

Epidemic trajectories and awareness diffusion among unequals in simplicial complexes

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ABSTRACT

The interplay between awareness diffusion and epidemic spreading has been an active topic of research in recent years. Studies have shown that group interactions are an important consideration in contagion processes, and that thus higher-order interactions should be introduced into epidemic modeling. Research has also shown that individual responses to an unfolding epidemic are often strongly heterogeneous. We therefore present a two-layer network model, where the diffusion of awareness unfolds over 2-simplicial complexes in one layer, and the actual epidemic spreading unfolds over pairwise physical contacts in the other layer. The model takes into account individual differences in the degree of acceptance of information and self-protection measures once the epidemic is perceived. We use the micro Markov chain approach to determine the epidemic threshold of the model, which agrees well with the results obtained by Monte Carlo simulations. We show that the synergistic reinforcement due to 2-simplicial complexes in the virtual layer can restrain epidemic spreading by facilitating awareness diffusion, and moreover, that individual heterogeneity in the physical layer can increase the epidemic threshold and decrease the size of epidemic transmission. However, heterogeneity in the perception can also have the opposite effect because it inhibits the diffusion of awareness. Our results reveal the intricate interplay between awareness diffusion and epidemic spreading, and we hope they can help determine effective control measures.

1. Introduction

The outbreak of infectious disease is a typical public health emergency and also becomes one of the major public safety issues that humans are facing in the 21st century [1–3], such as SARS, Ebola and COVID-19, and then it is necessary to adopt effective prevention and control strategies to avoid the global pandemic in the field of public health. Thus, the research on epidemic dynamics has attracted extensive interest from scholars [4–8]. Meanwhile, the rapid development of complex network theory has provided a powerful tool for studying the topological characteristics and dynamical properties of complex systems [9–13], where the nodes represent entities in the system and links denote the relationship between entities. Recently, many systems are often represented as multiple layers of closely related

networks [14–17]. Information related to epidemics spreads rapidly on social networks during the outbreak of infectious diseases owing to the rapid growth of information technology. Individuals will take some precautions to protect themselves when they are aware of epidemics, such as wearing masks and reducing the travel to avoid being infected, which inhibits the spread of infectious diseases to some extent [18–21]. Among them, Funk et al. [22] firstly investigated the coupled awareness and epidemic transmission model, and they found that awareness diffusion reduces the size of an infectious disease, but does not have influence on epidemic threshold. To be particularly mentioned, Granell et al. [23] analyzed the interplay between the epidemics and related information or awareness based on the framework of multiplex networks, and indicated that there exists a meta-critical point dictated by

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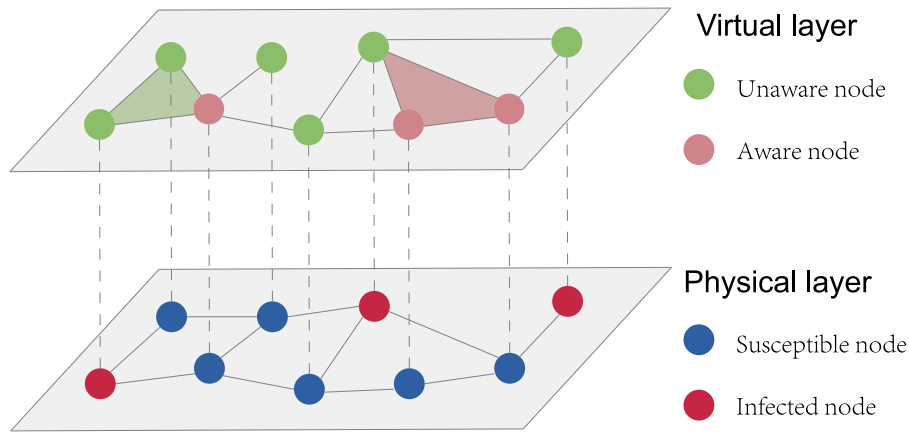


Fig. 1. The diagram of awareness-epidemic coupling spreading in two-layered networks. The top layer represents the virtual layer, which describes the diffusion of awareness. Nodes are either aware (A) or unaware (U). Shaded parts in the upper figure are the 2-simplex formed by three nodes. The green shaded part has only one aware node, which does not satisfy the propagation condition of the 2-simplex, while the red shaded part has two aware nodes so that information can diffuse by both the action of the 1-simplex and the 2-simplex. The bottom layer represents the physical layer, which describes the epidemic propagation, where nodes are either infected (I) or susceptible (S). We assume that the two-layered networks are not weighted or directed. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

the awareness and its virtual topology, and also the epidemic incidence is decreased from that point. After that, Guo et al. [24] presented a spreading threshold model that controlled by local awareness in two-layered networks, and their results demonstrated that the local awareness rate leads to an abrupt shift on epidemic outbreak threshold and affects the ultimate scale of epidemic spreading. Recently, Wang et al. [25] further investigated the effects of positive and negative disease prevention messages on outbreak threshold and the scale of infection when competing for dissemination in networks, they discovered that the epidemic outbreaks and spreading are more effectively curbed by facilitating the diffusion of positive disease prevention messages.

However, the above studies are often based on the pairwise interactions between nodes on complex networks. In fact, the effects of group interactions are prevailing in many biological [26–28], social systems [29–33] and ecology [34–36]. Higher-order network structures could better characterize these systems and encode the group interaction that contain multiple components [37–39]. As an example, Iacopini et al. [31] proposed a simplicial social infection model on higher-order networks, in which contagion can occur through pairwise and higher-order interactions, and it was found that simplicial structure induced a discontinuous transition and a bistable phenomenon appeared where susceptible and infected states coexist. Landry and Restrepo [40] analyzed the dynamics of Susceptible–Infected–Susceptible (SIS) models on heterogeneous hypergraphs by using a mean field approach, further exploring how the hyperedge structure affects the onset of epidemics as well as bistability and explosive transitions. Most of the researches on higher-order networks have been performed in single-layer network. Thus, it is necessary to consider synergistic reinforcement mechanisms of higher-order interactions in the two-layered model that simplicial awareness and epidemics coupled spreading.

