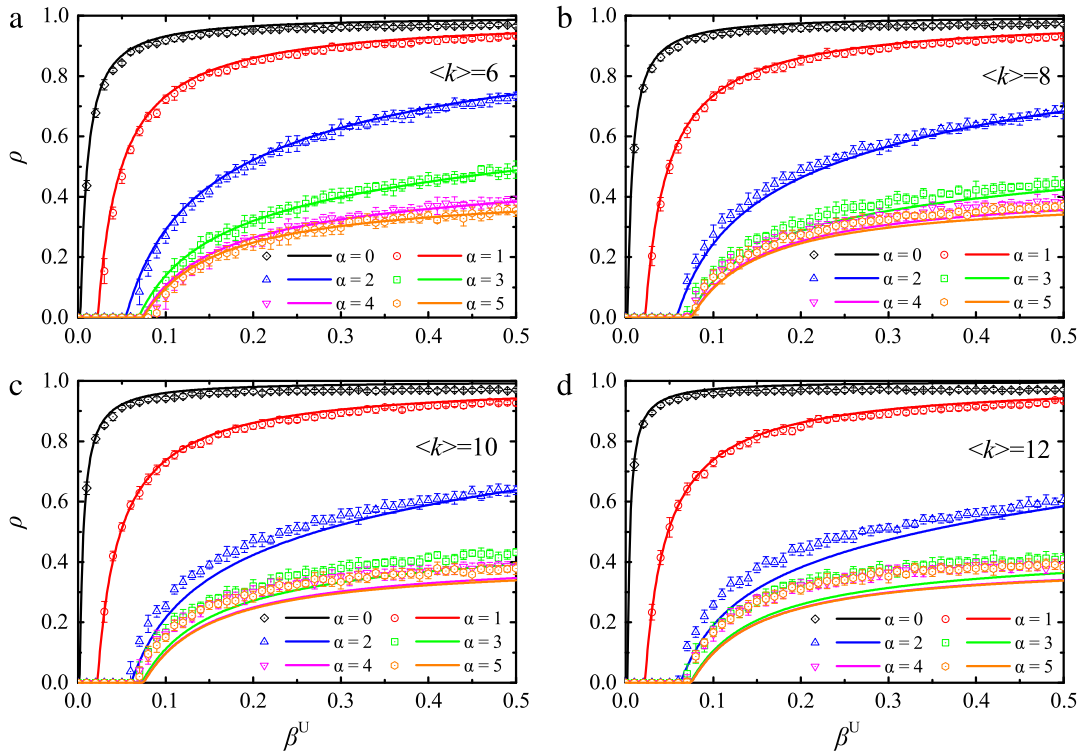

Fig. 2. Comparison of the stationary fraction of infected nodes ρ as a function of the infection rate β^U for different values of α. (a) $\langle k\rangle = 6$, (b) $\langle k\rangle = 8$, (c) $\langle k\rangle = 10$, and (d) $\langle k\rangle = 12$. From top to bottom, the value of α corresponds to $\alpha = 0, \ldots, 5$, respectively. The initial fraction of infected nodes is set to 0.2. The rate of being aware of the disease though a neighbor is $\lambda = 0.05$, the recovery rates of the two processes are $\delta = 0.02$, $\mu = 0.03$, respectively. Both layers are networks with power-law degree distribution where exponent $\gamma = 2.5$. The interlayer degree correlation are fully correlated, and therefore the two degrees in both layers are the same for each node. The size of the network is $N = 1000$. Symbols are the simulation results obtained by averaging over 50 different realizations, and curves are obtained by numerically solving Eqs. (2) under the equilibrium condition, i.e. $\mathrm{d}\rho^{US}_{k_1,k_2}/\mathrm{d}t = \mathrm{d}\rho^{AS}_{k_1,k_2}/\mathrm{d}t = \mathrm{d}\rho^{AI}_{k_1,k_2}/\mathrm{d}t = 0$.

