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Epidemic Spreading on Weighted Co-Evolving Multiplex Networks

Bo Song [1], Huiming Wu [2], Yurong Song [2,*], Xu Wang [3] and Guoping Jiang [2]

[1] School of Modern Posts, Nanjing University of Posts and Telecommunications, Nanjing 210023, China; songbo@njupt.edu.cn
[2] College of Automation & College of Artificial Intelligence, Nanjing University of Posts and Telecommunications, Nanjing 210023, China; 1020051513@njupt.edu.cn (H.W.); jiangjp@njupt.edu.cn (G.J.)
[3] GBDTC, University of Technology Sydney, Sydney, NSW 2007, Australia; xu.wang-1@uts.edu.au
* Correspondence: songyr@njupt.edu.cn; Tel.: +86-1395-1842-705

Abstract: The individual behaviors driven by information diffusion show an undeniable impact on the process of epidemic spreading and have been continuously evolving with the dynamic processes. In this paper, a novel weighted co-evolving multiplex network model is proposed to describe the interaction between information diffusion in online social networks and epidemic spreading in adaptive physical contact networks. Considering the difference in the connections between individuals, the heterogeneous rewiring rate, which is proportional to the strength of the connection, is introduced in our model. The simulation results show that the maximum infection scale decreases as the information acceptance probability grows, and the final infection decreases as the rewiring behaviors increase. Interestingly, an infection peak appears in our model due to the interaction between information diffusion and epidemic spread.

Keywords: co-evolving multiplex networks; epidemic spread; information diffusion; nonlinear differential systems

MSC: 65Q10

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1. Introduction

Information diffusion, i.e., positive information (e.g., authoritative information, news) and negative information (e.g., rumors and gossip) [1], is always accompanied by virus spreading on social networks or cascading failures on transportation networks, power grids, etc. To describe and analyze the coupling of information diffusion and virus spread/cascading failures, the study on multiplex networks becomes increasingly important as a result of the interactions among different real-world systems [2–5]. Both the difference in network structures of real-world systems and the interactions among different dynamic spread processes in real-world systems can be well described in the multiplex network model [6–10].

The impact of information diffusion on epidemic spread in multiplex networks has been widely studied in recent years [1,2]. The emergence and changing of information-related states enrich the whole epidemic spread process in social networks. First, the states of nodes have become more diversified since a single node owns two states at the same time; one is describing the physical state, and the other is describing the information-related state. And the increase in the node's states leads to the diversity of the propagation process. Furthermore, information diffusion leads to adaptive behavioral changes among individuals in response to epidemic outbreaks. When a public health event occurs on social networks, e.g., Coronavirus-2019 [11], people who had the awareness of prevention or received the relative information would actively take self-protective measures, such as wearing masks and washing their hands frequently. Then, a multiplex network with static network structures evolved into an adaptive multiplex network with a dynamic physical layer [12].

Although the interplay between information diffusion and biological infections has been extensively investigated within the framework of multiplex networks, there are still new challenges in the study of dynamic processes in multiplex networks. First, information diffusion affects not only the epidemic spread, but also the physical contact network structure. After accepting information, individuals are supposed to change their behavior to avoid infection; as a result, the physical network structure changes. Since the relationship between individuals is highly heterogeneous, individual behaviors are different in physical contact networks [13]. Moreover, most of the existing studies were focused on the impact of information diffusion on the epidemic spread, while epidemic spread also affects information diffusion, which is often ignored. As a matter of fact, information and epidemics are interacting and co-evolving. When the epidemic outbreaks, with the increasing number of patients, the epidemic itself receives more and more attention, and the relevant information spreads faster. That is to say, the probability of information diffusion changes as the virus spreads.

A weighted co-evolving multiplex network model with multiple time-varying parameters can be used to describe and analyze the challenges above. A two-layer multiplex network consisting of an information layer and a physical contact layer is introduced to describe the dynamic interaction between an online social (or communication) network and social contact network, where different dynamical processes can be supported. In online social networks, individuals exchange information related to disease, and a time-varying information acceptance rate is defined to describe the impact of the infection in social contact networks. While in a social physical network, actors also exchange biological elements that can carry on diseases. An aware and healthy individual can actively disconnect from infected neighbors and reconnect with healthy ones. The reconnecting rate between aware and unaware healthy nodes is time-varying due to the increase in aware ones. In addition, the heterogeneity of individual relationships in social contact networks cannot be ignored; therefore, both the infection rate and rewiring rate are closely related to the relationship, e.g., intimacy, social distance.

This paper presents a new mean-field model to describe the interaction between information dissemination and biological infections. Due to the new challenges, new nonlinear equations are necessary to extend the Susceptible–Infected–Susceptible (SIS) model and evaluate the impact of rewiring and weighted network links on the reliability of the adaptive weighted networks:

1. A novel weighted co-evolving multiplex network model is proposed to describe the interaction between information diffusion in online social networks and epidemic spreading in adaptive physical contact networks.
2. Two co-evolutionary processes have been considered in our model, the co-evolving of information diffusion and epidemic spreading between two layers and the co-evolving of epidemic spreading and network structure in the physical contact network.
3. Considering the difference in the connections between individuals, the heterogeneous rewiring rate, which is proportional to the strength of the connection, is introduced in our model.
4. Monte Carlo simulations in weighted co-evolving multiplex network models are carried out to describe and analyze the interaction between information diffusion and epidemic spreading.

The rest of this paper is organized as follows. In Section 2, the related works are reviewed. In Section 3, the structure of a weighted co-evolving multiplex network is described. Simulations have been conducted in different network structures to analyze and investigate the effect of network structure properties on propagation in weighted co-evolving multiplex networks in Section 4, followed by conclusions in Section 5.

2. Related Work

Many real-world systems are composed of multiple interacting subsystems, which can be described by multiplex networks, to provide an expressive model for modeling

real-world complex networks [14], such as multi-layer social networks with multiple social platforms interacting, and multi-layer transportation networks with multiple transportation channels cooperating and coupling. Furthermore, the interplay or co-evolution of dynamics between networks with different structures was simulated and analyzed by multiplex network models [1–7]. Granell et al. [10] pioneered the analysis of the interrelation between two processes accounting for the spreading of an epidemic, and the information awareness to prevent its infection, on top of multiplex networks. Soriano-Paños et al. [8] proposed a two-layer multiplex network to study the interplay between information spreading and opinion formation in social systems. Velásquez-Rojas et al. [9] studied the dynamics of the voter model for opinion formation intertwined with that of the contact process for disease spreading in multiplex networks and found that the opinion dynamics has striking consequences on the statistical properties of disease spreading. Xia et al. [15–17] proposed a new coupled disease spreading model on a two-layered multiplex network, where one layer denotes the underlying topology for the epidemics and the other one represents the corresponding topology for the awareness spread and extended the multiplex network model of awareness disease dynamics to a susceptible–infected–recovered (SIR) epidemic process that results in permanent immunity after infection.