Furthermore, the behaviors of individuals in many studies are generally treated homogeneously [41,42]. In other words, it is assumed that different individuals have the same acceptance to external information and the same intensity of response to infectious disease. Obviously, this assumption does not match the reality. Each independent individual has distinct behaviors and ideologies in the real world, i.e., there are differences among individuals. Therefore, individual heterogeneity has become an important factor for many researchers to consider during the epidemic modeling [43–46]. For example, Nie et al. [47] introduced inhibitory strength to study the heterogeneous response of individuals after becoming aware of the epidemic and investigated the difference of interlayer degree relativity in completely relevant and irrelevant networks, they discovered that the epidemic threshold was higher in

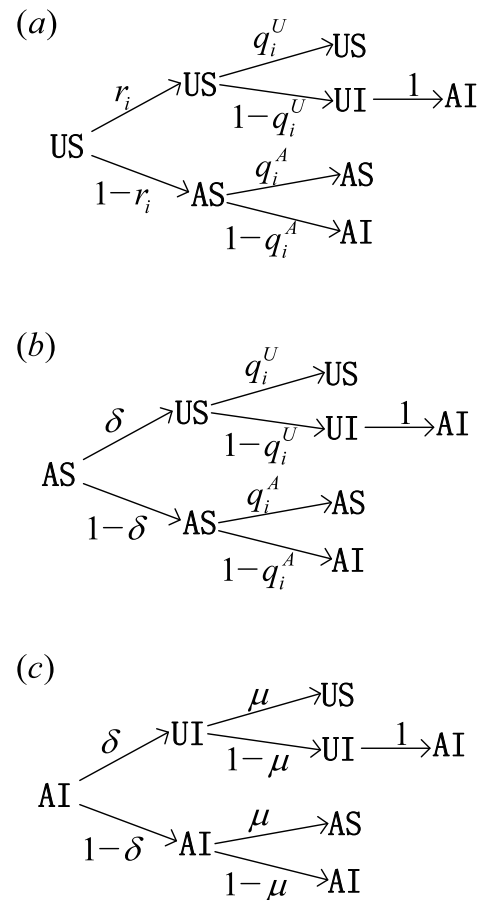


Fig. 2. Transition probability trees for 3 states (US, AS, AI). The probability r_i denotes that an individual will not acquire epidemic awareness from any neighbors. δ represents the probability that aware individuals forget the epidemic awareness. The probabilities q_i^U and q_i^A denotes that individuals in US and AS states have not been infected, respectively. μ denotes the probability that individuals who are infected reverts to the susceptible state.

the case of full correlation. Pan et al. [48] explored the influence of three different forms of individual heterogeneity on the spread of epidemics, their experimental outcomes proved that the heterogeneous

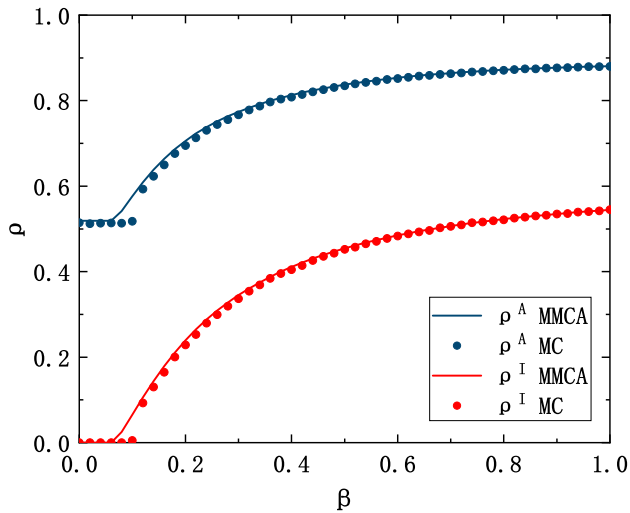


Fig. 3. Comparison of the proportion of infected individuals (ρ^I) and aware individuals (ρ^A) as a function of β utilizing MMCA and MC simulation experiments. The blue and red solid lines are ρ^A and ρ^I obtained from MMCA, respectively. The blue and red circles are ρ^A and ρ^I acquired from MC, respectively. The parameters are set as $\lambda = 0.2$, $\lambda_A = 0.4$, $\mu = 0.4$, $\delta = 0.5$, $\alpha = 2$ and $\eta = 2$. All outcomes are averaged over 100 independent runs.

control parameters of the information layer can result in an abrupt transition to outbreak threshold, but the heterogeneous parameters of the epidemic layer and the response of individuals only influence the epidemic threshold at higher stages.

Motivated further by the above works, we present a model of awareness and epidemics coupled spreading that consider the heterogeneous responses of individuals to explore the mutual interaction between awareness and epidemics in multiplex networks. We construct the virtual layer as a 2-simplicial complex network [31,49], where the information related to the epidemic is diffused. Meanwhile, the physical layer characterizes the disease spreading within the population. Then, we utilize microscopic Markov chain approach (MMCA) to analyze the model and obtain the epidemic threshold through theoretical derivation. By comparing the outcomes of theoretical analysis with experiments obtained by Monte Carlo (MC) simulation, it is observed that MMCA can well predict our model. Numerical simulation results indicate that introducing 2-simplex into the virtual layer can enhance the outbreak threshold and decrease the proportion of eventual infected individuals by promoting the diffusion of awareness. Furthermore, the individual heterogeneity also affects the epidemic transmission to some extent.

The remainder of this article is organized as follows. We first describe the two-layered network model in Section 2. Then, in Section 3, we utilize MMCA to analyze the model and deduce the theoretical expression of outbreak threshold. The effects of various parameters on awareness and epidemic processes are analyzed by extensive numerical simulations in Section 4. Lastly, we summarize this paper and propose several potential outlooks in Section 5.

