Case Report

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Epidemic Dynamics On Information-Driven Duplex Networks

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1. Abstract

Research on the interplay between different dynam- ics of epidemic spreading on multiplex networks has attracted considerable attention in recent years. When disease begins to spread publicly, people can obtain information in advance and take measures to prevent infection. Two actions performed by those who obtain disease information are (1) taking precautionary measures, such as wearing masks, to reduce infection probability and (2) rewiring connections from infected neighbors to other susceptible ones, that is, the target reconnection. Which of these two measures is better? In this study, we propose two information-driven adaptive models to investigate the interplay between epidemic and information spread on duplex networks, where the disease itself and information about it can evolve simultaneously. Monte Carlo simulations indicate interesting conclusions. Compared with no information-driven spreading process, both adaptive processes based on information-driven not only can slow down the speed of epidemic spread but can also diminish the epidemic prevalence at the final state. Furthermore, the target reconnection is more effective in restraining the epidemic spread than the reduction of infection probability. The target reconnection can help end the epidemic but reduction of infection probability cannot. In other words, social isolation measures have better inhibitory effects than wearing masks and taking other precautionary measures to improve individual immunity. Finally, the target reconnection on duplex containing two random networks displays a better effect of restraining the epidemic spread than that on the duplex containing two small- world networks.
Index Terms—Information-driven duplex networks; Epidemic dynamics; Adaptive process; Monte Carlo Simulations

2. Introduction

The spreading dynamic process that takes place on complex networks has attracted great attention for a long time [1–4]. Many studies initially focused on the spreading dynamic in isolated networks, such as the spread of computer viruses and sexually transmitted diseases on scale-free networks [5–8], and the diffusion of various types of information such as health behavior [9], exercise contagion [10], and online innovation [11]. However, many of these related studies have investigated the spreading dynamic independently in a single network. In the real world, many networks are interconnected with others and form components of larger, complex systems. For instance, in a social system, a group of individuals interact with each other through different modes: an individual interacts with Xiang Wei, Hongxiao Wang,Shuai Liu, Riu Li, Bo Yang and Zhiyong Li are with the School of Engineering, Honghe University, Yunan 661100, China. Junchan Zhao is with the School of Mathematics and Statistics, Hunan University of Technology and Business, Changsha, China. others through online social systems (such as Facebook and others) and physical contact systems (such as colleague cir- cles). Furthermore, diseases that spread in physical contact networks interact with the corresponding disease information diffusion in online networks. Thus, the spread of disease information may play an important role in controlling the epidemic. Therefore, establishing models and measuring the impact of information spread in complex systems has become an important research issue.

Many studies analyze dynamic processes that occur in interacting networks. Lee et al. [12] proposed a mathematical framework for describing an interactive network consisting of a set of coupled network layers with different and specific characteristics. Subsequently, many related topics, such as dif- fusion and synchronization [13–16], growing models [17, 18], cascades [19], evolutionary games [20, 21], among many others, are investigated on interacting networks or multiplex networks. Two competitive viruses were investigated in two- layer networks by using the bond percolation analysis [22]. A study on bisexual men in the United

States showed that they were the medium of connecting pathogens from a network of heterosexual men with that of homosexual men [23, 24]. The susceptible infected refractory (SIR) process was studied on interacting networks [25] and revealed that epidemics spread between network layers at a threshold of βc, below which the disease does not spread. The expressions of the epidem- ic threshold was developed to analyze the time evolution with respect to the changes of various parameters [26]. A synergistic behavior spreading model on two-layer networks was proposed [27], the results showed that the synergistic interactions can greatly promote the spread in both layers. Furthermore, some models are proposed to investigate the interplay between epidemic and information spread (IEIS) on multilayer networks. Epidemic spread and information spread were analyzed through a microscopic Markov chain [28], which revealed that information spread can prevent infec- tion. The simulations also revealed that information spread can effectively raise the epidemic threshold [29]. A local awareness-controlled contagion spread model was proposed on multiplex networks [30].