In recent years, the interaction between epidemic spreading and related information diffusion on multiplex networks has received widespread attention, as it can help model, predict, and control the spread of an epidemic. Clara et al. [10] presented an analysis of the interrelation between two processes accounting for the spread of an epidemic, and the information awareness to prevent its infection, on top of multiplex networks. Zhou et al. [18] developed a set of nonlinear differential equations that have a linearly growing state–space size to describe the epidemic spreading process in multilayer complex networks, including the spreading of viruses and information in computer networks and the spreading of multiple pathogens in a host population. Wang et al. [1] proposed a novel epidemic model based on two-layered multiplex networks to explore the influence of positive and negative preventive information on epidemic propagation. Wu et al. [19] proposed an aware–susceptible–infected model (ASI) to explore the effect of information literacy on the spreading process in multiplex networks by using the microscopic Markov chain method.

So far, however, most of the existing research assumed that information diffusion does not change the social network structure. In fact, as the information diffuses, some individuals who have risk awareness often change their behavior to avoid being infected, which leads to changes in the network structure. In single-layer networks, there has been extensive research on the collaborative evolution of network structure and propagation. Gross et al. [20] first proposed an adaptive network wherein susceptible nodes are able to avoid contact with the infected by rewiring their network connections, and they found that the interplay between dynamics and topology can have important consequences for the spreading of infectious diseases and related applications. Subsequently, more research has been conducted on adaptive networks. Adaptive (weighted) networks have become increasingly important, as a result of the proliferation of cloud computing [21,22], vehicular ad hoc networks (VANETs) [23], and social networks [24].

At present, based on the literature we have searched, there is relatively little research on the information and epidemic spreading in multiplex adaptive networks. Peng et al. [12] first developed a highly integrated effective degree approach to modeling epidemic and awareness-spreading processes on multiplex networks coupled with awareness-dependent adaptive rewiring. They derived a formula for the threshold condition of contagion outbreak and provided a lower bound for the threshold parameter to indicate the effect of adaptive rewiring. In this paper, based on the influence of information dissemination on both epidemic transmission and physical contact network structure, the influence of structural and propagation dynamics of physical contact networks on information dissemination is further introduced into the weighted co-evolving multiplex network

model, based on the consideration of the influence of information dissemination on both epidemic transmission and physical contact network structure.

3. The Weighted Co-Evolving Multiplex Networks Model

Consider a two-layer network of N nodes connected by L_1 and L_2 links on each layer, respectively. The upper network describes the information diffusion network, and the lower network describes the individual contact network. Multiplex networks explicitly incorporate multiple channels of connectivity in a system, and they provide a natural description for systems in which entities have a different set of neighbors in each layer. Here, we use the two-layer network to describe and study the co-evolving of two different dynamical processes and the adaptive changing of the physical contact network structure.

3.1. Description of the Co-Evolving Processes in Multiplex Network

The coupling of multiplex networks brings rich co-evolutionary processes, as shown in Figure 1. The first co-evolution is the interaction between information diffusion and epidemic spreading. At the initial stage of epidemic spreading, the relevant information is very little and unconcerned. With the explosion of virus transmission, there is more and more information, from which people can obtain methods and strategies to address epidemics. In this process, the epidemic spreading process promotes information diffusion, which in turn can inhibit the epidemic spreading. The second co-evolution hidden inside the physical contact network is the network structure and the epidemic spreading, which is the so-called adaptive network in the single-layer network study. After accepting information, individuals who awaken the risk awareness change their own behavior to protect themselves, which means that the epidemic spread and network structure interact in the physical contact layer. We introduce the two co-evolution processes in detail, including the changing rules for the states of nodes and edges.

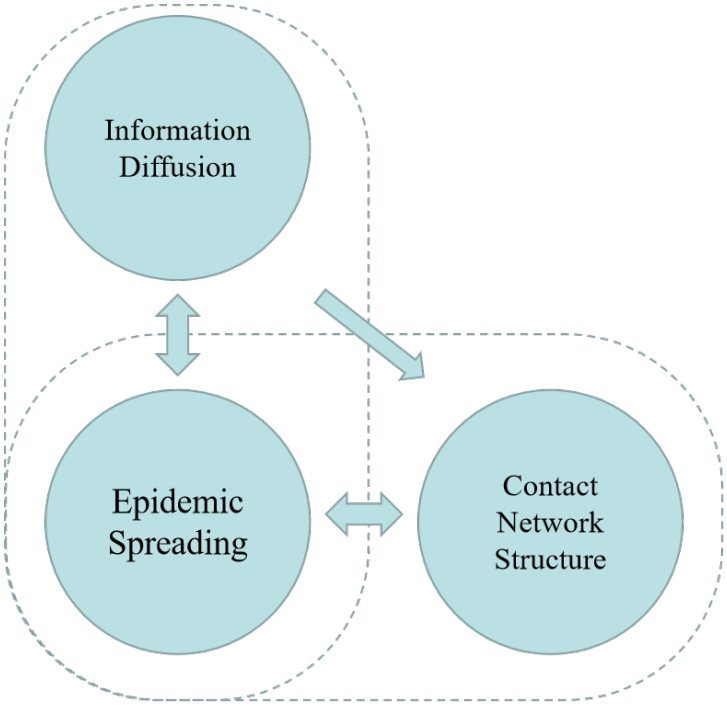


Figure 1. Interactions of inter-layer and inner-layer in multiplex network. Two co-evolutionary processes are shown in Figure 1: the first one is the interaction between information diffusion and epidemic spreading, and the second one hidden inside the physical contact network is the network structure and the epidemic spreading.