2. Description of the coupling model

In our work, we construct a model of simplicial awareness and epidemic spreading in two-layered multiplex networks. Meanwhile, we investigate the heterogeneous responses of individuals when receiving information and facing epidemics based on the difference of nodes degree in the network. As Fig. 1 shows, the top layer (virtual layer) illustrates the diffusion of awareness, and the bottom layer (physical layer) denotes the epidemic transmission. In addition, the nodes in the two-layered networks present one to one correspondence but the topology of different layers is distinct.

We structure the virtual layer as a 2-simplicial complex network, and the diffusion of awareness is described as a UAU (unaware-aware-unaware) process. Each node in this layer can be in either aware (A) or unaware (U) state. At each time step, unaware nodes can obtain information related with the disease by two ways. One is to obtain information by its aware neighbors with probability λ through pairwise interactions (i.e., 1-simplex infection) and another is acquired by the remaining two nodes in the 2-simplex who are both in aware state with an additional probability λ_A through the synergistic reinforcement mechanism (i.e., 2-simplex infection). Besides, aware nodes may lose awareness related with the epidemic and revert to the unaware state again with probability δ .

Then, in the physical layer, we employ the classical SIS (susceptible-infected-susceptible) epidemic model to express the epidemic propagation. At each time step, each node can be in one of two states: infected (I) or susceptible (S). Susceptible nodes will be infected if they have infectious neighbors and convert to the infected state with a probability of β . Meanwhile, infected nodes may revert to the susceptible state with a probability of μ .

It is worth noting that there are coupling interactions between the two propagation processes in the top and bottom layer networks. On the one hand, individuals who are infected in the physical layer will immediately acquire awareness related to the epidemic; on the other hand, individuals possessing the epidemic awareness in the virtual layer will adopt certain self-protective actions to reduce their probability of being infected. In addition, previous studies [16–18] usually assumed that the nodes in the virtual layer would be aware of the epidemic and take same precautions after receiving information, but in reality, individuals do not fully recognize and accept the information obtained from the outside world due to the differences between individuals. The degree of acceptance of information and self-protection measures will also have certain differences when epidemics are prevalent. In the two-layered networks, the degree of nodes can reflect the activity intensity and social ability of individuals, the greater the degree of nodes, the higher their attention to real information. Therefore, we consider a heterogeneous model based on the difference of nodes degree. To this end, we introduce the parameter ω_i to indicate the acceptance of information by individual i and γ_i to regulate the probability that aware individual i being infected.

Firstly, the nodes in the virtual layer differ in their acceptance of the obtained information according to the node degree and the number of 2-simplex they belong to, i.e., the probability of converting the information into their own consciousness is different. Thus, we define ω_i as

$$\omega_i = \left(1 - \frac{1}{k_i + k_i^A}\right)^\alpha. \quad (1)$$

In Eq. (1), k_i and k_i^A denote the node degree of individual i and the number of 2-simplex which individual i belongs in the virtual layer, respectively. Individuals with greater degrees are more likely to accept information. And $\alpha \geq 0$ regulates the individual heterogeneity in the virtual layer. For an individual, the influence of degree becomes larger with α increases. The acceptance level of information has no effect on individuals when $\alpha = 0$.

Secondly, the nodes in the physical layer adjust the probability of individuals being infected according to different degrees. Individuals with more neighbors will take stronger self-protection measures after realizing the epidemic. Then, we define γ_i as

$$\gamma_i = (k_i')^{-\eta}, \quad (2)$$

where k_i' represents the node degree of individual i and $\eta \geq 0$ controls the individual heterogeneity in the physical layer. As η increases, the restrain effect of awareness on epidemic transmission increases. When $\eta = 0$, awareness diffusion has no impact on the spread of epidemics. Thus, β_i^A can be described as follows:

