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Research article

Effect of decay behavior of information on disease dissemination in multiplex network

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Abstract: The diseases dissemination always brings serious problems in the economy and livelihood issues. It is necessary to study the law of disease dissemination from multiple dimensions. Information quality about disease prevention has a great impact on the dissemination of disease, that is because only the real information can inhibit the dissemination of disease. In fact, the dissemination of information involves the decay of the amount of real information and the information quality becomes poor gradually, which will affect the individual's attitude and behavior towards disease. In order to study the influence of the decay behavior of information on disease dissemination, in the paper, an interaction model between information and disease dissemination is established to describe the effect of the decay behavior of information on the coupled dynamics of process in multiplex network. According to the mean-field theory, the threshold condition of disease dissemination is derived. Finally, through theoretical analysis and numerical simulation, some results can be obtained. The results show that decay behavior is a factor that greatly affects the disease dissemination and can change the final size of disease dissemination. The larger the decay constant, the smaller final size of disease dissemination. In the process of information dissemination, emphasizing key information can reduce the impact of decay behavior.

Keywords: disease dissemination; information dissemination; decay behavior; multiplex network

1. Introduction

Disease dissemination has a great impact on social stability and human life [1]. Studying and mastering the law of disease dissemination can provide strategies for controlling disease dissemination,

which is of great significance to maintain social stability and people's happy life. Under the background of COVID-19, some scholars have used the epidemic dissemination models to predict the development of the local epidemic situation, so as to provide theoretical support for the government to grasp the development of the epidemic [2,3].

The study of disease transmission has a long history. Different from the research methods in many other fields, the experiments related to epidemic dissemination cannot be carried out in human groups. Therefore, when studying the dissemination of epidemic, scholars prefer to establish some theoretical models to describe the dissemination process of epidemic. McKendrick et al. divided the population into three categories according to the infection: susceptible, infected and removed. The famous SIR model is constructed [4]. And they put forward the SIS compartment model and the threshold theory to distinguish whether the disease is epidemic or not. In view of the differences in dissemination mechanisms, scholars expanded the classical models and established SEIR, SEIS and other models, which laid a foundation for the study of infectious disease dynamics.

As a typical dynamic process on complex networks [5], epidemic dissemination has attracted extensive attention with the development of complex network theory [6–10]. With the emerging social media such as Twitter, Facebook and WeChat in the past few years, the dissemination dynamics of information such as news and rumors on online social networks has aroused much attention [11]. Based on the development of network science, a large number of efforts have been devoted to the study of information dissemination on complex social networks [12–15]. But most complex systems in the real world are coupled by multiple single networks with different structures and functions. The single network is a subnetwork in the whole multiple network, and there are many connections and interactions between the subnetworks. In a multiple network, each layer has a different structure, and the nodes in all layers are the same [16,17]. Understanding the dissemination dynamics on multiple networks will help to formulate more effective strategies to control or make full use of these dynamic processes. A large number of scholars have studied the coupling between information and disease [18–22]. For example, Granell et al. through studying the relationship between disease transmission and infection awareness, found that the critical point for the onset of the epidemics has a critical value (metacritical point) defined by the awareness dynamics and the topology of the virtual network, from which the onset increases and the epidemics incidence decreases [23]. After that，them presented an extended analysis of a generalization of a model of competing spreading processes on multiplex networks. The results reveal that existence of a metacritical point is rooted in the competition principle and holds for a large set of scenarios [24]. Shang through studying the effects of three forms of awareness on the spread of a disease in a random network, found that awareness can raise the epidemic thresholds [25]. Wang et al. investigated the asymmetrical interplay between the two types of spreading dynamics and found that information spreading can effectively raise the epidemic threshold [26]. Zhang et al. studied the impact of individual behavioral response to disease information and found that the behavioral response brought by the number of infected people in adjacent nodes will reduce the scale of disease transmission [27].

Moreover, many scholars have coupled the unaware-aware-unaware(UAU) model with the infectious disease model to study the interaction between information and disease. Nie et al. introduced the inhibition intensity by taking the number of node neighbors as a reference. It was found that the infection threshold increased with the increase of inhibition intensity, and reached saturation when the inhibition intensity was large enough [28]. Fan et al. introduced the individual behavior state to each node in the multiple network. Through threshold analysis and numerical simulation, the research found

that the disease dissemination can be reduced by reasonably controlling the information transmission and individual behavior [29]. Hu et al. studied the impact of individual behavior on information dissemination on the basis of timeseries coupling network, and found that communication behavior will have a two-stage impact on information dissemination [30].

In the above multiplex network model, individuals are only divided into aware and unaware states. However, Rich et al. set up experiments to correct the behavior of people with different amounts of information, and concluded that implied misinformation is more difficult to correct than explicitly provided misinformation [31]. So, in the process of dissemination, the amount of real information is variable. Individuals with different amounts of information have different attitudes towards diseases. Elly et al. explained the problem of "why people accept information of different nature" from multiple psychological perspectives, such as emotionality, motivated reasoning and cognitive reasoning, including false news, misinformation and disinformation [32]. Shang studied a discrete-time epidemic dynamics with the presence of three forms of individual awareness and found that awareness can raise the epidemic thresholds [33]. People will change their behavior when they are aware of the existence of a disease [34,35].

