Dynamical Processes and the Onset of the Epidemic on Nonlinear Coupled Multiplex Networks

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Abstract

Recently, the interplay between epidemic spreading and awareness diffusion has aroused the interest of many researchers, whose studies are mainly based on models with linear coupling relations between information and epidemic layers. However, in real networks the relation between two layers may be closely correlated with the property of individual nodes and exhibits nonlinear dynamical features. Here we propose a nonlinear coupled information-epidemic model (I-E model) and makes a comprehensive analysis in a more general scenario where upload rate differs from node to node, deletion rate varies between susceptible and infected states, and infection rate changes between unaware and aware states. Theoretically, we demonstrate a probabilistic description of the intra-layer and inter-layer dynamical processes by microscopic Markov chain approach (MMCA), meanwhile the expression for the onset of the epidemic is also derived by MMCA. Surprisingly, our results indicate that the change of upload and deletion rate has little effect on the diffusion dynamics in the epidemic layer.

Keywords: Nonlinear coupled networks, Microscopic Markov chain approach, Epidemic spreading

1. Introduction

Recently, increasing attention has been paid to the study of diffusion process in complex networks [1, 2, 3, 4], especially the epidemic propagation [5, 6, 7, 8]. There have been plenty of propagation models to describe these

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dynamical processes, such as the susceptible-infected-susceptible model (SIS) [9], susceptible-infected-recovery model (SIR) [10], *et al.* [11, 12]. In these models, a variety of factors can have influence on epidemic spreading, such as the contact frequency between people [13, 14], disease duration [15], particular individuals' immunity [16], and risk perception [17]. In the last few years, epidemic spreading with human response has aroused the interest of researchers. Funk *et al.* showed that awareness of epidemic can only lead to smaller scale of epidemic, but cannot affect the onset of the epidemic in a well-mixed population [18]. Wu *et al.* divided the awareness into three categories: local awareness, global awareness, and contact awareness [17].

When actors in different layers of coupled networks are the same, we will call these networks multiplex networks [19, 20, 21, 22], which has been widely investigated as an important case of independent networks [23, 24]. On top of coupled multiplex networks, researchers can further investigate the interaction between epidemic spreading and awareness diffusion, where epidemic spreading in one layer is affected by information propagation taking place in another layer. Granell *et al.* used MMCA to derive the expression of epidemic threshold and found a metacritical point for the onset of the epidemic [25]. Moreover, they also investigated the influence of a massive broadcast of awareness (mass media) on the dynamical processes, which showed that mass media could make the metacritical point disappear [26]. Guo *et al.* proposed a local awareness-controlled contagion spreading model in which the awareness layer is a threshold model by using the same framework as the UAU-SIS model [27, 28]. To summarise, most of previous studies are based on models with linear coupling relations between information and epidemic layers, which could barely reveal the strong heterogeneity among individuals and the nonlinear dynamical features in real networks.

In this paper, we propose a nonlinear coupled I-E model on top of multiplex networks to study the interplay between epidemic spreading and awareness diffusion. To improve the previous model [25, 26], a full analysis is represented in a more general scenario where upload rate differs from node to node, deletion rate varies between susceptible and infected states, and infection rate changes between unaware and aware states. We mainly focus on how the three coupling parameters γ_1, γ_2 and α affect the dynamical processes in the nonlinear coupled I-E model. Our results indicate that γ_1 can influence the threshold and scale of the disease in the epidemic layer while γ_2 and α can only affect the diffusion dynamics in the information layer. Moreover, we also investigate the effects of different multiplex network topologies

and three transmission parameters on the epidemic threshold, which shows an interesting phenomenon: in the case of $\lambda < \lambda_c$, there exists an inflection point of β_c as a function of λ.

Our paper is organized as follows: in Sec.2, we describe the nonlinear coupled I-E model and the dynamical processes on it. In Sec.3, we demonstrate a probabilistic description of the dynamical processes and derive the expression for epidemic threshold by MMCA. In Sec.4, we compare numerical simulations with theoretical results, as well as investigate the effects of different network topologies and different parameters on the diffusion dynamics. In Sec.5, we conclude the paper and present some discussions.

