



# Competing spreading processes and immunization in multiplex networks



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## ABSTRACT

Epidemic spreading on physical contact network will naturally introduce the human awareness information diffusion on virtual contact network, and the awareness diffusion will in turn depress the epidemic spreading, thus forming the competing spreading processes of epidemic and awareness in a multiplex networks. In this paper, we study the competing dynamics of epidemic and awareness, both of which follow the *SIR* process, in a two-layer networks based on microscopic Markov chain approach and numerical simulations. We find that strong capacities of awareness diffusion and self-protection of individuals could lead to a much higher epidemic threshold and a smaller outbreak size. However, the self-awareness of individuals has no obvious effect on the epidemic threshold and outbreak size. In addition, the immunization of the physical contact network under the interplay between of epidemic and awareness spreading is also investigated. The targeted immunization is found performs much better than random immunization, and the awareness diffusion could reduce the immunization threshold for both type of random and targeted immunization significantly.

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## 1. Introduction

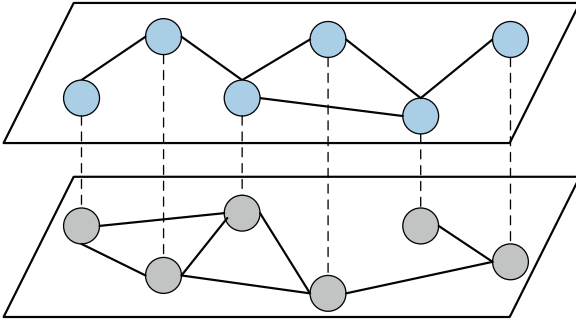
In the past years, complex network approach has proven to be a successful tool in describing a large variety of real-world complex systems, ranging from biological, technological, social to information, engineering, and physical systems [1–9]. However, most of previous works are mainly concentrated to the case of single network which treats all the network's links on an equivalent footing [1–3]. Such network modeling methods may occasionally result in not fully capturing the details present in some real-life problems, leading even to incorrect descriptions of some phenomena that are taking place on real-world systems. Recently, with the development of human cognition and “big data”, the focus on complex networks has been extended from single network to multiplex network which is composed of several network layers constructed by same nodes but with different topologies and dynamics [10–17]. Multiplex network explicitly captures the authentic and natural characteristics of real world systems: the same node may have different kinds of interactions and each channel of connectiv-

ity is represented by a layer. Thus far, the topological and dynamical characteristics of multiplex networks and various of dynamical process (such as epidemic spreading [12,18–23], evolutionary game [11,24–28] and synchronization [29–32]) upon them have attracted great attention in both theoretical and empirical areas, and a lot of remarkable results have been achieved.

As one of the hottest research topics of complex network science, epidemic spreading dynamic has centered on the modeling of different type of spreading processes and their control strategies [1,2,33–41]. The most successful epidemiology models include susceptible-infected (*SI*) model, susceptible-infected-susceptible (*SIS*) model, and susceptible-infected-recovered (*SIR*) model, both of which are good proxies for many real spreading processes involving disease in human contact networks, information and rumor in social networks, and virus in computer or communication networks, etc [1,2,33–35]. Correspondingly, many mitigation and prevention strategies of epidemics are also proposed, one of the most popular and effective methods is network immunization, such as random immunization, targeted immunization and acquaintance immunization, etc. [36–40], where certain nodes in a network acquire immunity, and are thus no longer able to transmit the disease to their neighbors.

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**Fig. 1.** A multiplex networks composed of two network layers interrelated with each other, nodes are the same in both layers and the connectivity inter-layer is from each node to itself.

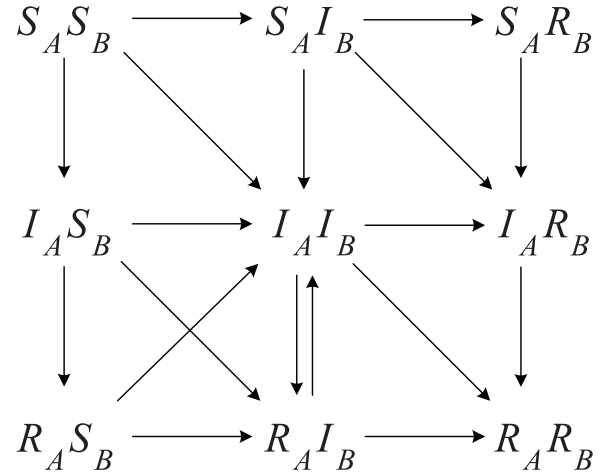
With the advent of multiplex networks, the traditional epidemic models and control methods were extended to incorporate the structure of multiplex networks. The most interesting topics are the multiple routes spreading processes [12,18,21–23], and their immunization [23,42–44]. In addition, another rapidly evolving research, the competing spreading on multiplex networks, has recently attracted considerable attentions [45–54]. The most representative example is that disease spreading on physical contact network will naturally introduce the human awareness information diffusion on virtual contact network, and the awareness diffusion will in turn depress the epidemic spreading, thus forming the competing spreading processes of epidemic and awareness in a two-layers networks. Granell et al. study the dynamical interplay between epidemic and awareness, both of which follow the SIS models, in multiplex networks. They found the critical onsets of both dynamics get intertwined and the onset of the epidemic starts depending on the incidence of aware individuals [45,46]. Wang et al. also investigate these two type of spreading dynamics where the disease obeys the *SIRV* model and the awareness the *SIR* model, and find epidemic outbreak on the contact layer can induce an outbreak on the communication layer, and information spreading can effectively raise the epidemic threshold [48].