Previous studies believe that the quality of information is invariable in the process of disease dissemination. In this paper, we will consider the impact of the quality of information about disease owned by individuals on disease dissemination. Decay behavior will lead to the loss of real information amount related to disease. When the amount of information is reduced through decay behavior, the disease prevention measures taken by individuals will be weakened, which will increase the probability of disease dissemination. Multiple decay eventually leads to the fact that the information has little inhibitory effect on the disease dissemination. Studying this factor can be closer to the actual situation, grasp the impact mechanism of information dissemination on disease dissemination and explore the strategies to inhibit disease dissemination. Based on the principle of dissemination and multiple networks, the paper connects the decay behavior of information with the disease dissemination model, and constructs the coupling network model of the information layer and physical contact layer. Based on the model, the threshold condition of disease dissemination in the coupled network is derived, and the numerical simulation is carried out to further analyze the decay constant, information spreading rate and conversion rate and other key factors affecting the spread of disease. Finally, some suggestions on the inhibitory effect of information dissemination on the spread of disease are given.

The rest of this paper is organized as follows. This paper introduces the multiplex networks model and analyzes the spreading threshold in Section 2. In Section 3, we perform numerical simulations. Finally, the conclusions and suggestions are given in Section 4.

2. Disease dissemination model on multiplex network

2.1. Model assumptions

The dissemination of disease and information is interactive. The physical contact between people makes the disease spread, and the dissemination process of disease is affected by the dissemination of relevant information. We construct a two-layer network to express and simplify the dissemination mechanism (see Figure 1).

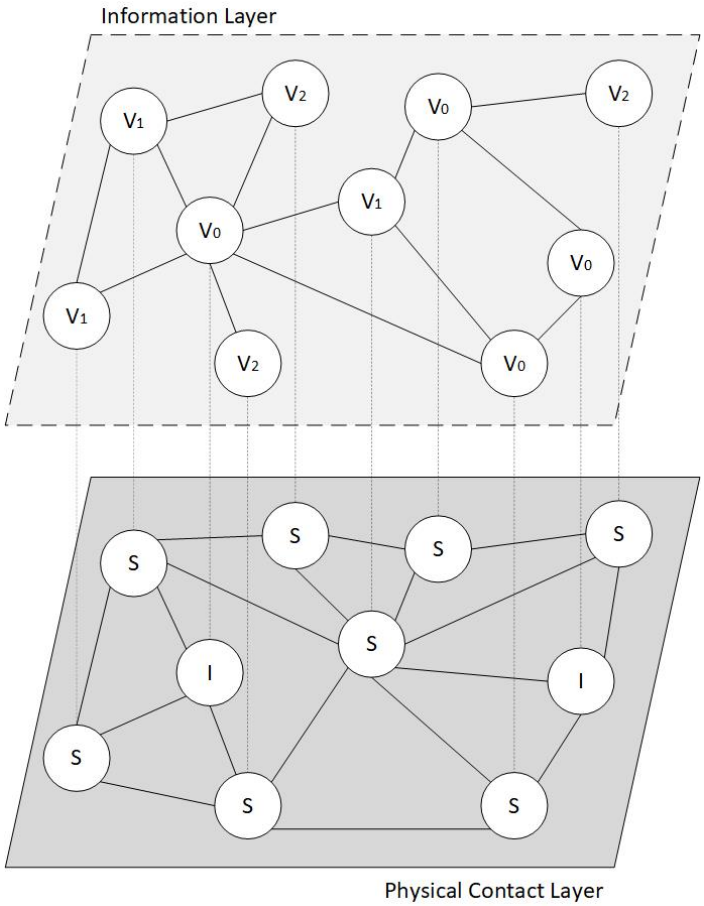


Figure 1. The coupled spreading process on a multiplex network with two layers.

The information dissemination process takes place in the upper network, in which the individuals have three states: the individuals with original information (V_0), the individuals with second-hand information (V_1) and the individuals with uncertain information (V_2). The disease dissemination process occurs in the lower network, in which the individuals have two states: susceptible (S) and infected (I). We construct a two-layer multiplex network that shares the same individuals in two layers, each node in one layer will be solely mapped into the corresponding node in the other layer.

One network (the upper network) is the information layer, and the other network (the lower network) is the physical contact layer. The number of nodes in the two layers is exactly the same. Since information can be spread through many ways, while disease can only be spread through physical contact, the degree of nodes in the two layers is different, where the degree of a node in the information layer (the physical contact layer) is denoted as $k_1(k_2)$. The joint degree distribution $P(k_1,k_2)$ represents the proportion of nodes whose information layer degree is k_1 and physical contact layer degree is k_2. The average degrees of information layer and physical contact layer are $\langle k_1\rangle=\sum_{k_1}k_1P(k_1,k_2)$ and $\langle k_2\rangle=\sum_{k_2}k_2P(k_1,k_2)$. Let's explain the dissemination mechanism in the two-layer network. We assume the multiplex dissemination process according to the following rules.