Through numerical simulations, the emergence of a threshold phase transition with the local awareness ratio was revealed. The interplay between risk perception and epidemic spreading on multiplex networks was investigated [31], which showed that the similarity between the information and real networks determined the possibility of stopping the infection. The epidemic threshold for IEIS model is derived [32], showing that the epidemic threshold depends on both the network structure and dynamics of information spreading in social networks. The individual behavior status was introduced for the IEIS model in which each individual can change their behavior toward contact with the infection or information source [33], this model displayed the decrease of the individual behavior rate leading to the reduction of disease spread and increasing the threshold. Based on the level of prevention based on individual heterogeneity [34], altruistic behavior of infected individuals can effectively inhibit the spread of epidemics. Most of the aforementioned studies on the diffusion dynamics of this complex interaction are based on static networks. However, when individuals are aware of the disease, they sometimes cut off contact with infected persons, resulting in changes of the network structure. Currently, researchers pay more attention to the IEIS model on adaptive networks. Many studies have shown that separating the susceptible individuals from adaptive behavior is an effective strategy to reduce the interaction between susceptible and infected (SI) persons, and also hinder the disease outbreak [35, 36].

A multiplex network with two layers is built [37]in which the information spreading layer is a time-varying network. Compared with Monte Carlo simulations, the multiplex network showed higher prediction accuracy for epidemic threshold. A concrete interplay model was proposed to investigate the interplay between epidemic spread and adaptive behavior in populations. Furthermore, the model revealed that the synchronous adaptive behavior of individuals has a greater impact on the epidemic thresholds and prevalence than asynchronous adaptive behavior [38]. The results showed that the adaptive process could not only slow down the spread of the epidemic but also significantly reduce the final epidemic. In fact, when people obtain disease information from their friends or the media, they take adaptive behavior of preventive measures such as reducing infection probability or cutting off contact with infected individuals from being infected. In this study, we investigate the IEIS model in multiplex networks on the same population. The mean-field theoretical analysis and pairwise approach are used to model the two information-driven adaptive behaviors, and corresponding Monte Carlo simulation results revealed that human adaptive behaviors can have beneficial effects on the spread of the disease. The rest of this paper is organized as follows. The models based on the mean-field and pairwise methods are proposed in section II. Then, the Monte Carlo simulations are used to show the effects of the interactions between two spreading processes in section III. Finally, some discussions and conclusions are provided in section IV.

3. Model

3.1. Model description

The multiplex networks with two-layer homogeneous com- plex networks named A and B have the same size with differ- ent intra-layer connectivity, as shown in Fig. 1. In the disease layer, the dynamics of disease spread satisfies susceptible- infected-susceptible (SIS) processes. Although people contract an infection in the disease layer, they can obtain disease infor- mation in advance.

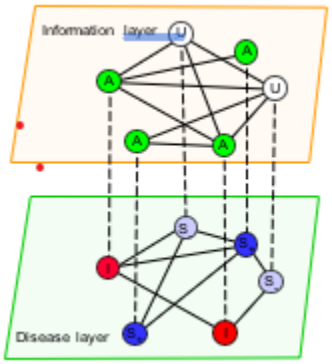

Fig. 1: A duplex networks model. Dashed and solid lines represent interlayer and intralayer links, respectively.

The dynamic of information spreading in the information layer is an unknown-known-unknown process. When the individuals have information on the epidemics, the individuals' states are known (A). Conversely, the individuals' states are unknown (U). The known individuals reduce the risk of infection, while the unknown ones have no information on how to prevent infection. Based on the model described, the spreading process is in accordance with the following rules:

(i) Information spreading in information layer. Information can come from two communication sources: known neighbors or individuals who are already infected and obtain information automatically. The unknown individuals can obtain informa-
tion from known neighbors with probability β1, while the known individuals can forget the information with probability μ1.

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(ii) Disease spreading in disease layer. Unknown susceptible individuals S− are infected by the infected neighbors with probability β2, while the infected individuals (I) recover with probability μ2.

Due to the impact of information spreading, known susceptible individuals S+ can use two adaptive meth- ods to prevent infection. One is to reduce the probability of being infected with parameter σβ2(σ < 1), as shown in the left panel of Fig. 2. The other is cutting off a certain number of connections with the infected ones, and then reconnecting to the same number of susceptible individuals in the disease layer, as shown in the right panel of Fig. 2. Therefore, the disease layer for physical contact is a time-varying network generated by the information-driven process, while the information layer for the virtual contact is a static network.