$$\beta_i^A = \gamma_i \beta_i^U = (k_i')^{-\eta} \beta, \quad (3)$$

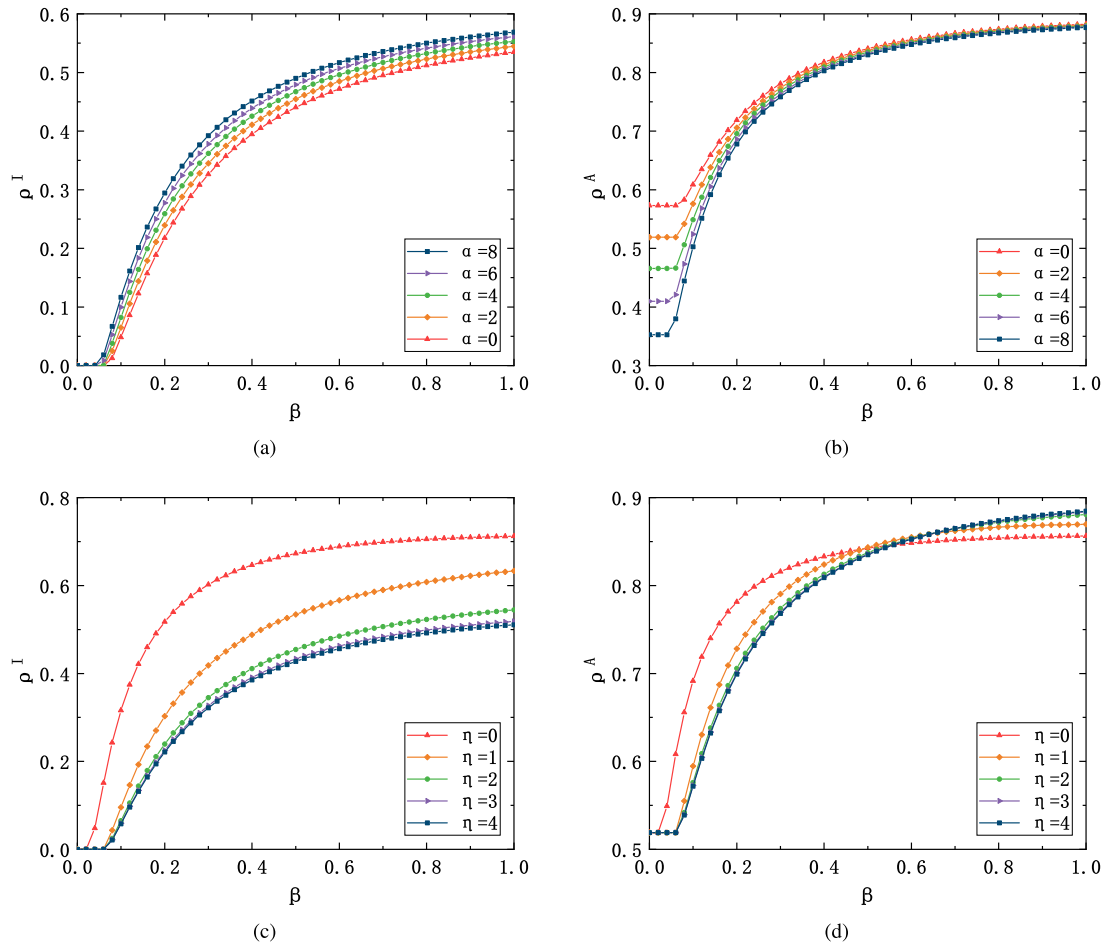


Fig. 4. Impacts of α and η on the awareness diffusion and epidemic spreading. Panels (a) and (b) depict ρ^I and ρ^A as a function of β for various values of α when $\eta = 2$, respectively. Panels (c) and (d) picture ρ^I and ρ^A as a function of β for various values of η when $\alpha = 2$, respectively. Other parameters are set as $\lambda = 0.2$, $\lambda_A = 0.4$, $\mu = 0.4$, $\delta = 0.5$.

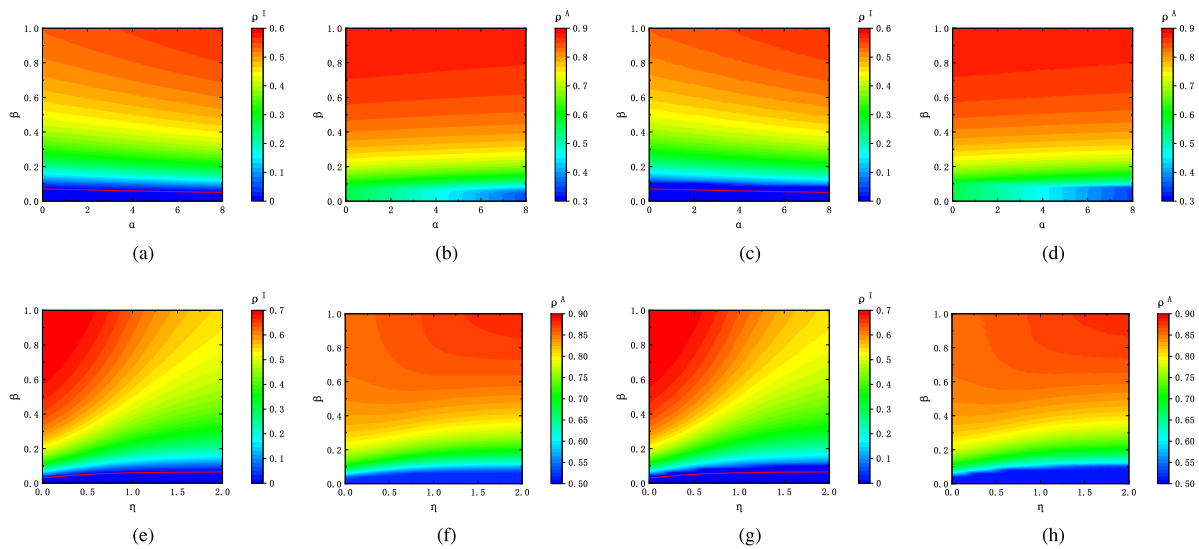


Fig. 5. The full phase diagrams (β - α) and (β - η) of ρ^I and ρ^A , respectively. Panels (a), (b), (e) and (f) are outcomes acquired by MMCA. Panels (c), (d), (g) and (h) are outcomes acquired by MC. In panels (a), (b), (c) and (d), $\eta = 2$. In panels (e), (f), (g) and (h), $\alpha = 2$. The remaining parameters are $\lambda = 0.2$, $\lambda_A = 0.4$, $\mu = 0.4$, $\delta = 0.5$. All outcomes are averaged over 100 independent runs and each point in the upper diagram lies within a grid of 50×50 .

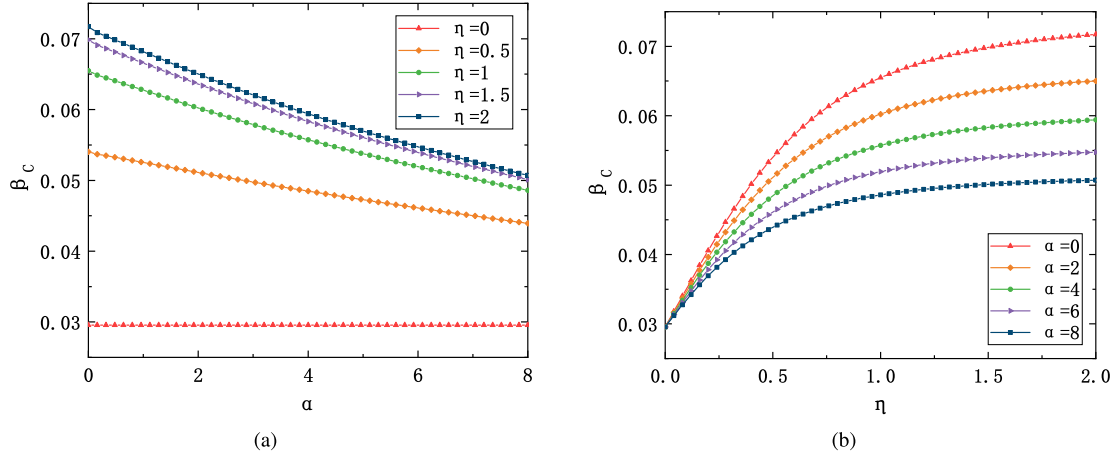


Fig. 6. Epidemic threshold (β_c) is described as a function of α and η . In panel (a), β_c as a function of α with various η . In panel (b), β_c is illustrated as a function of η with various α . Other parameters are set as $\lambda = 0.2$, $\lambda_d = 0.4$, $\mu = 0.4$, $\delta = 0.5$.

where β_i^U and β_i^A denote the probability that unaware and aware individuals may be infected by their infected neighbors, respectively.