Assumption 1: Disease layer. In the physical contact layer, SIS model is used to explain the dissemination process of the disease, where each individual is either susceptible state (S) or infected state (I). If the susceptible individual contacts with the infected individuals, it will become infected

with probability β and $0 < \beta < 1$. The infected individuals will be cured with a certain probability μ and transformed into healthy individuals, $0 < \mu < 1$.

Assumption 2: Information layer. With the popularization of informatization and the diversified development of official media, in addition to traditional media such as news and newspapers, the government has also registered official accounts of major content platforms, so the coverage of information is very wide and most people will get the original information. Others will get information in other ways. As the information is passed from person to person, it loses its quality. In other words, original information about a disease case will lead to a much more determined reaction than information that has passed through many people before arriving at a given individual. Similar views have been mentioned in [35]. This phenomenon can be interpreted as the decay behavior of the amount of information. Decay behavior will lead to the loss of real information amount related to disease. In 2020, some experts pointed out that 75% of alcohol can kill COVID-19. However, the news of "drinking high alcohol to fight the coronavirus" had spread widely. Although relevant elements are retained in the information, the amount of information has deteriorated. An individual obtains original information, which is the most authoritative and authentic. We name it original information and mark it with V_0. Individuals get information from V_0, we name it second-hand information and mark it with V_1. Individuals get information from V_1, we name it third-hand information and mark it with V_2. According to this law, individuals get information from V_{n-1}, we name it nth-hand information and mark it with V_n. Although information can be spread many times, in order to facilitate research, we only divide information into three categories: V_0 represents the original information; V_1 represents the second-hand information; V_2 represents the uncertain information (information is spread many times, the decay behavior of information quality will lead to a large loss of real information, and will bring a lot of false information). Uncertain information is not all false information, but the amount of real information possessed by this state is relatively minimal. People with more real information tend to be more persuasive in communication and can update the information of people with less real information. Multiple decay eventually leads to the fact that the information has little inhibitory effect on the disease dissemination. We think that the role of individuals who obtain uncertain information and those who do not have information is similar in the multiplex network.

Assumption 3: According to the model, the node has six states, V_0S, V_1S, V_2S, V_0I, V_1I, V_2I. After the node is infected with the disease, it will take the initiative to find the most real information. We assume that once the individual is infected it becomes the infected state with original information immediately, that is, V_1I and V_2I become V_0I immediately. So individuals have four states: V_0S represents susceptible individuals with original information; V_1S represents susceptible individuals with second-hand information; V_2S represents susceptible individuals with uncertain information; V_0I represents infected individuals with original information. In order to make the paper more concise, we use S_0, S_1, S_2 and I_0 instead of V_0S, V_1S, V_2S and V_0I respectively.

Assumption 4: The disease prevention measures taken by individuals with different amount of information are different, so their dissemination probabilities are different, which can be explained as follows: Individuals with more real information have relatively sufficient preventive measures for the disease, and the probability of contacting infected persons will be smaller, which can more effectively inhibit the dissemination of the disease. In order to vividly describe the impact of the decay behavior of the amount of information, we introduce the decay constant ρ, $0 < \rho < 1$, so that the disease dissemination probability increases with the loss of information quality, which is $\beta_i = \beta(1-\rho^{i+1})$, $i = 0$,

1, *2*. β_0 represents the probability of infection of individuals with original information V_0. β_1 represents the probability of infection of individuals with second-hand information V_1. β_2 represents the probability of infection of individuals with uncertain information V_2. In other words, with the continuous loss of real information about disease, the decay constant governs how much the tendency to act is reduced with decreasing quality of information [35]. The inhibitory effect of information on disease dissemination will disappear if information is not refreshed.

2.2. Dissemination process

The specific dissemination process of the information is shown in Figure 2. Assumption 2 mentions that the more individuals have real information, the greater their influence. So individuals with original information are the most authoritative and authentic. Individuals with second-hand information or uncertain information will be transformed into individuals with original information with a certain probability λ when they communicate. Individuals with uncertain information will be transformed into individuals with second-hand information with a certain probability λ when they communicate. λ represents information spreading rate and $0 < \lambda < 1$. Due to decay or other factors, the quality of information will be reduced with a certain probability δ. δ represents information conversion rate and $0 < \delta < 1$.

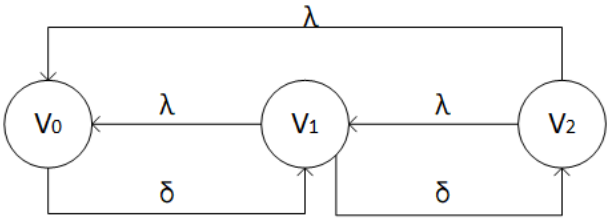


Figure 2. Dissemination process of information layer.