2. Nonlinear coupled information-epidemic model

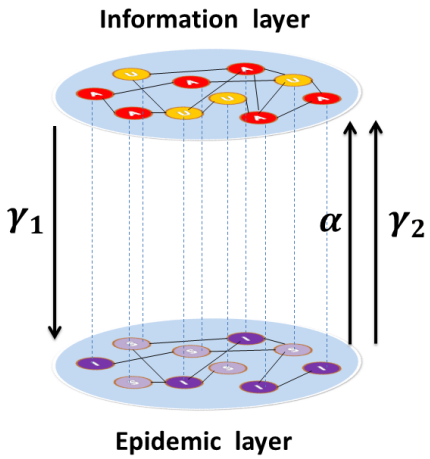

Figure 1: Illustration of nonlinear coupled I-E model. Information is propagating on upper layer, and the nodes on information layer have two possible states: unaware (U) or aware (A). Epidemic is spreading on lower layer, where the nodes also have two possible states: susceptible (S) and infected (I).

The SIS epidemic diffusion process takes place on the lower layer, where epidemic spreads with an infection rate β and a recovery rate μ; On the upper layer, where information of the epidemic propagates with a propagation rate λ and a information forgetting (or deleting) rate δ, the model for propagating is unaware-aware-unaware (UAU), similar with SIS that we all know.

Between the two layers in the model, there are three coupling relations we will analyze next in our paper.

When an individual receives epidemic information from social network and subsequently changes the state of the information layer from unaware to aware, the probability of being infected will be reduced by a factor γ_1. The connection between unaware infection rate β^U and aware infection rate β^A is:

$$\beta^A = \gamma_1 \beta^U.$$

When an individual is infected, the probability of forgetting or deleting information will be reduced by a factor γ_2. We use the following equation to connect the infected deletion rate δ_2 with the susceptible deletion rate δ_1:

$$\delta_2 = \gamma_2 \delta_1.$$

When an individual gets infected but dose not post any status about the disease on social network, he/she still have a great chance of updating his/her status from unaware state to aware state. However, the probability of information uploading differs from person to person. Individuals that have a lot of friends (nodes with large degrees) are more willing to upload information and post status on social networks. According to the above descriptions, we can approximate the following formula to represent the probability of information uploading:

$$1 - k_i^{-\alpha},$$

where k_i denotes the degree of node i, $\alpha > 0$.

We use Table 1 to describe each parameter more clearly.

3. Microscopic Markov Chain Approach

First of all, we divide the N nodes into four classes. Each node i can be in one of the following four states at time t: unaware and susceptible state (US), unaware and infected state (UI), aware and susceptible state (AS), aware and infected state (AI), whose probabilities can be represented by $p_i^{US}(t)$, $p_i^{UI}(t)$, $p_i^{AS}(t)$ and $p_i^{AI}(t)$.

Under the assumption that the probabilities of becoming aware or infected by any neighbor are independent, we use $r_i(t)$, $q_i^A(t)$, $q_i^U(t)$ to represent three basic quantities as follows:

Table 1: The descriptions of some key parameter

Parameter	Description
λ	Probability of becoming aware
δ_1	Probability of forgetting or deleting information for infected individuals
δ_2	Probability of forgetting or deleting information for susceptible individuals
β^A	Probability of getting infected for aware individuals
β^U	Probability of getting infected for unaware individuals
μ	Probability of recovery
k_i	Degree of node i
$1-k_i^{-\alpha}$	Probability of uploading information on social network for unaware and infected individuals

$$r_i(t) = \prod_j [1 - a_{ji} p_j^A(t)\lambda].$$

$r_i(t)$ denotes the probability for node i not getting the information by any neighbors.

$$q_i^A(t) = \prod_j [1 - b_{ji} p_j^I(t)\beta^A].$$

$q_i^A(t)$ denotes the probability for node i not being infected by any neighbors if i was aware.

$$q_i^U(t) = \prod_j [1 - b_{ji} p_j^I(t)\beta^U].$$

$q_i^U(t)$ denotes the probability for node i not being infected by any neighbors if i was unaware.

a_{ji} and b_{ji} in above equations represent the elements of the adjacency matrices of information layer and epidemic layer.