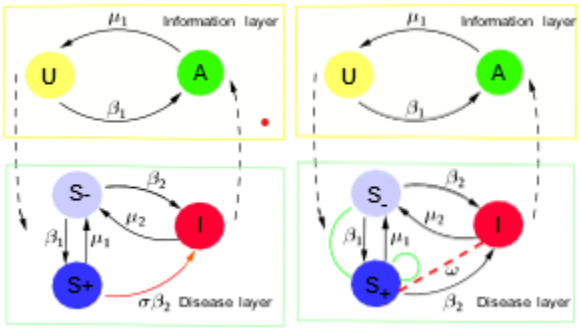

Fig. 2: The individuals adaptive behaviors toward reduction of infection

d[A]
= β [U][A] − μ [A] + [A] : ([S] + [I]) − [A]),11+dtd [U]
= μ1A − β1[U][A], dtd[S−]
= μ [S] + μ [I] − β [S] [S] − β [S] [I],1 +2 dt 1 − +2 −d[S+]
= −μ [S] + β [S][S] − β [S1 − +2 +] [I] + ([S+] [A] − [S+]),dtd [I]
= −μ2I + β2[S−][I] + β2[S+] [I] dtd [II] dt
= β2[S−I]([S−I] [S−] + 1) + β2[S+I] ([S+I] [S+] + 1) − 2μ2[II], d[S−S−]
= μ [S S] + μ [S I] − 2β [S S] − 2β [S S] dt

probability (left) and target reconnection (right). Red solid line on the left [S−S−][S−S+] [S−S−][S−I] panel represents aware susceptible individuals who reduce the probability of being infected with the parameter σβ (σ < 1). Dashed red line represents −β1 [S− — β2] , [S−] 2 aware susceptible individuals cutting off a certain number of connections with the infected ones and green solid line represents those who reconnect to the d[S+S+] dt = β2[S−S+] ([S−S+] [S−] + 1) + ω [S+I] − 2μ1[S+][S+] 2 same number of susceptible individuals on the right panel. −β2[S+ [S+I] S+](+ 1) + β1[S−S+ [S+] [S−S+]](+ 1) − β2 [S−] [S+S+][S+I] [S+] d[S+I] = −ω[S dtI] + β1[S−I] + 2β2[S+S+] + β2 [S−S+][S−I] [S−] (2) d[A] +β2 [S+S+] [S+I] [S+] + β1 [S−I][S−S+] [S−] — β2[S+I]([S+I] [S+]+ 1),
= β1(k)[U][A] + (1 − μ1)[A]([A] = [S+] + [I]), d[S−I] μ [S I] + β [S S] + β [S−S−][S−I] β [S−S+][S+I] dt dt 1 +2 − +2 [S−] 2 [S+] d [I]
= β2 (k) [S−][I] + σβ2 (k) [S+][I] + (1 − μ2)I (1) dt +2β [S S] − β [S I]([S−I] + 1) − β [S−] [S−I][S−S+] [S−] d[S−] d[S−S+] ω
= [SI] + 2μ [S S] + 2λ [S S] − α [S S] − β S−S+][S+I] dt
= μ1[S+] + μ2I − β2 (k) [S−][I] − β1 (k) [S−][S+], + dt 21 + + 1 − − 1 − +2 [S+] d[S+]
= β (k) [S] [S] + β (k) [S] [I] − σβ (k) [S] [I], +β1 [S−S−][S−S+] − β [S−] [S−S+][S−I] − β [S S] ([S+] [S−S+] + 1), [S−] dt 1 -μ1[S+], − +2 −2+ where the first to the fifth line has the same definition (3)

where the first line of Eq. (1) represent the information spreading dynamic in the information network, and the other lines of Eq. (1) represent the disease spreading dynamic in the disease network. Specifically, in the first line of Eq. (1), as the lines in Eq. (1). In the first line, the third term on the right side stands for the known susceptible individuals, which include those who are already infected and obtain information automatically. The adaptive process can be de-scribed by the last terms of d[II] , d[S−S−] , d[S+S+] , d[S−I] , the first term on the right side is the probability that unaware d[S+I] and d[S−S+] dt dt dt dt individuals [U] and those infected [A] by at least a aware dt dt in the pairwise approach. The pairwise