The specific dissemination process of the disease is shown in Figure 3. When individuals $S_i(i = 0,1,2)$ contacts I_0, S_i will become I_0 with a certain probability $\beta_i = \beta\left(1 - \rho^{i+1}\right)$. Individuals I_0 will be cured with a certain probability μ to convert the S_0.

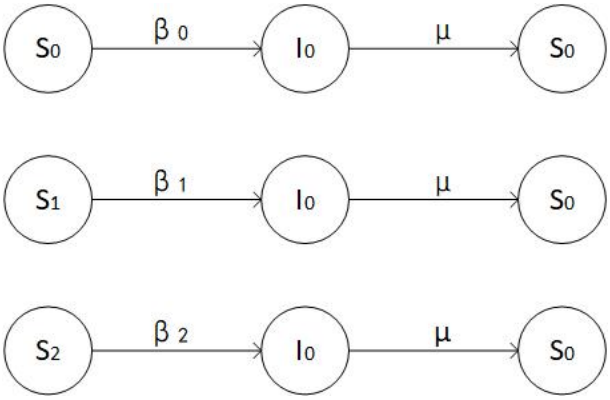


Figure 3. Dissemination process of physical contact layer.

The dissemination rules are as follows:

$$S_2 + S_1 \xrightarrow{\lambda} S_1 + S_1 \tag{1}$$

$$S_2 + S_0 \xrightarrow{\lambda} S_0 + S_0 \tag{2}$$

$$S_2 + I_0 \xrightarrow{\lambda} S_0 + I_0 \tag{3}$$

$$S_1 + S_0 \xrightarrow{\lambda} S_0 + S_0 \tag{4}$$

$$S_1 + I_0 \xrightarrow{\lambda} S_0 + I_0 \tag{5}$$

$$S_0 + I_0 \xrightarrow{\beta_0} I_0 + I_0 \tag{6}$$

$$S_1 + I_0 \xrightarrow{\beta_1} I_0 + I_0 \tag{7}$$

$$S_2 + I_0 \xrightarrow{\beta_2} I_0 + I_0 \tag{8}$$

$$S_0 \xrightarrow{\delta} S_1 \tag{9}$$

$$S_1 \xrightarrow{\delta} S_2 \tag{10}$$

$$I_0 \xrightarrow{\mu} S_0 \tag{11}$$

2.3. Dynamic model

Let $\rho_{k_1,k_2}^{S_0}, \rho_{k_1,k_2}^{S_1}, \rho_{k_1,k_2}^{S_2}, \rho_{k_1,k_2}^{I_0}$ be the fractions of nodes within degree compartment (k_1,k_2) in the states S_0 , S_1 , S_2 , I_0 , respectively. $\rho_{k_1,k_2}^{S_0} = \frac{S_0(k_1,k_2)}{N(k_1,k_2)}$, $\rho_{k_1,k_2}^{S_1} = \frac{S_1(k_1,k_2)}{N(k_1,k_2)}$, $\rho_{k_1,k_2}^{S_2} = \frac{S_2(k_1,k_2)}{N(k_1,k_2)}$, $\rho_{k_1,k_2}^{I_0} = \frac{I_0(k_1,k_2)}{N(k_1,k_2)}$, k_1 and k_2 are different node degrees of the same node in the information layer and the physical contact layer respectively. They satisfy the normalization condition $\rho_{k_1,k_2}^{S_0} + \rho_{k_1,k_2}^{S_1} + \rho_{k_1,k_2}^{S_2} + \rho_{k_1,k_2}^{I_0} = 1$, for each set of (k_1,k_2). A heterogeneous mean-field (HMF) theory could be developed for the evolution of these fractions as follows:

$$\frac{d\rho_{k_1,k_2}^{S_0}}{dt} = \lambda k_1 \theta_1 \rho_{k_1,k_2}^{S_2} + \lambda k_1 \theta_1 \rho_{k_1,k_2}^{S_1} - \beta_0 k_2 \theta_3 \rho_{k_1,k_2}^{S_0} + \mu \rho_{k_1,k_2}^{I_0} - \delta \rho_{k_1,k_2}^{S_0} \tag{12}$$

$$\frac{d\rho_{k_1,k_2}^{S_1}}{dt} = \lambda k_1\theta_2\rho_{k_1,k_2}^{S_2} - \lambda k_1\theta_1\rho_{k_1,k_2}^{S_1} - \beta_1 k_2\theta_3\rho_{k_1,k_2}^{S_1} + \delta\rho_{k_1,k_2}^{S_0} - \delta\rho_{k_1,k_2}^{S_1} \tag{13}$$