3.1.1. Co-Evolving of Information Diffusion and Epidemic Spreading between Two Layers

Consider a two-layer network of N nodes connected by L_1 and L_2 links on each layer, respectively. The upper information layer describes the online social (or communication) network, and the lower physical contact layer describes the social contact network, as shown in Figure 2. In the information diffusion layer, the Unaware–Aware (UA) model, where the node's state is unaware (U) or aware (A) of the existence of the epidemics and its prevention, is applied. In the UA model, U-state individuals do not have information about how to prevent infection, while A-state individuals reduce their risk to be infected. A U-state individual becomes aware with a probability α after communication with aware neighbors. Here, we assume that the A-state individual would remain aware of the infection due to the continuous spread of the epidemic.

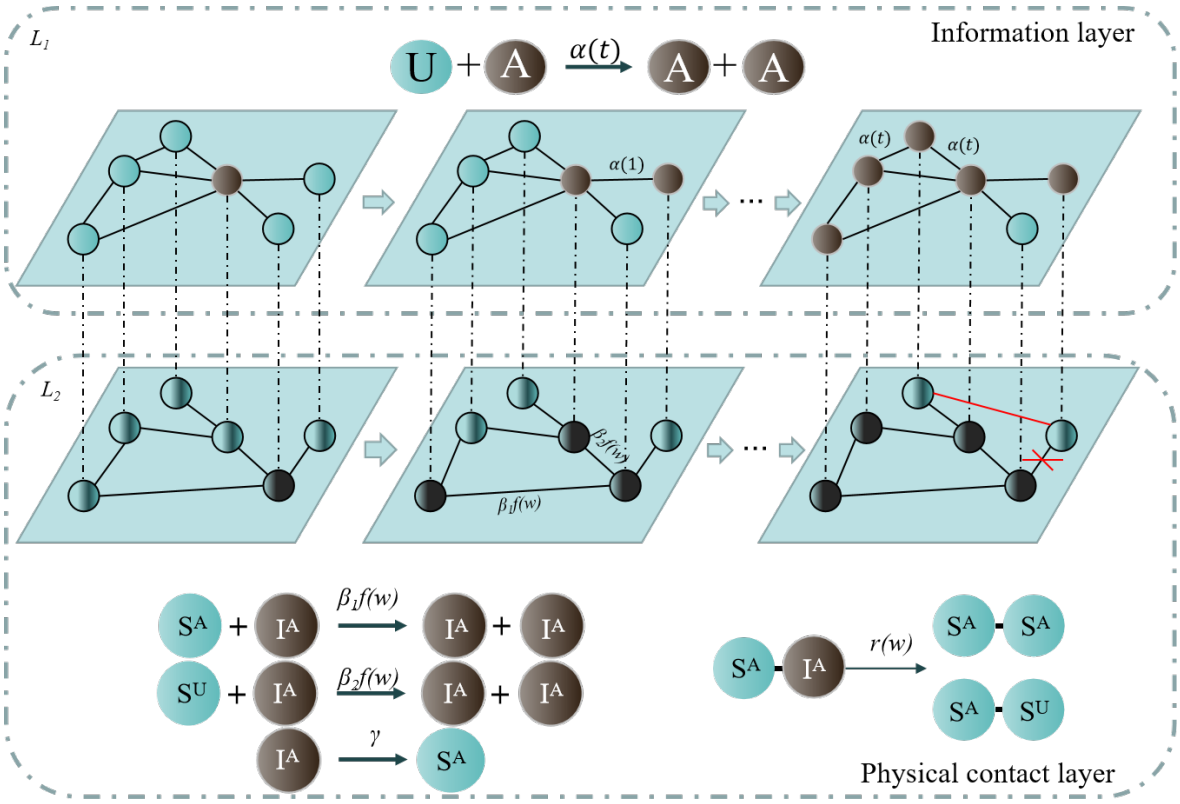


Figure 2. Schematic diagram of network state transition. The upper layer in the figure is the information layer (L_1), a U-state individual becomes aware with a probability $\alpha(t)$ after communication with an aware neighbor. The lower layer is the physical contact layer (L_2). In L_2, a healthy individual with risk awareness would disconnect the links with the infected person in a certain probability $r_w = r(w)$.

Different from the existing research, we take the impact of epidemic spreading on information diffusion into account. The probability of people acquiring information is not immutable, i.e., it is closely related to the spreading processes and the states of neighbors in the network. For example, when the scale of infected individuals becomes larger, people show stronger awareness and obtain information from more channels, which leads to faster information spreading. Therefore, we assume that a U-state individual becomes aware with a time-vary probability $\alpha(t)$, which is proportional to the infection density, i.e., $\alpha(t) \sim I(t)$.

3.1.2. Co-Evolving of Epidemic Spreading and Network Structure in the Physical Contact Network

Here, we use the SIS epidemic model in physical contact networks to simulate the epidemic spreading process [25–27]. There are three different states of node i: Susceptible with awareness (S^A), susceptible without awareness (S^U), and infected (I^A), who always

have awareness. An S^A-state node can be infected with probability $\beta_1 f(w)$ by an I^A-state neighbor, while the probability is $\beta_2 f(w)$ if it is a S^U-state node. $f(w)$ is a function positively related to the weight of the link, and $\beta_1 < \beta_2$. I^A-state ones return to S^A-state with probability γ. The change in node states is shown in Figure 2.

The awareness can not only decrease the probability to be infected, but also make individual behaviors change to isolated from infected ones. A healthy individual with risk awareness would disconnect their links with the infected person in a certain probability r_w. The higher the link weight, the harder it is to disconnect, i.e., $r_w \sim 1/w$. In order to ensure the functional completion of the network, we assume that a healthy person in the network who disconnected an edge has to find a healthy person to connect, as shown in Figure 2. For example, healthy employees will transfer work tasks from infected employees to healthy ones. In this way, a weighted co-evolving multiplex model is built, where information and virus propagation interact.

Figure 3 presents the operations of a node in a weighted co-evolving multiplex network. A healthy node without awareness can obtain information from its neighbor with awareness. A healthy node is more likely to be infected by an infected neighbor it interacts with frequently, i.e., the one with a larger link-weight, than by one it interacts with infrequently. Once one of its neighbors is infected, the node can observe the misbehaviors of the neighbor and rewire its link to bypass the infected neighbor, thereby preventing the propagation of the attacks or failures. As a result, the topology of the network keeps changing in response to the infection, quarantining infected individuals and counteracting the vulnerability explorations.