3.2. Dynamic Model

According to the rules in II-A, the dynamic processes of information and epidemic spreading are theoretically analyzed in this section. In particular, for the two individuals adaptive behaviors, the mean-field and pairwise analyses were adopted in the model. X is set as a state variable, so [X] represents the expected value of the individual in state X. The average degree of each layer is (k). According to the reduction of infection probability to prevent infection with the classical mean-field approach, one can obtain prevent infection with the pairwise approach, one can obtain neighbor, and the second term stands for the probability that aware individuals does not recover. In the second line, the first term on the right side stands for the probability that unaware susceptible individuals [S−] who are infected [I] by at least one infected neighbor, the second term stands for aware susceptible individuals [S+] who are infected [I] by at least one infected neighbor, and the third term stands for the infected individuals who do not recover. In the third line, the first and second terms on the right side stand for unaware susceptible individuals [S−] who recover from [S+] and [I], respectively. In the fourth line, the first and second terms on the right side stand for unaware susceptible individuals [S−] who are infected by at least a known neighbor (including known and infected individuals).

The third term stands for known susceptible individuals [S+] who are infected by at least one infected neighbor, and the fourth term stands for known susceptible individuals [S+] who recover to [S−]. Comparatively, let AB be the link, and the state of two con- necting nodes are A and B. [S−S+] represents the expected number of links connecting a unknown susceptible node to a known susceptible node. According to cutting off [S+I] link to analysis is based on a well-known closure approximation given by [ABC] = [AB][BC] with the assumption that the degree of each individual obeys Poisson-distribution [39]. In general, obtaining exact solutions to such complex differential equations in Eq. (2) may be difficult; thus, we show the Monte Carlo numerical results of the equations instead of the theoretical analysis in the following section.

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3.3. Monte Carlo Simulations and Analysis

To obtain a clear insight into the interplay between infor- mation diffusion and epidemic spreading on top of multiplex networks, we consider the duplex networks containing two homogeneous networks. The sizes of each layer N are 2000, 3000, 4000 and 5000 respectively, and the average degrees k are 100, 150, 200 and 250 respectively. The connection probability of links on each layer is 0.05. We perform our models with the following two topologies:

1. ER-ER duplex: Each layer is formed independently by the random network generation algorithm proposed by Erdo¨s and Re´nyi [40].
2. WS-WS duplex: Each layer is formed independently by the small-world networks generating algorithm proposed by Watts and Strogatz [41]. Specifically, starting from a ring, one end of each link is rewired with a probability of 0.3 to connect the other node randomly selected from the network.

A. Simulation with adaptive processes

Monte Carlo simulations for no information-driven and information-driven(Eqs.(1) and (2)) spreading processes are conducted to display the evolutionary effects. The initial fraction of infected nodes is set to 0.01, the values of spreading probability are $\beta 1 = 0.2$ and $\beta 2 = 0.2$, and the values of recovering probability are $\mu 1 = 0.5$ and $\mu 2 = 0.5$. When people obtain the disease information from the information network and take two preventive measures to prevent being infected. One measure is reducing the infection rate and the other is target reconnection. We reduce the infection rate $\beta 2$ by the factor σ to reduce the infection rate, and we set $\sigma = 0.5$. For the target reconnection, the reconnection rate is $\omega = 0.001$, which means that the known susceptible individuals S+ cuts off $N\omega$ connections with the infected individuals for target reconnection. The Monte Carlo simulation results of infection prevalence ρ evolving with time T for three spreading processes were shown for the ER-ER and WS-WS duplexes, respectively. The comparison of the average infection prevalence ρ for the mean-field and pairwise approaches with the corresponding no information-driven model are presented in this section. Figs. 3, 4, 5 and 6 display the simulation results of infection prevalence ρ evolving with time T for the ER-ER duplex with different N.