3. Theoretical analysis based on MMCA

We employ MMCA to analyze our model theoretically and deduce an expression for outbreak threshold β_c . According to the description of our model, infected nodes will become aware automatically so that the UI state is excluded. Thus, nodes may be in the following three state: US , AS or AI . At time step t , the probabilities that individuals i being in the above three states are denoted as $p_i^{US}(t)$, $p_i^{AS}(t)$, and $p_i^{AI}(t)$, respectively. In addition, each individual must satisfy the normalization condition $p_i^{AI}(t) + p_i^{AS}(t) + p_i^{US}(t) = 1$ at single time step t .

Matrices $A = (a_{ij})_{N \times N}$ and $B = (b_{ij})_{N \times N}$ are used to denote adjacency matrices among nodes in the top and bottom networks, respectively. At time step t , we define $r_i^1(t)$ and $r_i^2(t)$ to represent the probabilities that individual i cannot acquire awareness related to the epidemic from any of 1-simplex neighbors and 2-simplex neighbors, respectively. Then, the probability $r_i(t)$ that individual i cannot acquire awareness from any aware neighbors at time step t can be calculated by multiplying $r_i^1(t)$ and $r_i^2(t)$. The probabilities $q_i^U(t)$ and $q_i^A(t)$ denote that US and AS state individuals i have not been infected by any infective neighbors, respectively. Therefore, $q_i^U(t)$, $q_i^A(t)$, $r_i^1(t)$, $r_i^2(t)$ and $r_i(t)$ are expressed as the following equations:

$$q_i^U(t) = \prod_j (1 - b_{ij} p_j^{AI}(t) \beta^U), \quad (4)$$

$$q_i^A(t) = \prod_j (1 - b_{ij} p_j^{AI}(t) \beta^A), \quad (5)$$

$$r_i^1(t) = \prod_j (1 - a_{ij} p_j^A(t) \lambda \omega_i), \quad (6)$$

$$r_i^2(t) = \prod_{j,r} (1 - c_{ijr} p_j^A(t) p_r^A(t) \lambda_d \omega_i), \quad (7)$$

$$r_i(t) = r_i^1(t) r_i^2(t), \quad (8)$$

where $p_j^A(t) = p_j^{AS}(t) + p_j^{AI}(t)$. In Eq. (7), c_{ijr} is equal to 1 if nodes i , j and r compose a 2-simplex, otherwise c_{ijr} is equal to 0. In accordance with the above definitions, the transition probability trees of nodes are in each state can be constructed as shown in Fig. 2, and the state transition

equations can be obtained by using MMCA as

$$\begin{cases} p_i^{US}(t+1) = p_i^{AS}(t) \delta q_i^U(t) + p_i^{AI}(t) \delta \mu + p_i^{US}(t) r_i(t) q_i^U(t) \\ p_i^{AS}(t+1) = p_i^{AS}(t) (1 - \delta) q_i^A(t) + p_i^{AI}(t) (1 - \delta) \mu \\ \quad + p_i^{US}(t) (1 - r_i(t)) q_i^A(t) \\ p_i^{AI}(t+1) = p_i^{AS}(t) [\delta (1 - q_i^U(t)) + (1 - \delta) (1 - q_i^A(t))] \\ \quad + p_i^{US}(t) r_i(t) (1 - q_i^U(t)) + p_i^{AI}(t) (1 - \mu) \\ \quad + p_i^{US}(t) (1 - r_i(t)) (1 - q_i^A(t)). \end{cases} \quad (9)$$

When $t \rightarrow \infty$, the awareness diffusion and epidemic spreading in the coupled model will reach steady states. Thus, we can derive the equations as follows:

$$\begin{cases} p_i^{US}(t+1) = p_i^{US}(t) = p_i^{US} \\ p_i^{AS}(t+1) = p_i^{AS}(t) = p_i^{AS} \\ p_i^{AI}(t+1) = p_i^{AI}(t) = p_i^{AI}. \end{cases} \quad (10)$$

At the steady state, the percentage of infected individuals is tend to 0 as β nears β_c , which denotes the critical point for the onset of epidemics. So, we assume that $p_i^{AI} = \epsilon_i \ll 1$. By simplifying Eqs. (4) and (5), we can obtain the following approximations:

$$\begin{cases} q_i^U \approx 1 - \beta^U \sum_j b_{ij} \epsilon_j \\ q_i^A \approx 1 - \beta^A \sum_j b_{ij} \epsilon_j. \end{cases} \quad (11)$$

Then, substituting Eq. (11) into Eq. (9) and removing the higher-order terms, we can obtain equations as follows:

$$\begin{cases} p_i^{US} = p_i^{AS} \delta + p_i^{US} r_i \\ p_i^{AS} = p_i^{AS} (1 - \delta) + p_i^{US} (1 - r_i) \\ \mu \epsilon_i = (p_i^{AS} \beta^A + p_i^{US} \beta^U) \sum_j b_{ji} \epsilon_j. \end{cases} \quad (12)$$