$$\frac{d\rho_{k_1,k_2}^{S_2}}{dt} = -\lambda k_1\theta_1\rho_{k_1,k_2}^{S_2} - \lambda k_1\theta_2\rho_{k_1,k_2}^{S_2} - \beta_2 k_2\theta_3\rho_{k_1,k_2}^{S_2} + \delta\rho_{k_1,k_2}^{S_1} \tag{14}$$

$$\frac{d\rho_{k_1,k_2}^{I_0}}{dt} = k_2\theta_3\left(\beta_0\rho_{k_1,k_2}^{S_0} + \beta_1\rho_{k_1,k_2}^{S_1} + \beta_2\rho_{k_1,k_2}^{S_2}\right) - \mu\rho_{k_1,k_2}^{I_0} \tag{15}$$

where $\theta_1 = \frac{1}{\langle k_1\rangle}\sum_{k_1} k_1 P(k_1,k_2)\left[\rho_{k_1,k_2}^{S_0} + \rho_{k_1,k_2}^{I_0}\right]$ denoting the possibility that a randomly chosen link in the information layer will reach A_0 state node, $\theta_2 = \frac{1}{\langle k_1\rangle}\sum_{k_1} k_1 P(k_1,k_2)\rho_{k_1,k_2}^{S_1}$ denoting the possibility that a randomly chosen link in the information layer will reach A_1 state node, and $\theta_3 = \frac{1}{\langle k_2\rangle}\sum_{k_2} k_2 P(k_1,k_2)\rho_{k_1,k_2}^{I_0}$ denoting the possibility that a randomly chosen link in the physical contact layer will reach a I_0 state node.

Condition (12) describes the change process of S_0 state node. In the information layer, the probability of connecting an S_2 node with a degree of k_1 to an S_0 node or an I_0 node is $\theta_1 = \frac{1}{\langle k_1\rangle}\sum_{k_1} k_1 P(k_1,k_2)\left[\rho_{k_1,k_2}^{S_0} + \rho_{k_1,k_2}^{I_0}\right]$. Similarly, the probability of connecting an S_1 node with a degree of k_1 to an S_0 node or an I_0 node is $\theta_1 = \frac{1}{\langle k_1\rangle}\sum_{k_1} k_1 P(k_1,k_2)\left[\rho_{k_1,k_2}^{S_0} + \rho_{k_1,k_2}^{I_0}\right]$. Therefore, the number of S_0 nodes increased by information exchange is $\lambda k_1\theta_1\rho_{k_1,k_2}^{S_2} + \lambda k_1\theta_1\rho_{k_1,k_2}^{S_1}$. Due to the decay of information quality, the number of S_0 nodes reduced is $\delta\rho_{k_1,k_2}^{S_0}$. In the physical contact layer, the probability of connecting an S_0 node with a degree of k_2 to an I_0 node is $\theta_3 = \frac{1}{\langle k_2\rangle}\sum_{k_2} k_2 P(k_1,k_2)\rho_{k_1,k_2}^{I_0}$. Therefore, the number of S_0 nodes reduced by disease dissemination is $\beta_0 k_2\theta_3\rho_{k_1,k_2}^{S_0}$. Due to disease recovery, the number of S_0 nodes increased is $\mu\rho_{k_1,k_2}^{I_0}$. Corresponding to Eqs (13)–(15), S_1, S_2 and I_0 will also be affected by the information layer and the physical contact layer, so as to change their own state.

2.4. Threshold on heterogeneous networks

Order that $\frac{d\rho_{k_1,k_2}^{S_0}}{dt} = 0$, $\frac{d\rho_{k_1,k_2}^{S_1}}{dt} = 0$, $\frac{d\rho_{k_1,k_2}^{S_2}}{dt} = 0$, $\frac{d\rho_{k_1,k_2}^{I_0}}{dt} = 0$, the steady-state solution of Eq

(15) is $\rho_{k_1,k_2}^{I_0}=1-\frac{(\eta_1)}{(\eta_2*\eta_3-\eta_4)}$.

$$\eta_1=\delta\mu\left(-\lambda k_1\theta_1-\lambda k_1\theta_2-\beta_2 k_2\theta_3\right)\left(\frac{2\delta+\lambda k_1\theta_1+\beta_1 k_2\theta_3}{\delta}+\frac{\delta-\lambda k_1\theta_2}{\lambda k_1\theta_1+\lambda k_1\theta_2+\beta_2 k_2\theta_3}\right) \tag{16}$$

$$\eta_2=\left(-\lambda k_1\theta_1-\lambda k_1\theta_2-\beta_2 k_2\theta_3\right) \tag{17}$$

$$\eta_3=\left(-\left(\mu+\beta_0 k_2\theta_3\right)\left(-\delta-\lambda k_1\theta_1-\beta_1 k_2\theta_3\right)+\delta\left(\mu+\beta_1 k_2\theta_3\right)\right) \tag{18}$$

$$\eta_4=\delta\left(-\lambda k_1\theta_2\left(\mu+\beta_0 k_2\theta_3\right)+\delta\left(\mu+\beta_2 k_2\theta_3\right)\right) \tag{19}$$