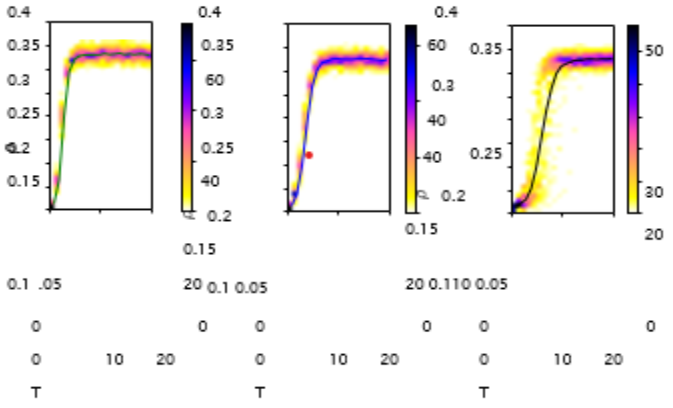

Fig. 3: (Color online). The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle), and target reconnection model (right) on ER-ER duplex (N=2000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

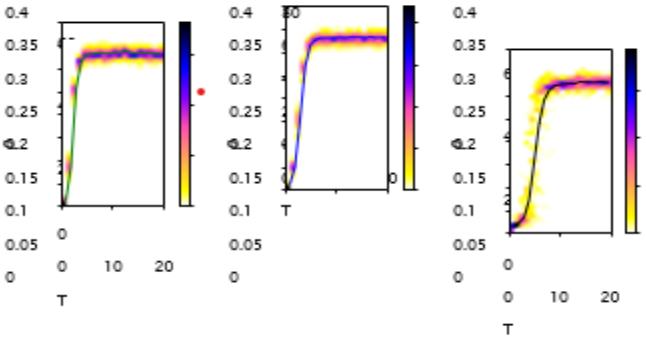

Fig. 4: (Color online). The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle), and target reconnection model (right) on ER-ER duplex (N=3000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

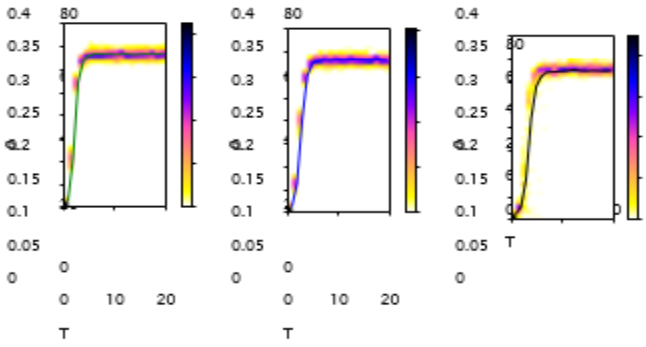

Fig. 5: The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle), and target reconnection model (right) on ER-ER duplex (N=4000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

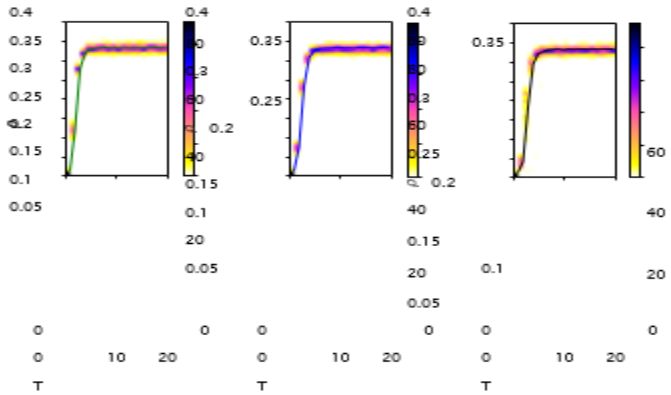

Fig. 6: The Monte Carlo simulation result of infections prevalence ρ evolving with time T for no information-driven model (left), reduction

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of infection probability model (middle), and target reconnection model (right) on ER-ER duplex (N=5000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

The colored area shows the number distribution of ρ and the lines show the average infection prevalence ρ based on 100 realizations. Fig. 7 displays comparison of the average infection prevalence for three models with different N. Simultaneously, Figs. 8, 9, 10 and 11 show the simulation results of infections prevalence ρ evolving with time T for the WS-WS duplex with different N , Fig. 12 displays comparison of the average infection prevalence for three models with different N . Figs. 7 and 12 show the results compared with the no information-driven process. Both adaptive processes not only slow down the speed of epidemic spreading but can also decrease the infection prevalence at the final state for both the ER-ER and WS-WS duplexes. Furthermore, this observation indicates that the adaptive process of the target reconnection results in better measures than the reduction of the infection rate based on the parameters set in this study.