Because p_i^{AI} is close to 0 near β_c , we get $p_i^A = p_i^{AS} + p_i^{AI} \approx p_i^{AS}$, $p_i^{US} \approx 1 - p_i^A$. Inserting these quantifies, the third equation in Eq. (12) can be further simplified as

$$\sum_j \left\{ [1 - (1 - \gamma_i) p_i^A] b_{ji} - \frac{\mu}{\beta} \delta_{ji} \right\} \epsilon_j = 0, \quad (13)$$

where δ_{ij} is the element of the identity matrix. We set the elements of the matrix Φ to be $\phi_{ij} = [1 - (1 - \gamma_i) p_i^A] b_{ij}$ whose maximum eigenvalue is $\Lambda_{\max}(\Phi)$. Therefore, we are able to convert the solution of Eq. (13) into an eigenvalue problem for the matrix Φ and calculate the epidemic threshold as

$$\beta_c = \frac{\mu}{\Lambda_{\max}(\Phi)}. \quad (14)$$

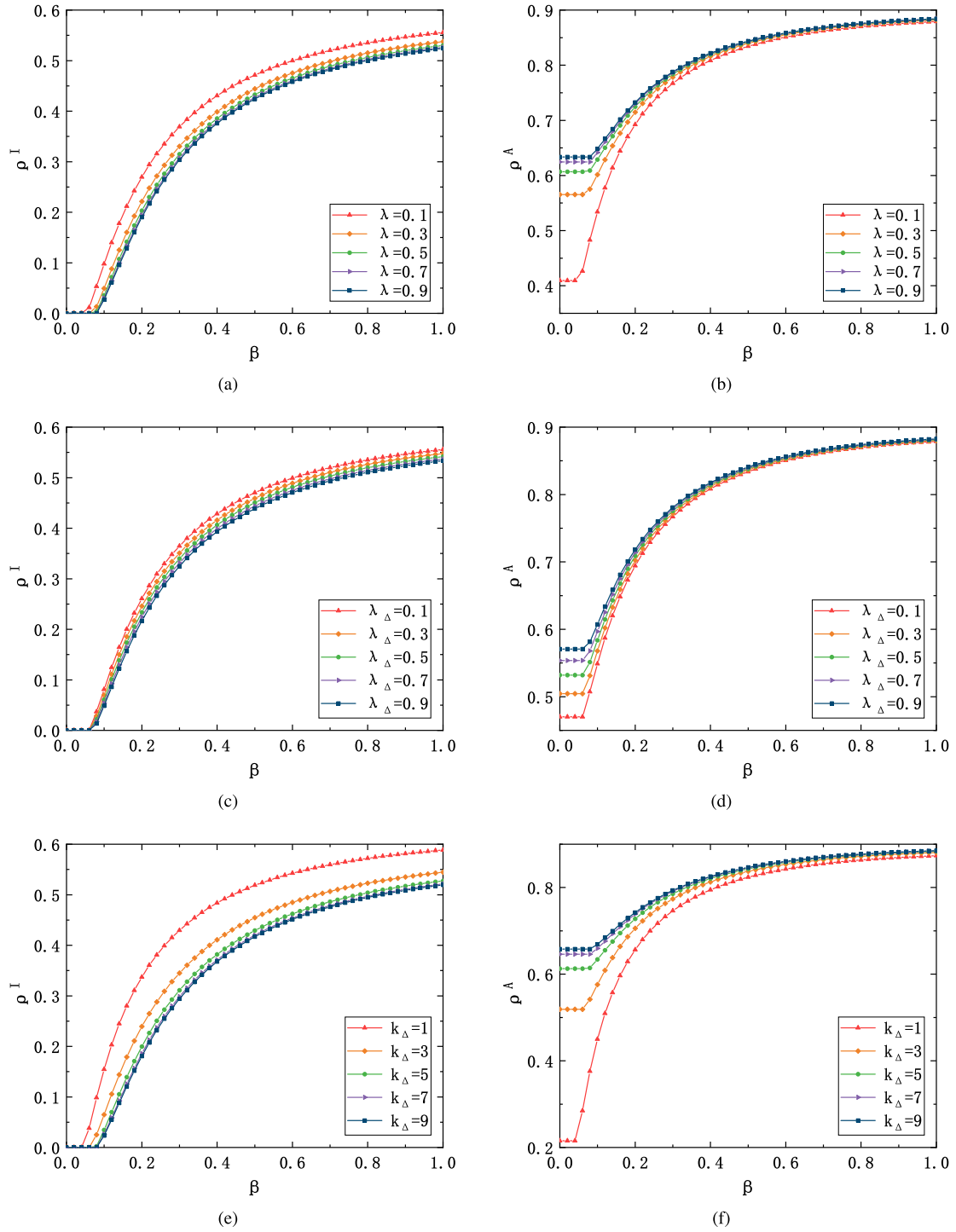


Fig. 7. Impacts of λ , λ_Δ , and k_Δ on the spread of awareness and epidemics. Panels (a) and (b) plot ρ^I and ρ^A as a function of β for various values of λ when $\lambda_\Delta = 0.4$, respectively. Panels (c) and (d) display ρ^I and ρ^A as a function of β for various value of λ_Δ when $\lambda = 0.2$, respectively. Panels (e) and (f) present ρ^I and ρ^A as a function of β for various values of k_Δ when $\lambda = 0.2$ and $\lambda_\Delta = 0.4$, respectively. Other parameters are set as $\mu = 0.4$, $\delta = 0.5$, $\alpha = 2$, $\eta = 2$.

According to Eqs. (13) and (14), it is clear that the self-protective measures of individuals (γ_i) and the diffusion of awareness (p_i^A) both affect outbreak threshold β_c . Besides, the topological structure of the physical layer and the recovery probability μ also have some influence on the threshold.