As shown in Figure 2, every time step is divided in three stages, so we can easily get the MMCA equations for each node i as follows:

$$p_i^{US}(t+1) = p_i^{UI}(t) r_i(t)\mu + p_i^{AI}(t)\delta_2\mu + p_i^{US}(t) r_i(t) q_i^U(t) + p_i^{AS}\delta_1 q_i^U(t), \quad (1)$$

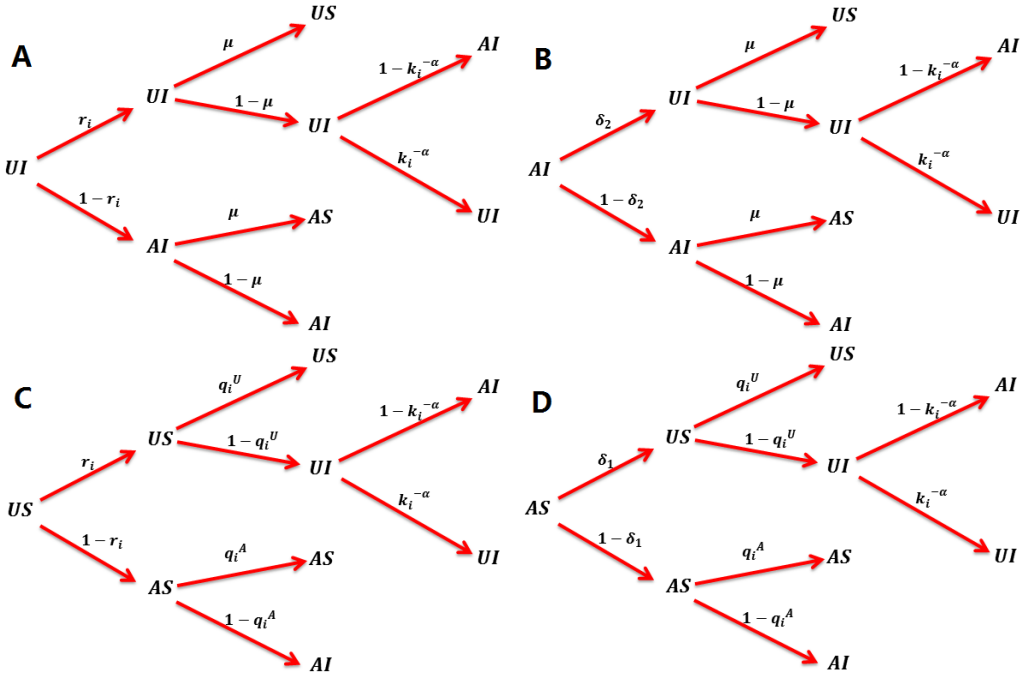

Figure 2: Transition probability trees for four possible states a node may be in: UI(A), AI(B), US(C), AS(D). The root of each tree represents the state of any node at time t, and the leaves represent their states at time $t+1$. Each time step is divided into three stages: information propagating (UAU process), epidemic spreading (SIS process), information uploading.

$$\begin{aligned} p_i^{UI}(t+1) = p_i^{UI}(t) r_i(t)(1-\mu)k_i^{-\alpha} + p_i^{AI}(t)\delta_2(1-\mu)k_i^{-\alpha} \\ + p_i^{US}(t) r_i(t)(1-q_i^U(t))k_i^{-\alpha} + p_i^{AS}\delta_1(1-q_i^U(t))k_i^{-\alpha}, \end{aligned} \tag{2}$$

$$\begin{aligned} p_i^{AS}(t+1) = p_i^{UI}(t)(1-r_i(t))\mu + p_i^{AI}(t)(1-\delta_2)\mu \\ + p_i^{US}(t)(1-r_i(t))q_i^A(t) + p_i^{AS}(t)(1-\delta_1)q_i^A(t), \end{aligned} \tag{3}$$

$$\begin{aligned} p_i^{AI}(t+1) = p_i^{UI}(t)[r_i(t)(1-\mu)(1-k_i^{-\alpha}) + (1-r_i(t))(1-\mu)] \\ + p_i^{AI}(t)[\delta_2(1-\mu)(1-k_i^{-\alpha}) + (1-\delta 2)(1-\mu)] \\ + p_i^{US}(t)[r_i(t)(1-q_i^U(t))(1-k_i^{-\alpha}) + (1-r_i(t))(1-q_i^A(t))] \\ + p_i^{AS}(t)[\delta_1(1-q_i^U(t))(1-k_i^{-\alpha}) + (1-\delta_1)(1-q_i^A(t))]. \end{aligned} \tag{4}$$