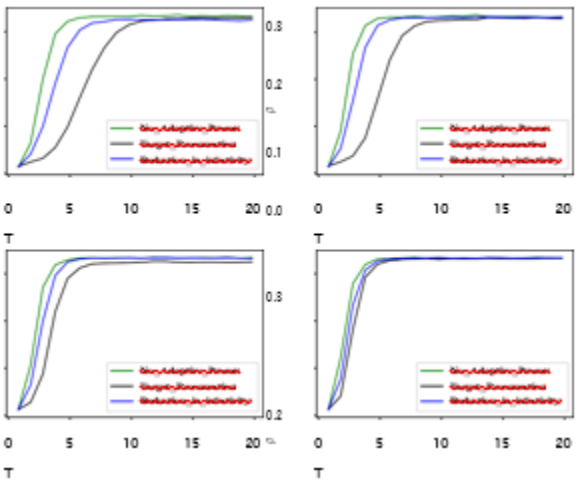

Fig. 7: (Color online). The Monte Carlo simulation results of average infection prevalence of ρ 100 realizations evolving with time T for three models with N=2000, 3000, 4000 and 5000 on ER-ER duplex

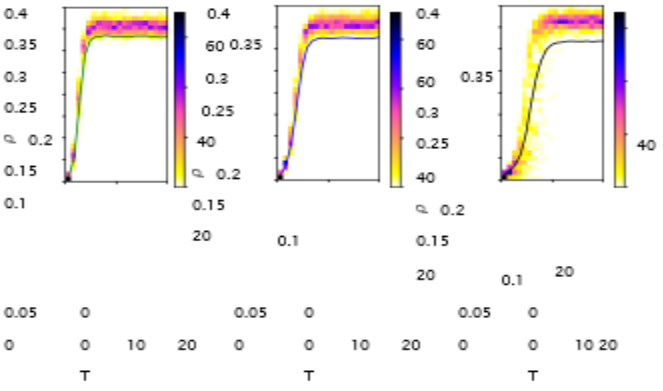

fig. 8: The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle) and target reconnection model (right) on WS-WS duplex (N=2000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

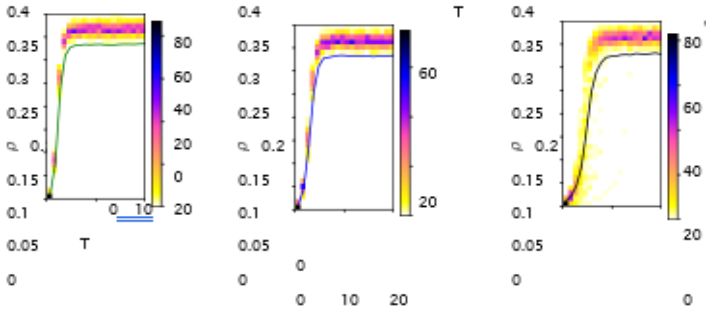

Fig. 9: The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle) and target reconnection model (right) on WS-WS duplex (N=3000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

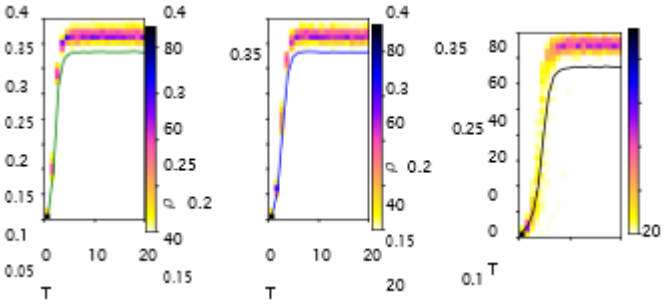

Fig. 10: The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle) and target reconnection model (right) on WS-WS duplex (N=4000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