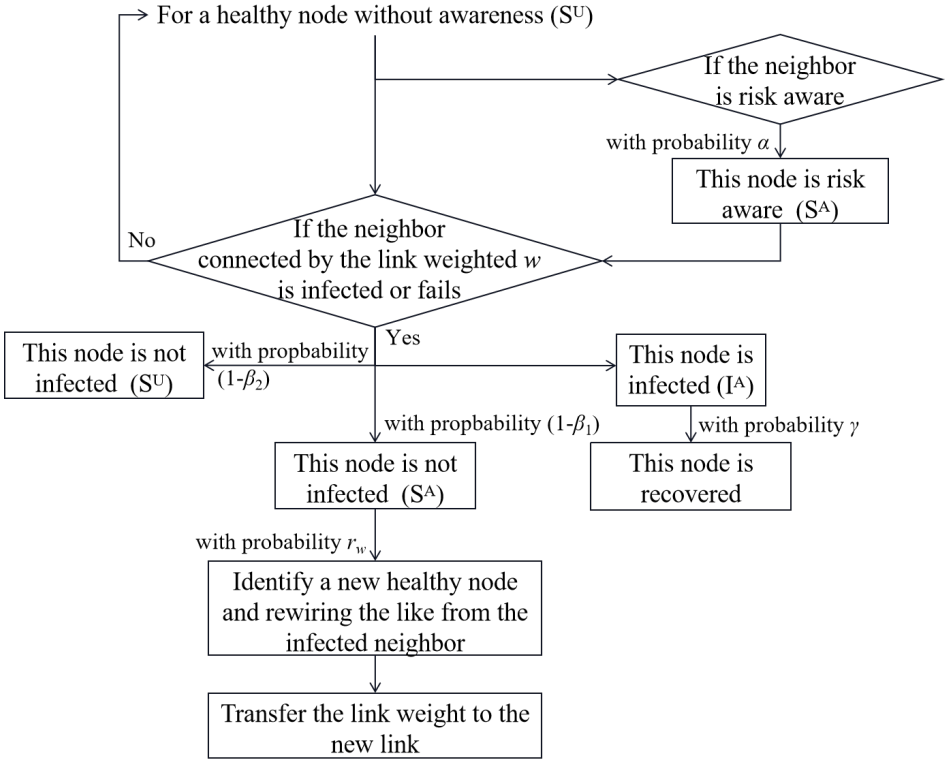


Figure 3. The flowchart of a node regarding a w-weighted link.

3.2. Mathematical Description of the Weighted Co-Evolving Multiplex Networks Model

Every node i has a certain probability of being in one of the three states at time t, denoted by $[S^A]$, $[S^U]$, and $[I^A]$, respectively. We provide the definition of the notations used in the model in Table 1, including node density, edge density in different states, and relevant parameters in the model.

Table 1. The notation used in the model formulation and analytical approximation.

Term	Definition
$[S^A]$	Fraction of aware susceptible nodes
$[S^U]$	Fraction of unaware susceptible nodes
$[I^A]$	Fraction of aware infected nodes
$[S^A I^A]_w$	Fraction of links between an aware susceptible node and an aware infected node with w-weight
$[S^U I^A]_w$	Fraction of links between an unaware susceptible node and an aware infected node with w-weight
$[S^A S^A]_w$	Fraction of links between two aware susceptible nodes with w-weight
$[S^U S^U]_w$	Fraction of links between two unaware susceptible nodes with w-weight
$[S^U S^A]_w$	Fraction of links between an unaware susceptible node and an aware susceptible node with w-weight
$[I^A I^A]_w$	Fraction of links between two aware infected nodes with w-weight
$\beta_1 f(w)$	Rate that an unaware susceptible node infected by an infected neighbor though a link with weight w
$\beta_2 f(w)$	Rate that an aware susceptible node infected by an infected neighbor though a link with weight w
α	The rate that an unaware node accepts the information and becomes aware
r_w	The rewiring rate that is proportional to the link weight w
br_w	The rate at which $[S^A I^A]_w$ link becomes $[S^A S^A]_w$ link due to the rewiring, and b is the scale parameter. Here, we set $b = \frac{[S^A]}{[S^A]+[S^U]}$. Then, the rate at which $S^A I^A$ link becomes $S^U S^A$ link due to the rewiring is $(1-b)r_w = \frac{[S^U]}{[S^A]+[S^U]} r_w$.

- Change the process of node state over time:

$$\frac{d[S^A]}{dt} = \gamma[I^A] - \sum_w \beta_1 f(w)[S^A I^A]_w + \alpha \sum_w ([S^U I^A]_w + [S^U S^A]_w) \tag{1}$$

$$\frac{d[S^U]}{dt} = -\sum_w \beta_2 f(w)[S^U I^A]_w - \alpha \sum_w ([S^U I^A]_w + [S^U S^A]_w) \tag{2}$$

$$\frac{d[I^A]}{dt} = -\gamma[I^A] + \sum_w \beta_1 f(w)[S^A I^A]_w + \sum_w \beta_2 f(w)[S^U I^A]_w \tag{3}$$

We call the system of Equations (1)–(3) the node-state changing model. On the right-hand side (RHS) of Equation (1), the first term accounts for the recovery of I^A-state node at rate γ. The second term indicates the infection process, where an S^A-state node is infected by an I^A-state neighbor through the w-weighted link at rate $\beta_1 f(w)$. The third term indicates the information transmission process, where the S^U-state individuals receive information from an A-state neighbor at rate α and change their state to S^A. Here, we assume that the probability of individuals accepting information and being aware of risks increases with the spread of infection $\alpha \sim p_1 I(t)$, where $p_1 \in [0,1]$ is an adjustment parameter and α is proportional to $I(t)$. On the RHS of Equation (2), the first term and second term are the infection process and information transmission process, respectively. A S^U-state node is infected by an I^A-state neighbor through the w-weighted link at rate $\beta_2 f(w)$, where $\beta_2 > \beta_1$ indicates that people without risk awareness are more likely to be infected. On the RHS of Equation (3), the first term is the recovery process and the second and third terms are the infection process.