4. Numerical simulation

According to the state transition equations in Eq. (9), when the initial setting is given and the system reaches a steady state, we

can calculate the proportions of aware and infected individuals by MMCA iterative computations as $\rho^A = \sum_i \frac{(p_i^{AS} + p_i^{AI})}{N}$ and $\rho^I = \sum_i \frac{p_i^{AI}}{N}$, respectively. For the Monte Carlo (MC) numerical simulations, $\rho^A = \frac{(N_{AS} + N_{AI})}{N}$ and $\rho^I = \frac{N_{AI}}{N}$, where N_{AS} and N_{AI} are the total amount of nodes in AS and AI states, respectively. Considering that our model is based on the differences of nodes degree to study the individuals heterogeneity, and thus we structure a scale-free simplicial complex (SFSC) network in the virtual layer [31,49], and we set $N = 1000$, $m = 1$, $k_1 = 8$ and $k_\Delta = 3$. The physical layer constructed as a BA

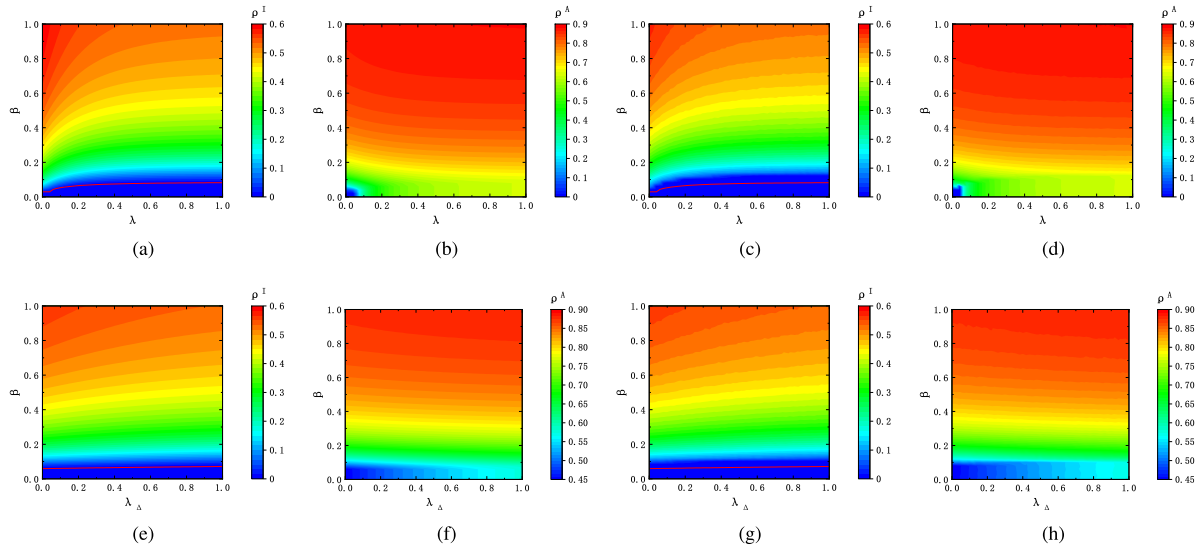


Fig. 8. The full phase diagrams ($\beta - \lambda$) and ($\beta - \lambda_d$) of ρ^I and ρ^A , respectively. Panels (a), (b), (e) and (f) are outcomes acquired by MMCA. Panels (c), (d), (g) and (h) are outcomes acquired by MC. In panels (a), (b), (c) and (d), $\lambda_d = 0.4$. In panels (e), (f), (g) and (h), $\lambda = 0.2$. Other parameters are set as $\mu = 0.4$, $\delta = 0.5$, $\alpha = 2$ and $\eta = 2$. All outcomes are averaged over 100 independent runs and each point in the upper diagram lies within a grid of 50×50 .

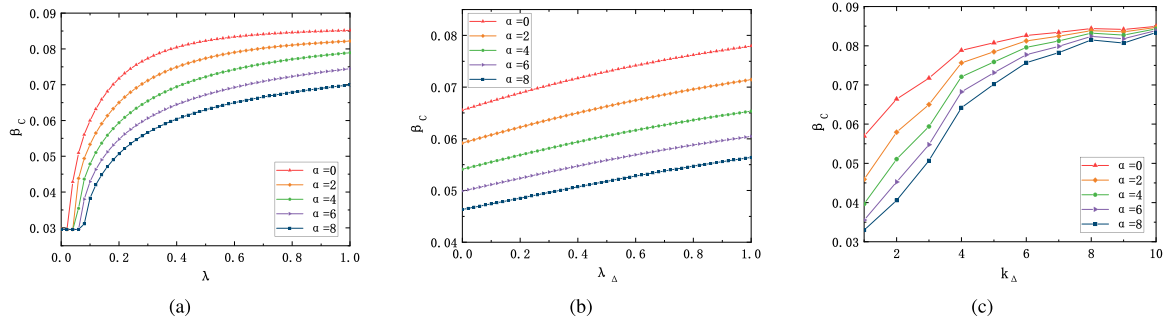


Fig. 9. Epidemic threshold (β_c) as a function of λ , λ_d , and k_d with various values of α . In panel (a), $\lambda_d = 0.4$. In panel (b), $\lambda = 0.2$. In panel (c), $\lambda = 0.2$, $\lambda_d = 0.4$. Other parameters are set as $\mu = 0.4$, $\delta = 0.5$, $\alpha = 2$, $\eta = 2$.

scale-free network, which $N = 1000$, $m = 3$. Moreover, we set the preliminary proportion of infected nodes ρ^I to be 0.1 at each simulation experiment.

First, we employ MC simulations to examine the accuracy of MMCA. Fig. 3 illustrates ρ^I and ρ^A acquired by MMCA and MC simulations as a function of β at the state steady. In order to concretely quantify the errors between MMCA and MC simulations, we express the relative errors of ρ^I and ρ^A as $(|\rho_{MC}^I - \rho_{MMCA}^I|) / \rho_{MC}^I$ and $(|\rho_{MC}^A - \rho_{MMCA}^A|) / \rho_{MC}^A$, respectively. Thus, the average relative errors of ρ^A and ρ^I in Fig. 3 can be calculated to be about 0.8% and 1.7%, respectively, which verifies that MMCA can well solve the coupled dynamics problem in this paper.