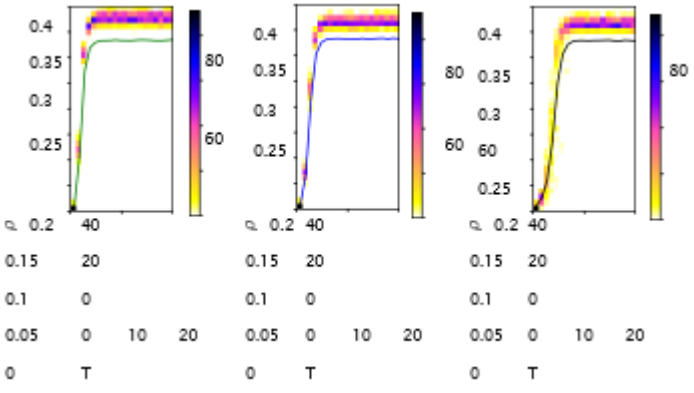

Fig. 11: The Monte Carlo simulation result of infection prevalence ρ evolving with time T for no information-driven model (left), reduction of infection probability model (middle) and target reconnection model

(right) on WS-WS duplex (N=5000). The colored area shows the number distribution of ρ based on 100 realizations and the lines show the average of the 100 realizations.

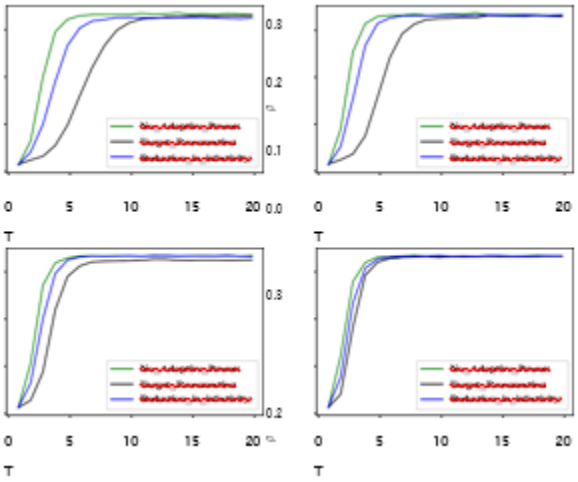

Fig. 12: (Color online). The Monte Carlo simulation results of average infection prevalence ρ of 100 realizations evolving with time T for three models with N=2000, 3000, 4000 and 5000 on WS-WS duplexes.

B. Sensitivity analysis of parameters

Finally, the Monte Carlo simulations are used for sensitivity analysis of the parameters. Considering the ER-ER and WS- WS duplexes with N = 3000, the comparison of ρ for different values of reducing the infection rate σ and target reconnection are shown in Figs. 13 and 14. Fig. 13 presents the average final state with different parameters for the WS WS duplex. We can obtain similar observations from Figs. 7 and 12 for the ER-ER and WS-WS duplexes. The left panel reveals that a smaller reduction of spreading probability σ leads to better suppression of virus transmission. The right panel reveals that more reconnections lead to better suppression of virus transmission. Fig. 14 displays the same conclusion as in Fig. 14 for ER-ER duplex. The same observations for different models and parameters are obtained, which reveal that the conclusions are not sensitive to the parameters. Note that the target reconnection can make the epidemic disappear but reduction of infection probability cannot do so on the ER- ER duplex. The target reconnection method displays a better effect of restraining the spread of the epidemics spreading for the ER-ER duplex than the WS-WS duplex. The right panel of Fig. 14 shows that when the reconnections are m = 4, the infections prevalence ρs toward zero evolves with time T in some simulations for the ER-ER duplex. This condition

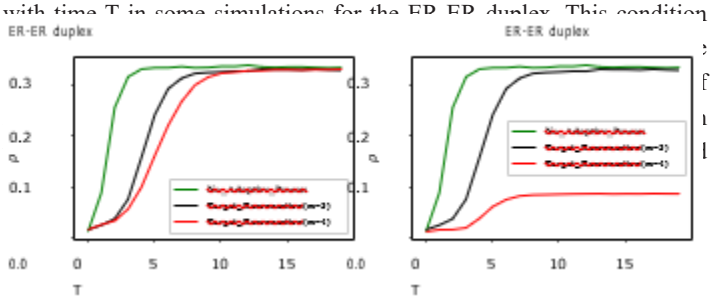

Fig. 13: (Color online). Comparison of infection prevalence ρ evolving with time T for reducing infection probability with different values of σ (left) and target reconnection for different values of m (right) on information-driven process.