- Change the process of link state over time:

$$\begin{aligned} \tfrac{d[S^A I^A]_w}{dt} &= -\beta_1 f(w)[S^A I^A]_w + \sum_{w'} f(w')(\beta_1 [S^A S^A I^A]_{ww'} + \beta_2 [S^A S^U I^A]_{ww'} \\ &- \beta_1 [I^A S^A I^A]_{w'w}) + 2\gamma[I^A I^A]_w - \gamma[S^A I^A]_w + \alpha[S^U I^A]_w - r_w[S^A I^A]_w \end{aligned} \tag{4}$$

$$\begin{aligned}\frac{d[S^U I^A]_w}{dt} &= \sum_{w'} f(w')(\beta_1[S^U S^A I^A]_{ww'} - \beta_2[I^A S^U I^A]_{w'w} + \beta_2[S^U S^U I^A]_{ww'}) \\ &- \beta_2 f(w)[S^U I^A]_w - \gamma[S^U I^A]_w - \alpha[S^U I^A]_w\end{aligned} \tag{5}$$

$$\frac{d[S^A S^A]_w}{dt} = -\beta_1 \sum_{w'} f(w')[S^A S^A I^A]_{ww'} + \gamma[S^A I^A]_w + \alpha[S^U S^A]_w + br_w[S^A I^A]_w \tag{6}$$

$$\frac{d[S^U S^U]_w}{dt} = -\beta_2 \sum_{w'} f(w')[S^U S^U I^A]_{ww'} \tag{7}$$

$$\begin{aligned}\frac{d[S^U S^A]_w}{dt} &= -\sum_{w'} f(w')(\beta_1[S^U S^A I^A]_{ww'} + \beta_2[I^A S^U S^A]_{w'w}) + \gamma[S^U I^A]_w \\ &-\alpha[S^U S^A]_w + (1-b)r_w[S^A I^A]_w\end{aligned} \tag{8}$$

$$\begin{aligned}\frac{d[I^A I^A]_w}{dt} &= \sum_{w'} f(w')(\beta_1[I^A S^A I^A]_{ww'} + \beta_2[I^A S^U I^A]_{ww'}) + \beta_1 f(w)[S^A I^A]_w \\ &+\beta_2 f(w)[S^U I^A]_w - 2\gamma[I^A I^A]_w\end{aligned} \tag{9}$$

Equations (4)–(9) characterize the time-varying numbers of links weighted by different weights and connecting nodes in different states. The reasons for the changes in the states of the links can be broadly divided into three, the infection and recovery process associated with the physical contact layer, the information diffusion process associated with the dissemination of the information layer, and the rewiring process of the physical contact layer. For example, Equation (4) captures the time-changing number of the w-weighted links connecting an aware susceptible node and an aware infected node. The first term on the RHS of Equation (4) results from the infection of the susceptible ends of the links with the probability of $\beta_1 f(w)$. The second term is the number of previous w-weighted $S^A S^A / S^A S^U$ links which become $S^A S^A$ links due to the infection at one end of the links through a w' weighted link with the probability of $\beta_1 f(w') / \beta_2 f(w')$. $[ABC]_{ww'}$ denotes the number of triplets $A - B - C$, with edge AB weighted w and edge BC weighted w', $A, B, C \in \{S^U, S^A, I^A\}$. The third and fourth term on the RHS of Equation (4) results from the recovery of the infected ends of the links with the probability of γ. The fifth term on the RHS of Equation (4) results from the information diffusion with the probability of α, and the last term results from rewiring to bypass an infected node with the probability of r_w.

Specifically, in the stable state, due to information dissemination, all nodes eventually become risk-aware, so there are only two types of state left in the network, namely $[S^A]$ and $[I^A]$. In addition, all nodes and links in different states achieve dynamic stability, i.e., $(\frac{d[S^A]}{dt}, \frac{d[I^A]}{dt}, \frac{d[S^A I^A]}{dt}, \frac{d[S^A S^A]}{dt}, \frac{d[I^A I^A]}{dt}) = (0, 0, 0, 0, 0)$. The Equation of state in the stable state satisfies

$$\gamma[I^A] - \sum_w \beta_1 f(w)[S^A I^A]_w = 0 \tag{10}$$

$$\zeta\beta_1 \frac{[S^A S^A]_w - [S^A I^A]_w}{S^A} \sum_{w'} f(w')[S^A I^A]_{w'} - (\beta_1 f(w) + \gamma + r_w)[S^A I^A]_w + 2\gamma[I^A I^A]_w = 0 \tag{11}$$

$$-\zeta\beta_1 \frac{[S^A S^A]_w}{S^A} \sum_{w'} f(w') + (\gamma + br_w)[S^A I^A]_w = 0 \tag{12}$$

$$\beta_1 f(w)[S^A I^A]_w - 2\gamma[I^A I^A]_w + \zeta\beta_1 \frac{[S^A I^A]_w}{S^A} \sum_{w'} f(w')[S^A I^A]_{w'} = 0 \tag{13}$$

Based on the approximation in [13], $[ABC]_{ww'} = \zeta \frac{[AB]_w [BC]_{w'}}{B}$, $A, B, C \in \{S^U, S^A, I^A\}$, $b = 1$.

An interesting result is shown in Equations (10)–(13). In a stable state, all nodes already have risk awareness, and we can see from the equations that the probability of information acceptance shows no impact on the final stable state of the network. That is, the result of our analysis is that the probability of information acceptance does not affect the final infection scale. Due to the isolation effect of the rewiring process on the infected nodes from susceptible ones, the rewiring rate shows an important impact on the final infection of the network.

We can see from the equations that information diffusion has changed the rules of epidemic spread and also changed the network structure. Conversely, the epidemic spreading affects the probability of information acceptance, i.e., the rules of information diffusion. Therefore, information diffusion, epidemic dynamics, and network structure interact with each other. In the next section, we conduct simulation experiments on the above processes through Monte Carlo methods to further explore the relationships among them.

4. Simulation Results

In this section, simulations are applied to analyze the propagation dynamics processes on the proposed multi-layer dynamic network model. Figures are plotted based on discrete-time Monte Carlo simulations of 100 iterations. Therefore, each data point in the figures is the average result of 100 independent runs. For each of the runs, a single infected node is randomly chosen at $t = 0$, as the initial point of infection.