Now, let's analyze the epidemic threshold by microscopic Markov chain approach. Add Eqs.(2)and(4) to get the equation:

$$\begin{aligned} p_i^I(t+1) = p_i^I(t)(1-\mu) + p_i^{US}(t)[r_i(t)(1-q_i^U(t)) + (1-r_i(t))(1-q_i^A(t))] \\ + p_i^{AS}(t)[\delta_1(1-q_i^U(t)) + (1-\delta_1)(1-q_i^A(t))]. \end{aligned}$$

To compute the epidemic threshold, we derive the equation from above in the stationary state of the system:

$$\mu p_i^I = p_i^{US}[r_i(1-q_i^U) + (1-r_i)(1-q_i^A)] + p_i^{AS}[\delta_1(1-q_i^U) + (1-\delta_1)(1-q_i^A)]. \tag{5}$$

The probability of node i to be infected, near the epidemic threshold, is approximately equal to zero, i.e. $p_i^I = \epsilon_i \ll 1$. We can obtain the approximations of q_i^A and q_i^U as follows:

$$q_i^A \approx 1 - \beta^A \sum_j b_{ji}\epsilon_j,$$

$$q_i^U \approx 1 - \beta^U \sum_j b_{ji}\epsilon_j.$$

Taking the approximate equations above into Eqs.(1)and(3) and omitting the $O(\epsilon_i)$ terms, we get

$$p_i^{US} = p_i^{US} r_i + p_i^{AS}\delta_1, \tag{6}$$

$$p_i^{AS} = p_i^{US}(1 - r_i) + p_i^{AS}(1 - \delta_1). \tag{7}$$

Then substituting Eqs.(6) and (7) into Eq.(5) leads to

$$\mu\epsilon_i = (p_i^{AS}\beta^A + p_i^{US}\beta^U)\sum_j b_{ji}\epsilon_j.$$

Near the onset of the epidemic, the probability of an individual to be infected is close to zero, so $p_i^{AS} \approx p_i^A$, $p_i^{AS} + p_i^{US} \approx p_i^A + p_i^U = 1$. Inserting them into above equation, we obtain

$$\sum_j [[1 - (1 - \gamma_1)p_i^A]b_{ji} - \frac{\mu}{\beta^U}\delta_{ji}]\epsilon_j = 0. \tag{8}$$

Where δ_{ji} denote the elements of the identity matrix. Note that we can define a new matrix H, where h_{ij} satisfy: $h_{ij} = [1 - (1 - \gamma_1)p_i^A]b_{ji}$ to simplify Eq.(8). The epidemic threshold is equal to the minimum value of β^U satisfying Eq.(8). We can obtain the onset of the epidemic by denoting $\Lambda_{max}(H)$ the maximum eigenvalue of H,

$$\beta_c^U = \frac{\mu}{\Lambda_{max}(H)}. \tag{9}$$

Note that the value of $\Lambda_{max}(H)$ depends on p_i^A, we can substitute $p_i^{AS} \approx p_i^A$ and $p_i^{US} \approx 1 - p_i^A$ into Eq.(7) and obtain

$$(1 - p_i^A)(1 - r_i) - p_i^A\delta_1 = 0, \tag{10}$$

where

$$r_i = \prod_j [1 - a_{ji}p_j^A\lambda]. \tag{11}$$

Then it is effortless to get p_i^A, the stable solution by solving Eqs.(10)(11) with iteration method. Note that, near the onset of the epidemic, the probability of being infected and the number of infected individuals is almost zero. Eq.(10) can be easily understood as the dynamic contagion is mostly just performing in the information layer.

4. Results

In order to exam the validity of MMCA, we propose the I-E model on computer and perform extensive Monte Carlo (MC) simulations. It is not difficult for us to obtain p_i^A, the stable solution by solving Eqs.(1)(2)(3)(4) with iteration method. Then we use $\rho^A = \frac{1}{N}\sum_i p_i^A$ and $\rho^I = \frac{1}{N}\sum_i p_i^I$, which represent the ρ^A and ρ^I in MMCA, to compare with the ρ^A and ρ^I in MC simulations (run 100 times and average the results).