Next, we consider the impacts of heterogeneity control parameters α and η on the spread of awareness and epidemics in the coupled model. Fig. 4 shows the changes in ρ^I and ρ^A when the values of α and η are different. We observe that ρ^I increases and ρ^A decreases as α becomes larger for the same infection probability in Fig. 4(a) and 4(b). This is because as the value of α gets larger, the ability of individuals to convert information into epidemic awareness decreases, thus reducing the number of aware individuals and making it easier to spread the epidemic. In Fig. 4(c), as the value of η increases, the probability of individuals being infected decrease so that ρ^I decreases. For curves where η is greater than 0, the value of β to make the propagation tends to steady state gets larger, due to the inhibitory effect of the upper layers on epidemic transmission. In Fig. 4(d), it can be observed that ρ^A decreases first and then increases when η gets larger. The turning point at which this phenomenon occurs is around $\beta \approx 0.55$. We

consider that the percentage of infected individuals increases rapidly at the beginning of an epidemic outbreak, but due to the inhibitory effects of awareness on the epidemic transmission, the growth of ρ^I for $\eta > 0$ is lower than $\eta = 0$. Thus, there are relatively few nodes acquire epidemic awareness through the infection. When $\beta > 0.55$, the proportion of infected individuals tends to saturate at $\eta = 0$, while it still shows an increasing trend when $\eta > 0$, thus the proportion of the aware individuals continues to rise and together with the effects of aware individuals spreading disease information in the top layers, results in the condition shown in Fig. 4(d). Moreover, when the value of η increases to a certain value, the effects of η on ρ^I and ρ^A are no longer apparent.

In order to explore the influences of α and η more fully, we draw the full phase diagrams of ρ^I and ρ^A as shown in Fig. 5. The panels (a), (b), (e) and (f) in Fig. 5 illustrate the outcomes acquired by MMCA and panels (c), (d), (g) and (h) in Fig. 5 indicate the outcomes obtained by MC simulations. The results also suggest that increasing the heterogeneity control parameter α in the virtual layer is not conducive to awareness diffusion and leading to a rise in the proportion of infected individuals. However, increasing the heterogeneity control parameter η in the physical layer renders that individuals take stronger self-protection measures to prevent epidemic, thus restraining the spread of epidemic. Moreover, since η acts directly on the epidemic layer, we can observe a more pronounced effect of η on ρ^I than of α on ρ^I .

In Fig. 6, we further investigate the effects of α and η on the epidemic threshold β_c . From Fig. 6(a), it can be seen that when $\eta = 0$,

the increase of α does not affect the threshold since the diffusion of awareness has no inhibitory effect on the epidemic. When $\eta > 0$, the increase of α leads to a decrease of β_c . Panels (a) and (b) in Fig. 6 show that the threshold can be increased to some extent when η gets larger. Thus, it is feasible to restrain epidemic outbreaks by improving the individual's acceptance of information and enhancing self-protection measures for aware individuals.

Then, the effects of parameters λ , λ_A , and k_A relating to the UAU process on the coupled dynamics are discussed in Fig. 7. Panels (a), (c) and (e) describe the impact of λ , λ_A and k_A on ρ^I , meanwhile panels (b), (d) and (f) picture the impact of λ , λ_A and k_A on ρ^A . Experimental results show that these parameters have similar impacts on awareness diffusion and epidemic transmission, i.e., when λ , λ_A or k_A gets larger, the ratio of infected individuals is reduced and the diffusion of awareness is facilitated. This is mainly due to the fact that the increase in these three parameters promotes the dissemination of information among the population, and thus allowing more individuals to become aware of epidemics and take some self-protection actions can effectively suppressing the spread of epidemics. From the outcomes of λ_A and k_A , considering the simplicial complex structure plays a great role in the coupled dynamics.

The awareness diffusion and epidemic spreading within a wide range of $(\beta - \lambda)$ and $(\beta - \lambda_A)$ are illustrated in Figs. 8. It is discovered that ρ^A gradually increases with λ and λ_A get larger, and the impact on ρ^A is more obvious when β is smaller. By comparing panels (a) and (e) in Fig. 8 with panel (e) in Fig. 5, it can be observed that η has a stronger inhibition to ρ^I when β is larger. Therefore, increasing individual self-protection measures by increasing η is a more efficient method to contain the epidemic when the transmission rate of the epidemic is high.

Finally, we further analyze the impact of λ , λ_A , and k_A on the epidemic thresholds β_c in Fig. 9. It can be found that β_c increases with λ , λ_A , and k_A for different values of α , which indicates that the threshold can be raised by facilitating the information dissemination in the top layer network, and the outbreak of epidemics becomes more difficult for the smaller values of α .

5. Conclusions

In this paper, in order to investigate the interaction between the spread of awareness and epidemics when considering the heterogeneous responses of individuals, we construct a coupled spreading model based on the difference of nodes degree in two-layered networks. At the same time, in the virtual layer we consider the effect of 2-simplicial complex, i.e., awareness of epidemics can simultaneously diffuse through pairwise and group interactions. Different from previous studies, we take into account the differences between individuals for the degree of acceptance of information and self-protection measures taken in the face of epidemics. We perform a theoretical analysis of the current model by using MMCA and derive outbreak threshold. Through extensive simulation experiments, we test the accuracy of theoretical analysis. The experimental outcomes indicate that the synergistic reinforcement effect of higher-order structures could promote the spreading of awareness and make more individuals take certain self-protective actions to avoid the likelihood of being infected and hence increase the epidemic threshold. Furthermore, the heterogeneous control parameters α and η also affect the diffusion dynamics of the model. As the suppression strength η increases, the inhibitory effect on epidemic becomes more pronounced. Our findings may contribute to understanding the interrelation between the spreading of awareness and epidemics and offer great enlightenment for reality of epidemic prevention. In the future, on the one hand, considering the fact that there is the temporal properties of topology or dynamical switching of links in social interactions, we will further investigate the impact of individuals heterogeneity on epidemic propagation in time-varying networks. On the other hand, the individual decision could be influenced by many realistic factors, we will combine the evolutionary game theory with the prevention and vaccination of epidemics for diseases of vaccine available.

CRediT authorship contribution statement

Lijin Liu: Investigation, Visualization, Software, Writing. **Meiling Feng:** Visualization, Software, Formal analysis, Writing. **Chengyi Xia:** Conceptualization, Methodology, Validation, Writing – review & editing, Funding acquisition. **Dawei Zhao:** Conceptualization, Visualization, Software, Funding acquisition. **Matjaž Perc:** Conceptualization, Visualization, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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