when $\theta_3=0$, it means that all nodes are in the susceptible state. Therefore, only $\theta_3>0$ can the disease be prevalent. Considering the equation $\theta_3=\frac{1}{\langle k_2\rangle}\sum_{k_1,k_2}k_2P(k_1,k_2)\rho_{k_1,k_2}^{I_0}=f(\theta_1,\theta_2,\theta_3)$ and $f(\theta_1,\theta_2,\theta_3)$ is strictly monotonically increasing function, the existence condition of non-zero solution for θ_3 is $\left[\frac{\partial f(\theta_1,\theta_2,\theta_3)}{\partial\theta_3}\right]_{\theta_3=0}>1$. Then, we can get

$$\frac{\langle k_2^2\rangle}{\langle k_2\rangle}\frac{\left(\delta^2\beta_2+\lambda k_1\left(\delta\beta_1(\theta_1+\theta_2)+\beta_0\theta_1\left(\delta+\lambda k_1(\theta_1+\theta_2)\right)\right)\right)}{\mu\left(\delta+\lambda k_1\theta_1\right)\left(\delta+\lambda k_1(\theta_1+\theta_2)\right)}>1 \tag{20}$$

here, $\langle k_2^2\rangle=k_2\sum_{k_2}k_2P(k_1,k_2)$, $\beta_0=\beta(1-\rho)$, $\beta_1=\beta(1-\rho^2)$, $\beta_2=\beta(1-\rho^3)$. Therefore, the threshold of the model is

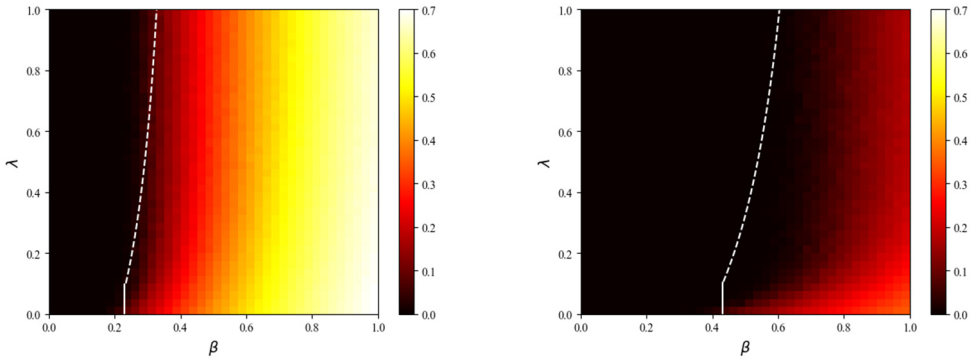
$$\frac{\beta}{\mu}>\frac{\langle k_2\rangle}{\langle k_2^2\rangle}\frac{\left(\delta+\lambda k_1\theta_1\right)\left(\delta+\lambda k_1(\theta_1+\theta_2)\right)}{\left(\delta^2(1-\rho^3)+\lambda k_1\left(\delta(1-\rho^2)(\theta_1+\theta_2)+(1-\rho)\theta_1\left(\delta+\lambda k_1(\theta_1+\theta_2)\right)\right)\right)}=\beta_c \tag{21}$$

we get the epidemic threshold β_c, above which epidemics are possible, but below which epidemics cannot occur. As can be seen from Eq (21), the threshold β_c is determined by θ_1 and θ_2. The number of infected nodes is almost zero when β approaches the threshold β_c. Therefore, the nodes in the multiplex network reflect the dissemination process of information. When β is close to the threshold β_c, θ_1 and θ_2 are irrelevant to the epidemic dissemination. Obviously, when $\theta_1=\theta_2=0$, the inhibition of information layer disappears, and the situation in the physical contact layer returns to the single layer. we can get $\beta_c^{'}=\frac{\langle k_2\rangle}{\langle k_2^2\rangle}\frac{1}{(1-\rho^3)}$, which is the critical threshold of disease only under the influence content of the decay behavior of information. As ρ increases, $\beta_c^{'}$ decreases and the disease becomes more easily transmitted. Therefore, decay behavior is an important factor in the disease dissemination. when $\rho=0$, we can get $\beta_c^{''}=\frac{\langle k_2\rangle}{\langle k_2^2\rangle}$, which is consistent with the threshold of

the traditional SIS model.

3. Numerical simulation

In this paper, a multi-layer network model is constructed by using the scale-free network generation algorithm, which is composed of 5000 nodes. The new nodes added in each iteration of the information layer have 6 edges, and the new nodes added in each iteration of the disease layer have 3 edges. Each simulation takes the average of 50 iterations as the output of the result.



(a) (b)

Figure 4. Stationary state and critical thresholds of the disease with different spreading rates.