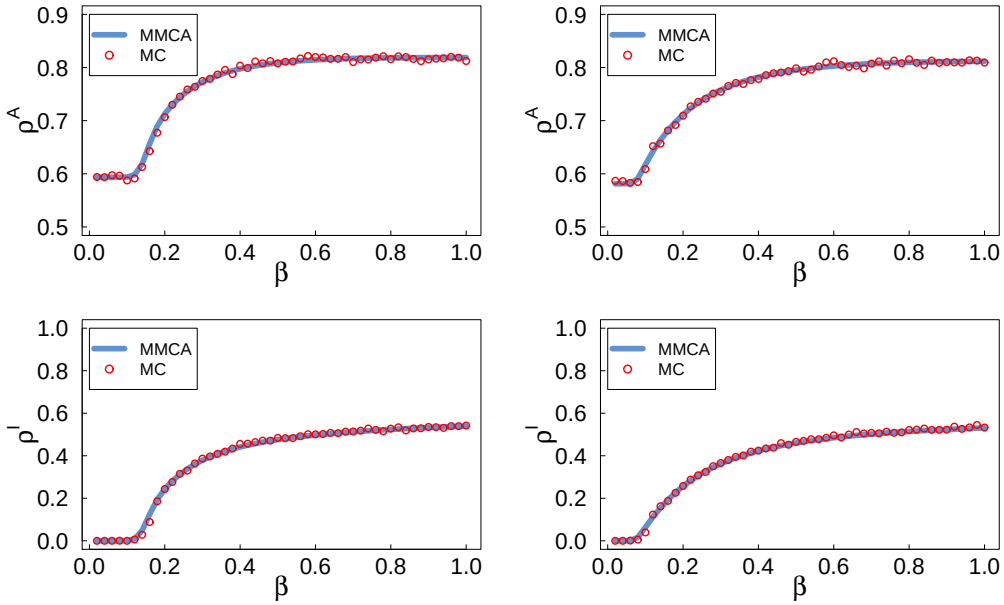

Figure 3: Results on Erdős-Rényi (ER) networks and scale-free (SF) networks obtained by using MC simulations (red circles) and MMCA (blue line). Top: comparisons between the ρ^A using MMCA and MC simulations as functions of the parameter β. Bottom: comparisons between the ρ^I using MMCA and MC simulations as functions of the parameter β. Left: Erdős-Rényi network. Right: scale-free network, which has the same number of nodes and edges with ER network. (i)Epidemic layer, a network generated by 1000 nodes and 5000 edges. (ii)Information layer, the same network than in the epidemic layer but with 1000 extra random edges. The values of fixed parameters are: $\lambda = 0.7$, $\mu = 0.3$, $\delta_1 = 0.6$, $\gamma_1 = 0.2$, $\gamma_2 = 0.5$, $\alpha = 0.5$.

We can see from Figure 3 that the theoretical calculations (blue line) are in good agreement with the extensive MC simulations (red circles). Therefore, MMCA can play a significant role in the theoretical analysis of the dynamical processes on the nonlinear coupled network.