Firstly, we constructed a two-layer network of size $N = 500$, where the information dissemination layer is a scale-free network [28], and the physical contact layer is a BBV-weighted scale-free network [29]. First, we establish a fully connected network with n initial nodes and assign each edge of the network the initial weight w_0. In our simulation, we set $n = 3$, $w_0 = 1$. In each time interval, add a new node with m edges, which are preferentially attached to existing nodes with a greater strength. Here, we set $m = 3$. The strength preference probability can be defined as $\prod_{new \to i} = \frac{s_i}{\sum_j s_j}$, where s_i represents the strength of node i, which can be expressed by $s_i = \sum_j w_{ij}$ with w_{ij} representing the weight of the edge between nodes i and j. When the new node j is linked to an existing node i, the weights of the edge between node i and its existed neighbors, such as node j, evolve as $w_{ij} \to w_{ij} + \Delta w_{ij}$, where $\Delta w_{ij} = \delta \frac{w_{ij}}{s_i}$ and δ is a constant. The average degree of both layers is $\langle k \rangle = 6$ and the average weight of the physical contact layer is $\langle w \rangle = 6$. The degree distribution, node strength distribution, and weight distribution of the BBV network all conform to the power-law distribution, and the degree and strength of the nodes have a positive correlation, as shown in Figure 4.

We aim to investigate the interplay between propagation processes on the multi-layer network. As described in the previous section, we use the UA model for information diffusion and the SIS model to describe the epidemic spread. The acceptance of positive epidemic-related information is closely related to the level of epidemic infection; here, the information acceptance probability of an unaware node from an aware neighbor is set as $\alpha(t) = p_1 I(t)$, $p_1 \in [0, 1]$. In the physical contact network, the rewiring rate is set to $r(w) = p_2 \frac{1}{w}$, $p_2 \in [0, 1]$. Both p_1 and p_2 are the adjustment parameters of $\alpha(t)$ and $r(w)$, respectively. And the larger p_1/p_2 is, the higher the information acceptance probability/rewiring rate. We explore the relationship between information dissemination and epidemic transmission processes through the changes in p_1 and p_2.

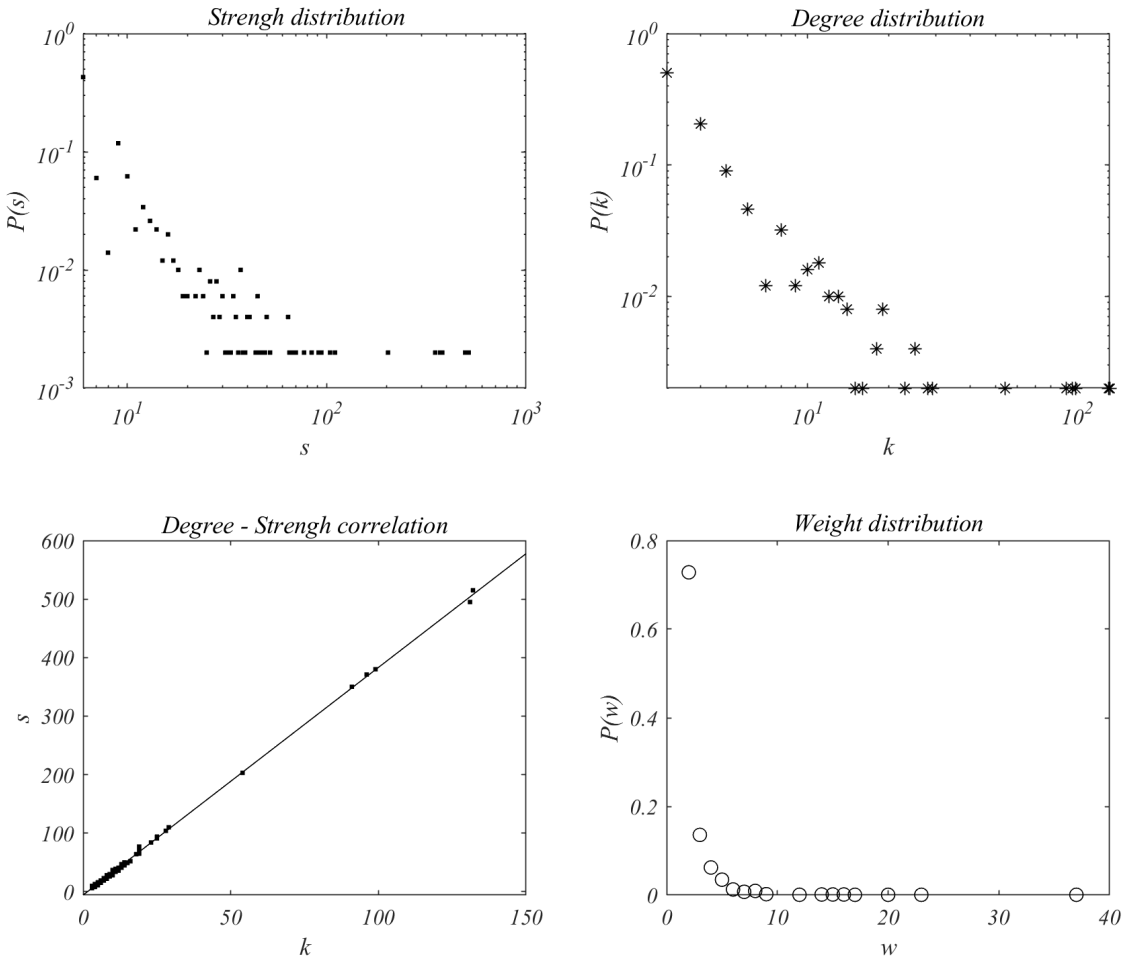


Figure 4. The distribution characteristics of the BBV network. The degree distribution, node strength distribution, and weight distribution of the BBV network all conform to the power-law distribution, and the degree and strength of the nodes have a positive correlation.

We first study the impact of information diffusion on the epidemic spread, as shown in Figure 5. The curves show the changes in the number of infected individuals $I(t)$ over time t under different p_1 in each subplot. We can see from each subplot that the maximum infection scale, i.e., peaks of the curves, decreases with the increase in p_1. When p_1 grows, the information acceptance probability becomes larger. Then, the number of S^A-state nodes that can be infected with a smaller probability increases, and infection velocity becomes slower. However, we can see from each of the subplots that the probability of information acceptance shows no impact on the final infection of the network, which is consistent with our theoretical analysis result in Section 3.

Interestingly, each curve in Figure 5 shows an infection peak due to the fact that all infected individuals are risk-aware, i.e., I^A-states, and the I^A-state nodes revert to the S^A-state with the recovery probability γ. Therefore, the S^A-state nodes in the network are increasing continuously, and the infection in the network is slowed down before reaching stability. When the recovering process disappears, i.e., $\gamma = 0$, the peak disappears, as shown in Figure 6 (the red curve).