The color maps represent the prevalence levels of disease. Dynamical parameters: information layer conversion rate $\delta = 0.4$, physical contact layer recovery rate $\mu = 0.6$, $\rho = 0.5$(a), $\rho = 0.8$(b). Each point in the grid 40 × 40 of the figure is obtained by averaging 50 numerical simulations.

As depicted in Figure 4, an increase in β can promote an epidemic. In the single subgraph of Figure 4, when the system is not affected by the information layer, the dissemination threshold is $\beta_c^{'} = \frac{\langle k_2 \rangle}{\langle k_2^2 \rangle} \frac{1}{(1-\rho^3)}$, which is the solid line part in the Figure 4. When the information layer will affect the dissemination of disease, the critical threshold shifts significantly, as shown in the dotted line. From Figure 4 (a),(b), we can find that the decay constant increases, the dissemination threshold of disease becomes larger and the dissemination scale of disease becomes smaller. And the change is obvious. The main reason for this result is that the greater the decay constant, the smaller the impact on the individual receiving information and taking precautionary measures.

We can see the final size of I_0 with the influence of information dissemination (see Figure 5). Under the same decay constant ρ, the final size of I_0 will increase with the increase of disease spreading rate β. Under the same disease spreading rate β, the final size of I_0 will decrease with the increase of decay constant ρ. When the decay constant ρ varies from 0.2 to 0.4, the final size of I_0 does not change greatly. When the decay constant ρ varies from 0.4 to 0.8, the final size of I_0 has decreased significantly. It shows that the inhibitory effect on disease has an incremental trend with the increase of decay constant ρ. With the increase of decay constant, the threshold of disease increases and the transmission scale decreases. From the above description, it can be concluded that the decay constant ρ, as the control parameter of decay intensity, has a great impact on the

dissemination of disease. Therefore, the government needs to improve official credibility and strive to cultivate people's cognitive ability to reduce the decay of information quality, which will effectively curb the dissemination of disease.

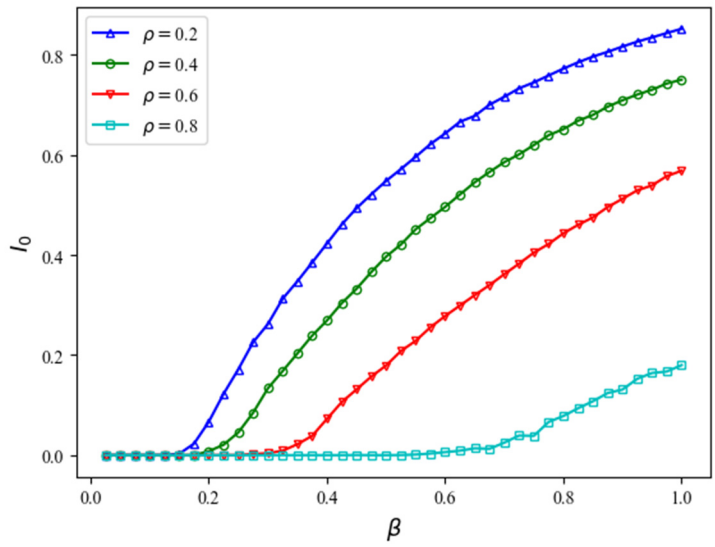


Figure 5. The final size of spreading dynamics I_0 with different disease spreading rate β and decay constant ρ. Dynamical parameters: information layer spreading rate $\lambda = 0.5$ and conversion rate $\delta = 0.4$, physical contact layer recovery rate $\mu = 0.6$.

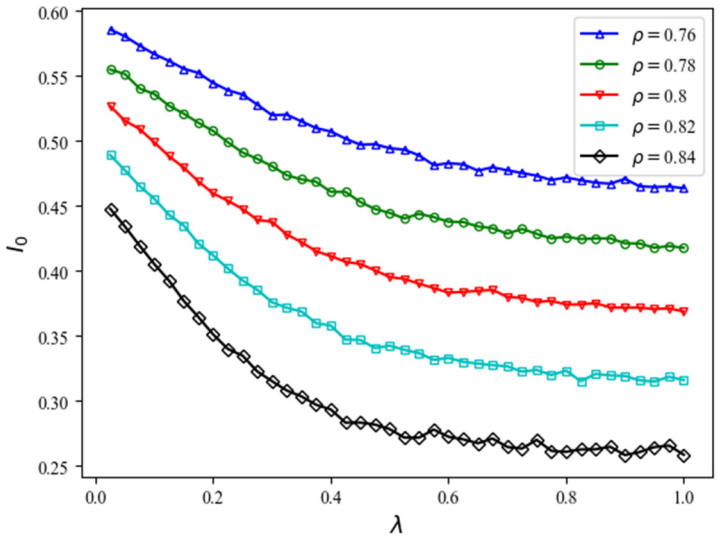


Figure 6. The final size of spreading dynamics I_0 with different information spreading rate λ and decay constant ρ. Dynamical parameters: information layer conversion rate $\delta = 0.4$, physical contact layer spreading rate $\beta = 0.5$ and recovery rate $\mu = 0.6$.