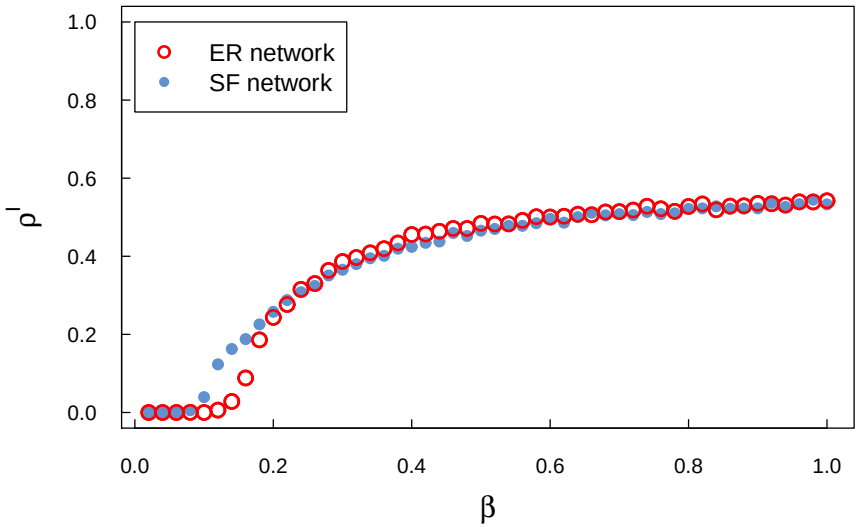

Figure 4: Comparison between ρ^I as a function of the parameter β in ER network and in SF network. The topologies of the networks and the parameters of processes are the same as above.

In Figure 4 we plot the density of infected individuals as a function of β on networks with different topologies. Though the number of nodes and edges of two networks are the same, the onset of the epidemic on SF network is smaller than it on ER network. One possible explanation for this is that the degree distribution of the SF network is not uniform, such that if nodes with large degrees are infected, the epidemic spreading will accelerate substantively and the scale of epidemic exploration will increase. Therefore, it is evident that smaller infection rate β can lead to epidemic out-breaks on SF networks, which means epidemic threshold of SF network is smaller.

See Figure 5, parameter γ_2, the factor of a coupling relation, can't affect the onset of epidemic, and it also has minimal influence on epidemic layer even though the epidemic has broken out. But in the information layer, with the decrease of γ_2, there is less probability of forgetting or deleting information for infected individuals. It only led to a significant increase in the rate of awareness of disease information, no significant effect on scale of epidemic.

We can find through Figure 6: the parameter α, similar with γ_2, can't affect the onset of the epidemic, and it also has little influence, which can be

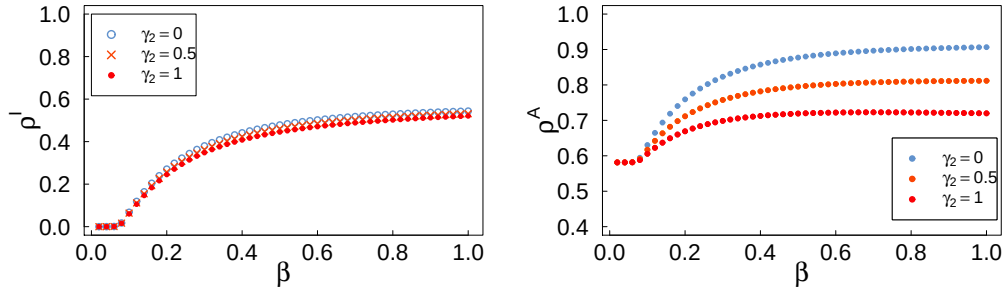

Figure 5: ρ^I and ρ^A as functions of parameter β, for different values of γ_2. The values of other fixed parameters are: $\lambda = 0.3$, $\mu = 0.7$,$\delta_1 = 0.6$, $\gamma_1 = 0.2$, $\alpha = 0.5$.

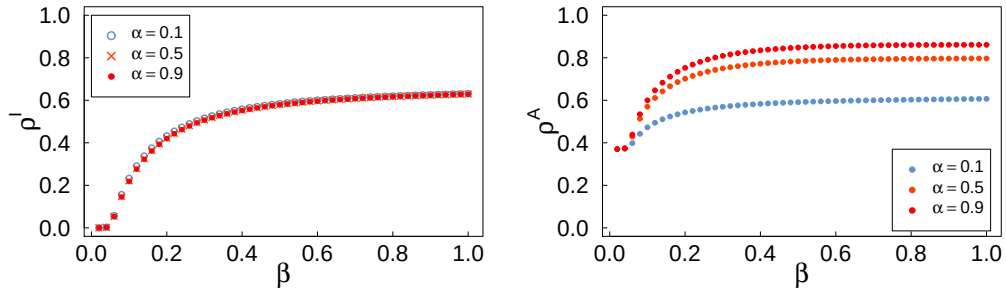

Figure 6: ρ^I and ρ^A as functions of parameter β, for different values of α. The values of other fixed parameters are: $\lambda = 0.1$, $\mu = 0.5$, $\delta_1 = 0.6$, $\gamma_1 = 0.2$, $\gamma_2 = 0.5$.

neglected, on epidemic layer even though the epidemic has broken out. But in the information layer, with the decrease of α, there is less probability of forgetting or deleting information for infected individuals. It only leads to a significant increase in the rate of awareness about disease information, little effect on scale of epidemic. Because the upload rate $1 - k_i^{-\alpha}$ is related to the degree of node i, not a fixed value, α is the parameter of the nonlinear coupling relation.