However, up to present, no one has yet studied the competitive spreading where both epidemic and awareness follow the *SIR* process. Thus it follows that no effective methods are proposed to calculate the epidemic threshold and the outbreak size of such epidemiological model. In addition, no works have paid attention to the immunization of multiplex networks under the competitive spreading of epidemic and awareness. How to incorporate the immunization into the competing dynamics and build up effective mathematical analysis framework are important challenges currently facing.

To fill this gap, in this paper, we study the competing dynamics of epidemic and awareness, both of which follow the *SIR* process and the self-protection and the self-awareness of individual are also incorporated, in a two-layer networks based on microscopic Markov chain approach and numerical simulations. We will investigate the impacts of awareness diffusion and the capacities of self-protection and self-awareness of individuals on the epidemic threshold and the final outbreak size of the epidemic. Furthermore, the efficiency of random and targeted immunizations of multiplex network under the interplay between of epidemic and awareness spreading will be studied.

## 2. Models and analysis

The proposed model consists of a multiplex networks coupled by two network layers and two spreading processes proliferated by each layer. As shown in Fig. 1, the up layer and below layer indicate the virtual contact network and physical contact network



**Fig. 2.** Transitions between states of nodes, the arrow out from a given state of node at time step  $t$  points to its possible successor state at time step  $t + 1$ .

respectively, denoted by  $A$  and  $B$ . Both of them have the same  $N$  nodes with different intra-layer topologies.  $(a_{ij})_{N \times N}$  and  $(b_{ij})_{N \times N}$  are defined as the adjacency matrices of  $A$  and  $B$  respectively, where  $a_{ij} = 1$  indicates there is a link from node  $i$  to node  $j$  in layer  $A$ , otherwise  $a_{ij} = 0$ , and a similar definition applies to  $b_{ij}$ .

For the spreading processes of awareness and epidemic, we assume both of them follow the *SIR* epidemiology models. In the *SIR* model, each node can be in one of the three states: susceptible state ( $S$ ) in which the individual is free of the epidemic but can be infected via contacts with infected individuals; infected state ( $I$ ), where the individual carries the disease and can transmit it to susceptible individuals; and recovered state ( $R$ ), in which the individuals recovered from the disease and cannot pass the disease to other nodes or be infected again. The classic *SIR* model uses discrete time step for its evolution and at each time step, the infected node can infect its susceptible neighbors with transmissibility  $\beta$ , and then becomes recovered or removed node with probability  $\delta$ . Here, we denote  $\beta_A$  ( $\beta_B$ ) and  $\delta_A$  ( $\delta_B$ ) as the transmissibility and recover rate of the nodes in layer  $A$  ( $B$ ). Moreover, we assume the  $R$  state nodes in layer  $A$  still have the knowledge of risk information, but just have no willing to pass the information.

In our model, the awareness diffusion in layer  $A$  and the epidemic spreading in layer  $B$  are not two irrelevant processes, they are dynamic interplay and influence with each other: a node that is aware ( $I$  state) in layer  $A$  will take measures for preventing infection which is called the self-protection of the individual, this behavior can be reflected by the reduction of individual's own infectivity with a factor  $\gamma$  ( $0 \leq \gamma \leq 1$ ) in layer  $B$ ; a node that is infected in layer  $B$  will become aware in layer  $A$  with probability  $\kappa$  ( $0 \leq \kappa \leq 1$ ), which indicates the self-awareness ability of individual due to the infection of the epidemic.

Summing up, in our proposed model, every node of the multiplex network falls into the following nine states:  $S_A S_B$ ,  $S_A I_B$ ,  $S_A R_B$ ,  $I_A S_B$ ,  $I_A I_B$ ,  $I_A R_B$ ,  $R_A S_B$ ,  $R_A I_B$  and  $R_A R_B$ , where  $X_A Y_B$  refers to node is  $X$  state in layer  $A$  and  $Y$  state in layer  $B$  respectively. Fig. 2 shows the possible transitions between states of nodes, the arrow out from a given state of node at time step  $t$  points to its possible successor state at time step  $t + 1$ . The transition probability  $p_i^{X_A Y_B \rightarrow X'_A Y'_B}(t)$  from state  $X_A Y_B$  to its successor  $X'_A Y'_B$  of node  $i$  at time step  $t$  is given as follows:

$$p_i^{S_A S_B \rightarrow S_A I_B}(t) = q_i(t)(1 - q_i^A(t))(1 - \kappa),$$

$$p_i^{S_A S_B \rightarrow I_A S_B}(t) = (1 - q_i(t))q_i^A(t),$$

$$p_i^{S_A S_B \rightarrow I_A I_B}(t) = q_i(t)(1 - q_i^A(t))\kappa + (1 - q_i(t))(1 - q_i^A(t)),$$

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