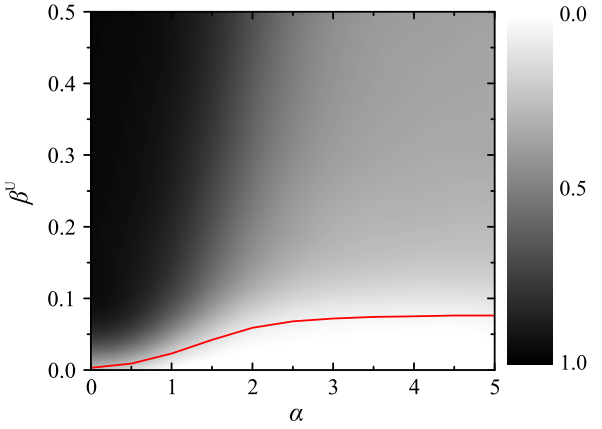

Fig. 3. Gray-scale plot of stationary infected individuals on α–β^U plane for the same network as described in Fig. 2. The red solid line denotes the theoretically predicted epidemic threshold β^U_c in Eq. (4), and the white region represents the zero epidemic prevalence. The simulation results are obtained by averaging over 50 different realizations. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

where the theoretical results are confirmed by the simulation results. Fig. 4(b) shows the gray-scale plot of ρ on the α–β^U plane, and the behavior has a similar performance with that of the full correlated joint degree distribution, i.e. larger α has a stronger suppressing effect and the suppressing effect approaches a saturated level when α is large enough. Note that in Figs. 2 and 4(a), when $\alpha = 0$, it returns back to the traditional case, where the epidemic threshold is $\beta^U_c = \langle k_2\rangle / \langle k_2^2\rangle$.

An interesting thing is to compare the behavior of the infection thresholds for the two cases that the interlayer degree correlation are fully correlated and uncorrelated. Fig. 5 shows the behavior of β^U_c for full correlated case and non-correlated

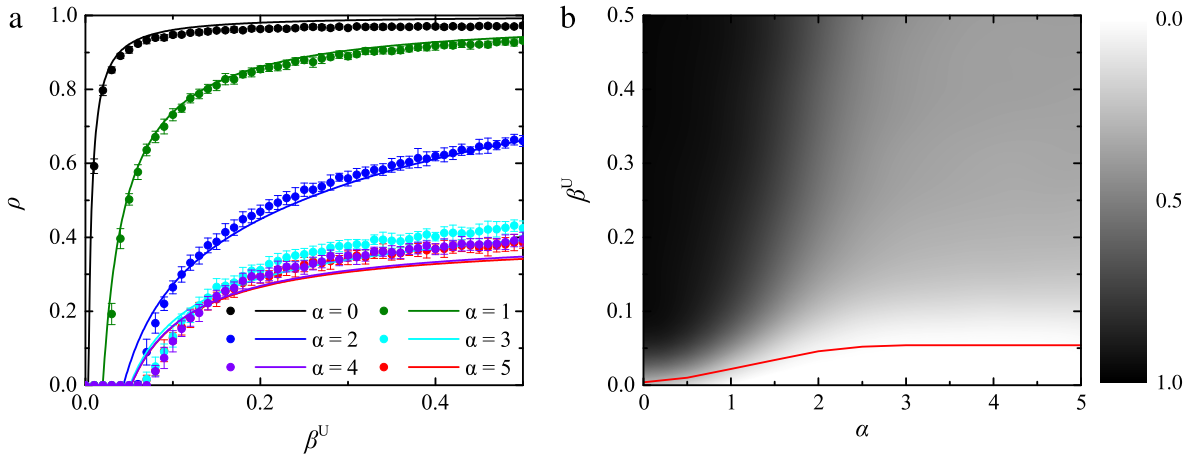

Fig. 4. Effect of suppression strength α on the epidemic threshold and prevalence on multiplex networks with no interlayer degree correlation: (a) The fraction of infected individuals ρ as a function of the infection rate β^U for different α. From top to bottom, the curves correspond to the cases of $\alpha = 0, \ldots, 5$, respectively; (b) The gray-scale plot of ρ on α–β^U plane. The red solid line denotes the theoretically predicted epidemic threshold β_c^U in Eq. (4). The two layers of the network are constructed with power-law degree distribution where exponent $\gamma = 2.5$ and average degree $\langle k \rangle = 8$. The interlayer degree correlation are uncorrelated, i.e. $P(k_1, k_2) = P(k_1) P(k_2)$. The simulation results are obtained by averaging over 50 different realizations, and curves are obtained by numerically solving Eqs. (2) under the equilibrium condition. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