Figure 6 shows the impact of different information spreading rates and decay constants on disease dissemination. Under the same decay constant ρ, the final size of spreading dynamics I_0 will decrease with the increase of information spreading rate λ. However, this change is not obvious, indicating that the impact of information spreading rate is limited. Under the same information spreading rate λ, the final size of spreading dynamics I_0 will decrease with the increase of decay constant ρ. When the change of decay constant ρ is very small, the change of the final size of I_0 is still obvious, which further explains the importance of decay behavior. Therefore, in the process of controlling the dissemination of disease, improving the information quality may be more effective and cost-effective than expanding the dissemination channels of information and increasing the probability of information acquisition.

Figure 7 reflects the impact of the information layer on disease dissemination. Under the same information spreading probability, the final size of I_0 will increase with the increase of conversion rate. This change is relatively obvious, so the information needs to be repeated in the process of dissemination to reduce the conversion rate. Under the same conversion rate, the final size of I_0 will decrease with the increase of information spreading rate. When λ is between 0–0.4, the final size change of infected nodes is obvious, and when it is between 0.4–1, the change is not so obvious. In other words, in order to suppress the dissemination of disease, it is necessary to increase the spread of information, but when the information spreading rate increases to a certain value, the inhibitory effect is not so obvious. Therefore, when the government takes relevant measures to curb the dissemination of the disease, it should not simply expand the dissemination of information. Considering the cost, it is time to take other measures after the dissemination of information reaches a certain intensity, which can not only achieve a good suppression result and reduce the cost of spreading information, but also further suppress the spread of the disease through other means.

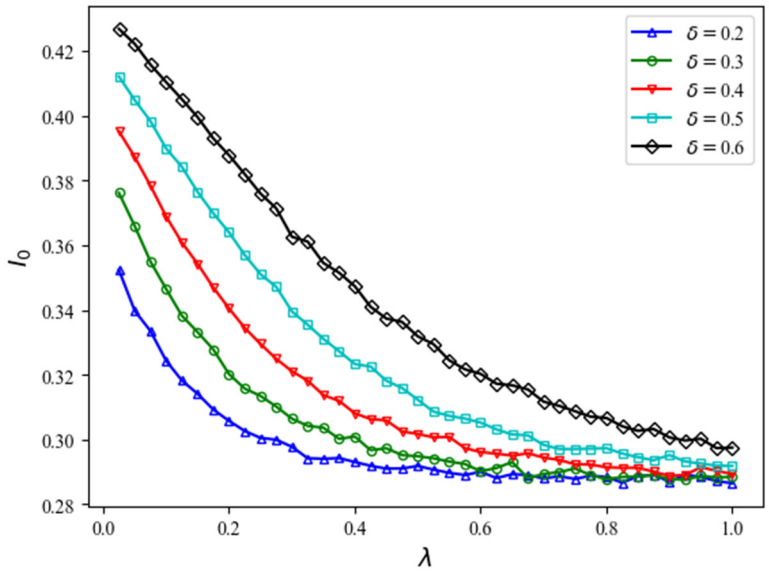


Figure 7. The final size of spreading dynamics I_0 with different information spreading rate λ and conversion rate δ. Dynamical parameters: physical contact layer disease spreading rate $\beta = 0.5$, recovery rate $\mu = 0.6$ and decay constant $\rho = 0.5$.

4. Conclusions

In this paper, the decay constant is introduced to build a multiplex network model of interaction between information and disease. Through mathematical analysis and numerical simulation, the influence of information dissemination on disease dissemination was finally concluded. Compared with the single study of information dissemination or disease dissemination, it is more practical and persuasive. Through the simulation analysis of the model, we concluded that the information layer has a certain inhibitory effect on the dissemination of disease. The influence of information spreading rate and conversion rate is relatively small, but the reduction of information quality caused by decay behavior will greatly affect the spread of disease.

Therefore, the government should pay attention to the dissemination of disease-related information during epidemic prevention and control. Our suggestions are as follows:

(1) In addition to expanding the intensity of information dissemination, we need to take timely measures to curb the dissemination of diseases, for example, the government issued masks, restricted travel and so on during the period of COVID-19. Because after the information spreading rate increases to a certain value, its inhibitory effect on diseases decreases to a great extent.

(2) The government should emphasize the importance of key information to minimize the impact caused by information decay behavior. For example, the conditions and environment of disease dissemination should be repeatedly emphasized for many times. Individual grasping key information can reduce the impact of decay behavior.

This paper directly considers the influence of information decay behavior on disease dissemination. In the future, we hope to build a multi-layer network model containing individual behavior to study the impact of information on behavior and the impact of behavior on disease dissemination, which will be closer to reality. In addition, the active state of individuals at different times is different. It is also a good choice to discuss temporal networks with decay behavior.

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Conflict of interest

The authors declare there is no conflict of interest.

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