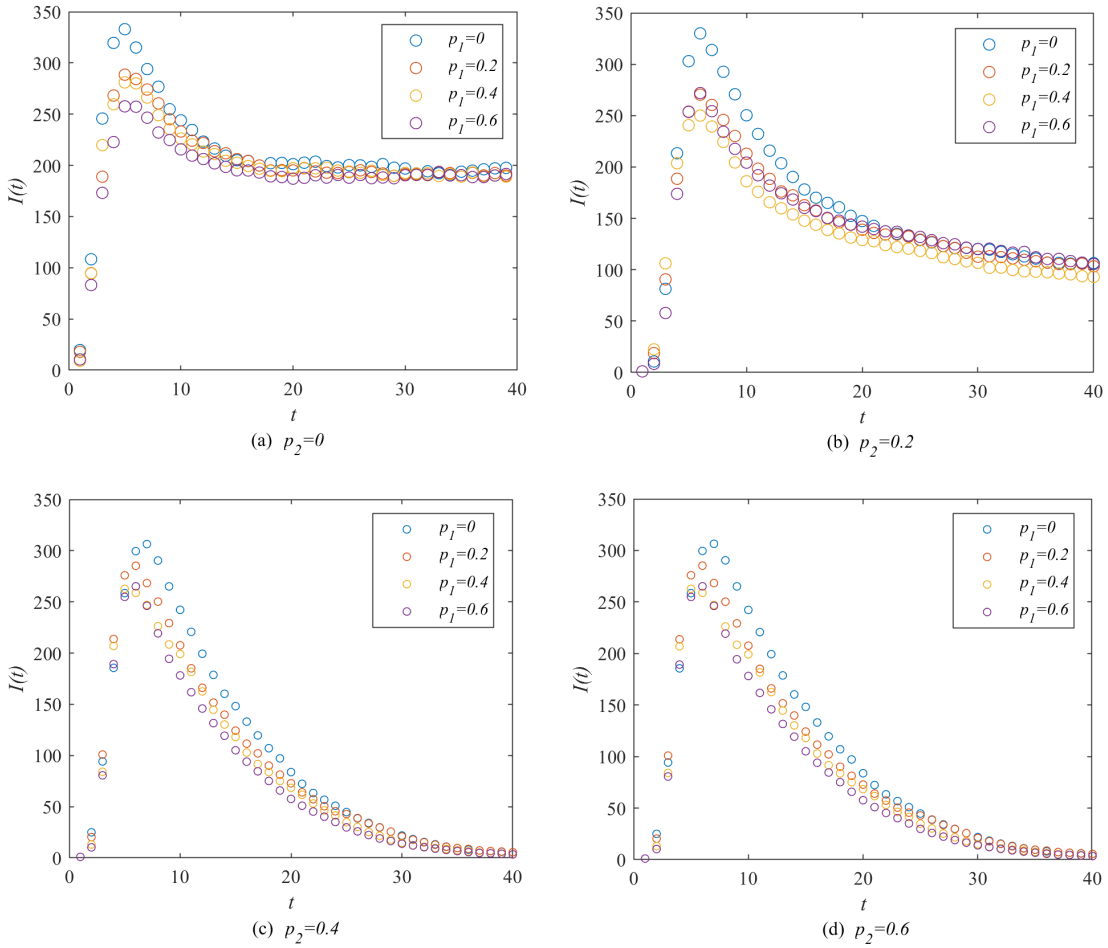


Figure 5. Theimpact of information diffusion on the epidemic spread under different parameter p_2 in rewiring rate r_w. Curves are the number of infected individuals $I(t)$ over time t as p_1 increases. In each subplot, we can see that the final infection ($I(t = 40)$) is almost the same, which means that the information diffusion has no impact on the final infection. But a high probability of receiving information can reduce the speed of epidemic spread.

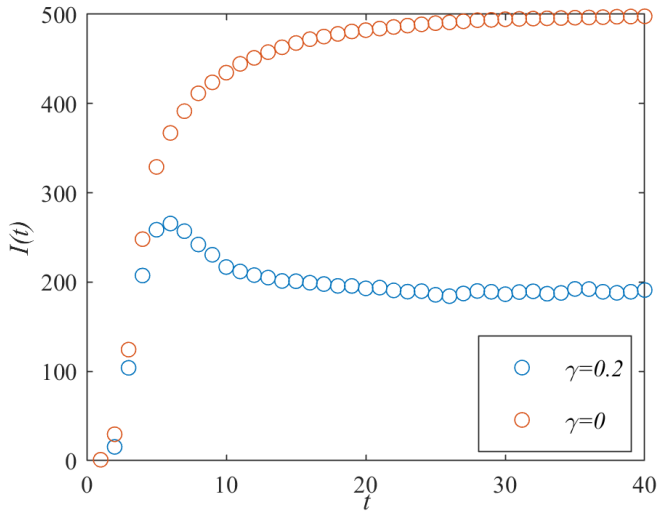


Figure 6. Comparison of infection processes with and without recovery process. Here, we set $p_1 = 0.8$, $p_2 = 0$.

We can also see from Figure 5 that, with the increase in the rewiring parameter p_2, the infection in the network has been greatly improved. We can see from Figure 5a–d that, as p_2 increases, the final infection ($I(t = 40)$) in the network significantly decreases. When $p_2 = 0$, the number of final infections is about 200; however, when $p_2 = 0.4/0.6$, the final infection disappears. Furthermore, we continue to study the impact of rewiring behaviors on the epidemic spread process. Figure 7 shows the impact of rewiring behaviors on the epidemic spread under different information acceptance parameters p_1. Curves are the number of infected individuals $I(t)$ over time t as p_2 increases. We can see from each subgraph that as p_2 increases, the scale of infections in the network decreases. The rewiring behavior has effectively inhibited the prevalence of the virus as it blocks the path of infection. Especially in the initial stage of infection, the higher the rewiring rate, the easier it is for the infection to eventually die out.