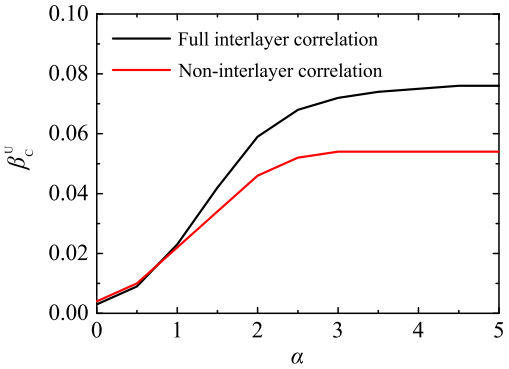

Fig. 5. Comparison of the epidemic threshold β_c^U as a function of suppression strength α of two different interlayer degree correlation. The black curve is exactly the red curve in Fig. 3 in which case the interlayer degree correlation are fully correlated, while the red solid curve is exactly the red curve in Fig. 4 in which case the interlayer degree correlation are uncorrelated. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

case. It can be seen that the case of full correlation has a stronger suppressing effect with a higher value than the case of non-correlation. This phenomenon can be understood that under full interlayer degree correlation an individual which has a larger degree in the communication layer also have a larger degree in the physical contact layer. Therefore, he will has a higher possibility of acquiring the information of the disease because of larger number of neighbors. Consequently, owing to the positive interlayer correlation he will take stricter preventive measures because of the larger number of neighbors in the physical layer, which results in a higher spreading threshold compared to the case where the interlayer degree correlation is uncorrelated.

Finally, to have a detailed observation on the effect of suppression, we show the fraction of infected nodes in their respective degree class, ρ_k, as a function of degree class k for different suppression strength α in Fig. 6. We can see that when $\alpha = 0$, ρ_k increases with k, which comes from the fact that a node with more neighbors is easier to be infected. With the increasing of suppression strength α, the tendency that ρ_k increases with k diminishes. Furthermore, when α is large enough the tendency becomes even negative, i.e. ρ_k decreases with k, reflecting the effectiveness of the suppression mechanism.

In summary, inspired by the work of Ref. [26], we study the epidemic spreading on multiplex networks where individuals may not only transmit disease through physical contacts but also spread the alerting information of epidemics through communication. Specifically, for the convenience of illustration, a multiplex network is decomposed into two layers, one is the physical contact layer and the other is the communication layer to describe the respective contact structure. When an individual acknowledges of the alerting information that a disease is spreading, he may take measures to protect himself. In this paper, which is different from previous works where individuals take preventive measures at a uniform manner, we

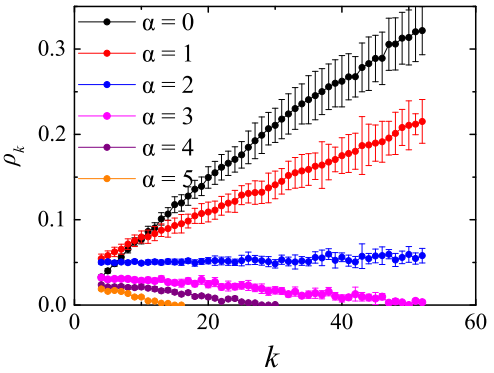

Fig. 6. Degree dependent steady state infection density ρ_k for the same multiplex networks as described in Fig. 2. From top to bottom, the suppression strength corresponds to $\alpha = 0, \ldots, 5$, respectively. The results are obtained by averaging over 50 different realizations. Other parameters are the same as those in Fig. 2.

consider that different individuals may take preventive measures to heterogeneous extent with respect to their number of neighbors in the physical contact layer. As a comparison to Ref. [26] where the scheme of homogeneous response under the awareness is adopted, our results show how the heterogeneous response influences the epidemic spreading. First, the final infected density could be strongly influenced by the suppression strength α, and the suppression effect of an infectious disease becomes more evident with larger α. Second, we studied two cases that the interlayer degree correlations are fully correlated and uncorrelated, respectively, and found that the case of full correlation has a stronger suppressing effect, reflected by a higher epidemic threshold than the other case. Finally, we found that the infection density varies with degree k under different tendency for different suppression strength α. Specifically, when α is small, the degree-dependent infection density has the tendency of increasing with degree k, while when α is large, the tendency becomes negative. As our work reflects the diverse response when people realize the prevalence of an infectious disease, we expect our study may stimulate studies regarding the problem of spreading dynamics under diverse individual responses and therefore contribute to the theoretical epidemiology.

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