It is not difficult to understand the minimal influence on the epidemic threshold by parameters γ_2 and α: In theoretical analysis, Eq.(9), the expression for β_c^U, does not contain parameters α and γ_2; In the macroscopic view, the two coupling relations only act on the nodes of infected state. But near the epidemic threshold, the density of infected individuals is far less than 1 i.e. $\rho^I \ll 1$, therefore the influence of the two parameters γ_2 and α on the epidemic threshold can be neglected.

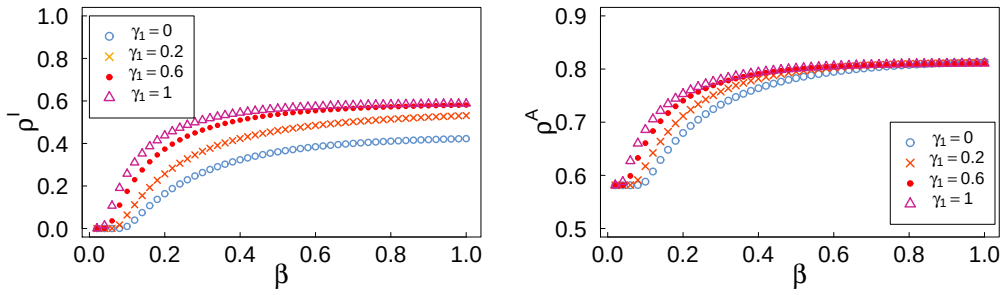

Figure 7: ρ^I and ρ^A as functions of parameter β, for different values of γ_1. The values of other fixed parameters are: $\lambda = 0.3$, $\mu = 0.7$, $\delta_1 = 0.6$, $\gamma_2 = 0.5$, $\gamma_2 = 0.5$.

With the decrease of γ_1, $\beta^A = \gamma_1 \beta^U$ will also be reduced. When $\gamma_1 = 0$, individuals with aware states are completely immune to the epidemic. From Figure 7 it can be seen clearly, with the disease prevention of aware people increased, the onset of the epidemic will significantly enlarge, and the scale of the epidemic will be reduced. In the information layer, the increase of γ_1 can also have a certain inhibitory effect on the scale of information propagation when the value of β is small.

We can get from Figure 8: with the increase of λ, the onset of the epidemic increases rapidly at the beginning, then tends to be gentle and finally becomes constant. Therefore, in the case of small λ value, it is very effective to control the disease by improving the propagation rate of information λ. We can also

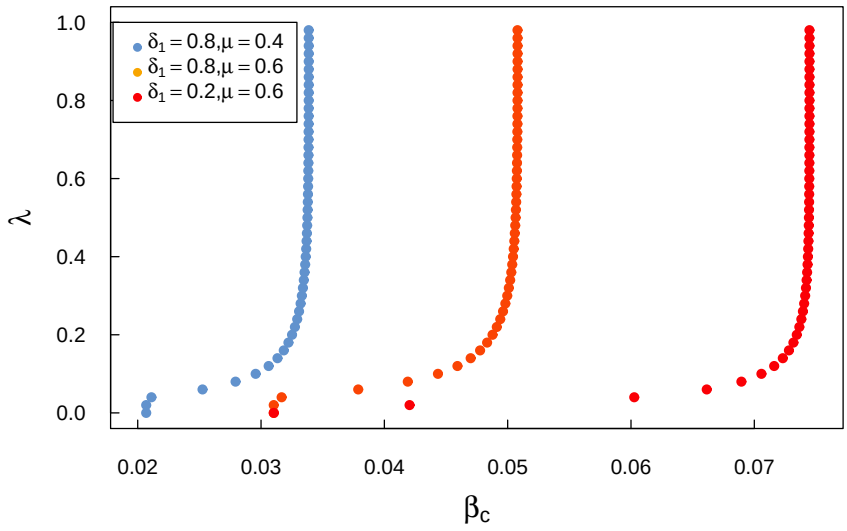

Figure 8: Value of the onset of the epidemic β_c as a function of the parameter λ. The values of other fixed parameters are: $\gamma_1 = 0.3$, $\gamma_2 = 1$, $\alpha = 10$.