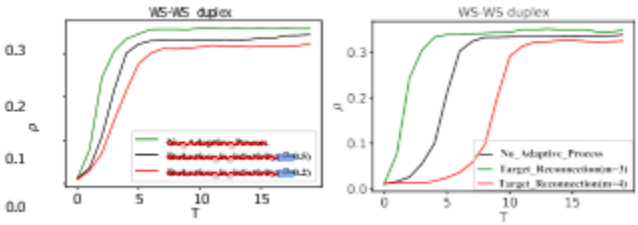

Fig. 14: (Color online). Comparison of infection prevalence ρ evolving with time T for reducing infection probability with different values of σ (left) and target reconnection for different values of m (right) on information-driven process.

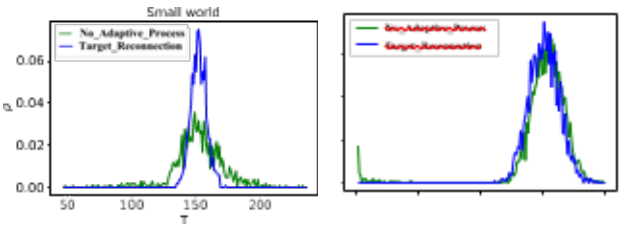

Fig. 15: (Color online). Degree distribution of original disease network and that after the adaptive process of the target reconnection. The original disease network is a random network(left) and the original network is a small- world(right).

The difference might be due to the network structure variation in the adaptive process of the target reconnection method. When the degree distribution of the original net- work is approximate to the Poisson-distribution with mean degree of approximately 150, the green spot marker on the left panel of Fig. 15 shows the degree distribution of the original random network, and that on the left panel shows the degree distribution of original small-world networks. After their connections are rewired, the degree distribution of the two kinds of networks at the final state deviates from the original distribution. For the ER-ER duplex, the degree distribution deviates slightly from the original distribution. However, for the WS-WS duplex, the degree distribution deviates more from the original distribution. The reason is that when the initial network is random network, many long-range connections exist in the random network, so that the virus can spread evenly to the entire network, and the information-obtained nodes are evenly distributed. The reason is that when the initial network is a random network with many long-range links, the virus can evenly spread to the entire network, and the nodes that obtain information are evenly distributed. When the nodes that obtain the information are reconnected with the broken edges, they can evenly select any healthy nodes to reconnect. When the initial network is a small- world, where only a small number of long-range connections exist, the virus can only spread along the neighbors of the infected node, while the healthy node far from the infected node obtains more connection opportunities, thereby

forming a larger degree of nodes. The existence of these larger degree of nodes accelerate the spread of the virus when these nodes are infected. Therefore, for the adaptive process of reconnection, the initial network is a random network, that can obtain an improved suppression effect than the initial network, which is a small-world.

4. Conclusions

The interplay between the spread of disease and the diffu- sion of information was investigated. Two information-driven adaptive models were proposed to reveal the interplay between the epidemic and information spread on duplex networks. Analogous conclusions were obtained for the duplex networks formed by the random and small-world networks. Monte Carlo simulations verify that both the adaptive process based on the information-driven models not only can slow down the speed of epidemic spreading, but can also diminish the epidemic prevalence at the final state. Furthermore, the target reconnection method displays a better effect of restraining the epidemic spread than the method of reducing infection probability because the target reconnection can make the epidemic disappear but the method of reducing the infection probability cannot. Finally, the target reconnection on the duplex containing two random networks was more effective in restraining the epidemic spread than that on the duplex containing two small-world networks. The reason was that the target reconnection of the WS-WS duplex led to an increase in the number of nodes with high degree, thereby accelerating the spread of the virus when the nodes were infected. This work may shed light on understanding that which method does human should choose to suppress the spread of disease.

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