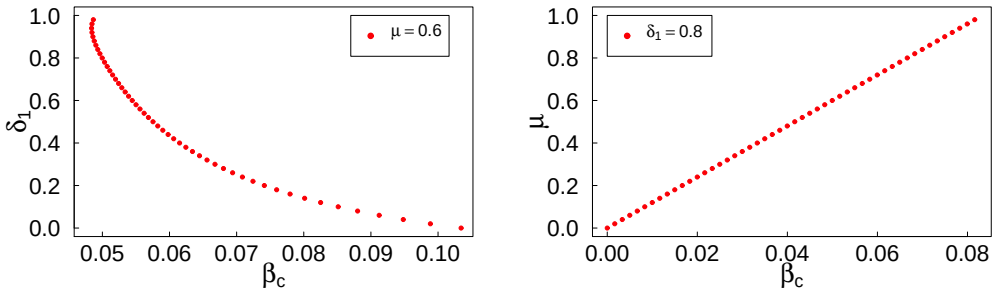

Figure 9: Left: value of the the onset of the epidemic β_c as a function of the parameter δ_1. Right: value of the the onset of the epidemic β_c as a function of the parameter μ. The values of other fixed parameters are: $\gamma_1 = 0.3$, $\gamma_1 = 0.3$, $\alpha = 10$.

find an interesting phenomenon: for the blue and orange dotted lines, β_c does not change with the increase of λ in the initial stage. The explanation for this is: the information propagation can not affect the process of epidemic spreading in lower layer when $\lambda < \lambda_c$(λ_c is the threshold of information propagating in upper layer), but can begin to affect the epidemic layer in the case of $\lambda > \lambda_c$. At the same time, the larger μ and the smaller δ_1 will lead to the increase of epidemic threshold, which can be seen more clearly in Figure 9.

5. Conclusions

In this paper, we investigate the dynamical processes and the epidemic threshold on nonlinear coupled multiplex network. We first propose a nonlinear coupled I-E model, which is a good abstraction for the scenario: the epidemic spreads in the physical contact network from person to person while the epidemic information disseminates in online social network, which can interact with the disease transmission.

Then we perform a theoretical analysis to demonstrate a probabilistic description of intra-layer and inter-layer dynamical processes by MMCA. The expression for the onset of the epidemic is also derived by MMCA and all the theoretical calculations are in good agreement with the extensive MC simulations. Therefore, MMCA can play a significant role in the theoretical analysis of the dynamical processes on the coupled network. By analyzing the expression of β_c, we find that the epidemic threshold is determined by the topology of the coupled network and p_i^A. Comparing β_cs on ER and SF networks, we find that the inhomogeneity of the degree distribution can reduce the the epidemic threshold, which suggests that we should limit contacts with the sociable persons to prevent diseases.

We also study the effects of the three parameters γ_1, γ_2, α corresponding to the three coupling relations on the dynamical processes. Intuitively, the parameter γ_1 represents the extent of disease prevention for aware individuals while the parameters γ_2 and α denote the infected individuals' degree of attention paid to the epidemic information. On the basis of extensive simulation, we discover that the extent of disease prevention for aware individuals can influence the threshold and scale of the disease in the epidemic layer while the infected individuals' degree of attention can only affect the dynamic diffusion in the information layer. This may be due to γ_1 is based on the state of upper layer to affect the epidemic spreading, but γ_2 and α are based

on the state of lower layer to influence the information propagating, which coincides with the lack of γ_2 and α in the expression of β_c. This phenomenon implies that to control the epidemic spreading needs to enhance protection and immunity of people after they have known the epidemic information.

Finally, we study the effects of the parameters λ, δ, μ on the onset of the epidemic, and find the inflection point of β_c as a function of λ in the case of $\lambda < \lambda_c$. To prevent the epidemic, we should strengthen the information communication, reduce the information forgetting, and speed up the cure of epidemic.

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