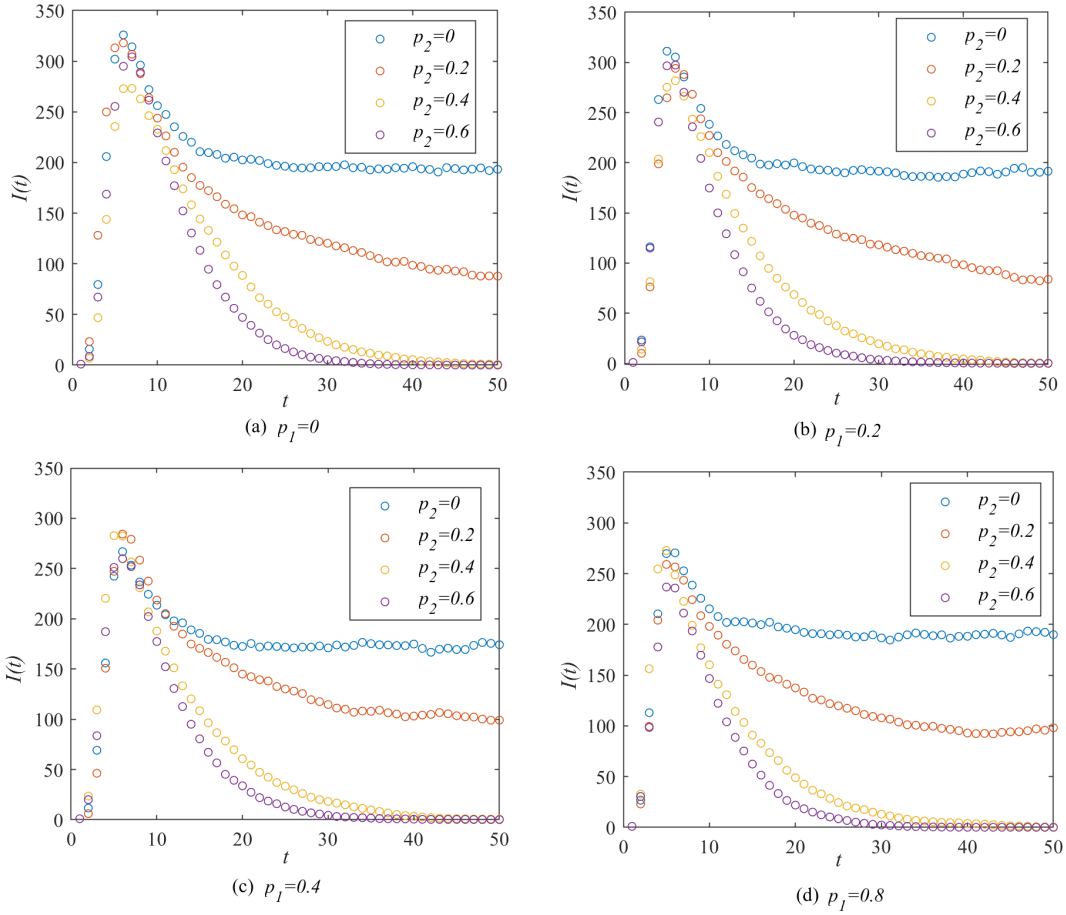


Figure 7. Theimpact of rewiring behaviors on the epidemic spread under different parameters p_1 in information acceptance rate. Curves are the number of infected individuals $I(t)$ over time t as p_2 increases. We can see from each subplot that the final infection was greatly inhibited with the increase in the rewiring parameter p_2.

Compared to the impact of information dissemination on the infection process, the rewiring behavior has a greater impact on the final infection scale, as shown in Figure 8. When we increase the rewiring parameter p_2, the final infection scale ($t = 100$) continuously decreases. As shown in Figure 8, when $p_2 > 0.32$, the final infection of the network approaches 0.

Another ongoing collaborative evolution is the structure of physical contact networks: both the degree distribution and weight distribution evolve along with the rewiring process. Adaptive rewiring of high-risk links leads to the breakdown of edges that connect a susceptible node with risk awareness and an infected one, and meanwhile, this gives

rise to the formation of low-risk links connecting toward a randomly chosen susceptible node. As demonstrated in Figure 9, the degree distribution of the physical contact network exhibits time-varying scaling behaviors: In the initial stage without rewiring (Case of $t = 0$ in Figure 8), both the node degree and link weights follow a perfect power law, while as the rewiring process unfolds, the degree/weight values become closer to the average degree/weight, approximating a Poisson distribution.

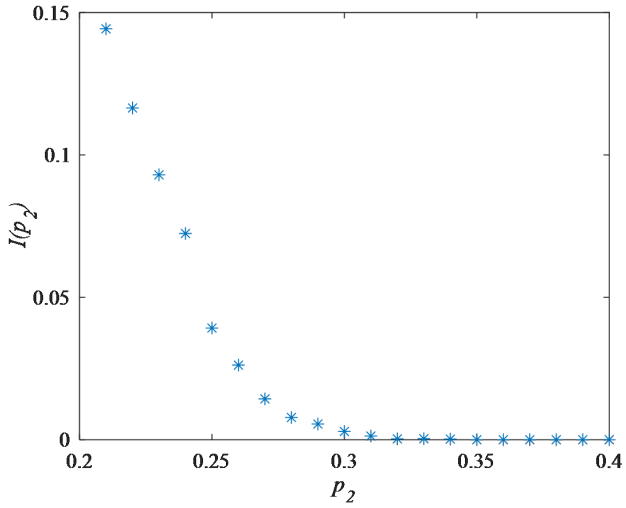
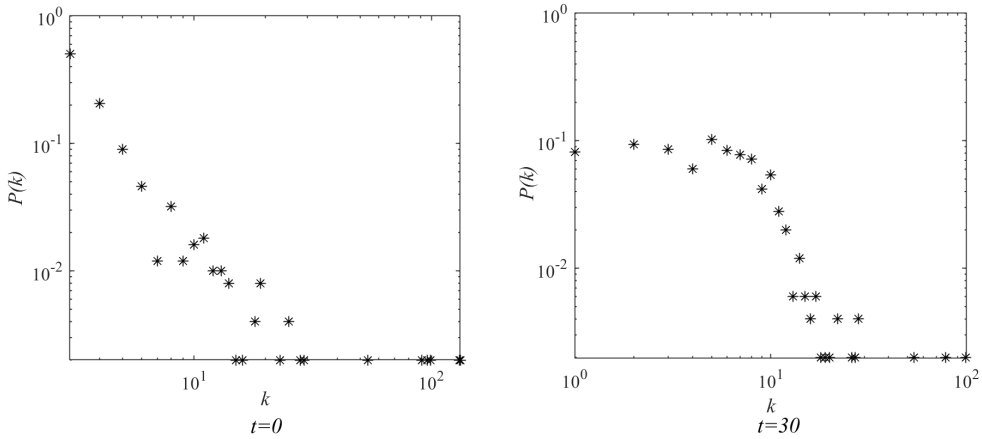
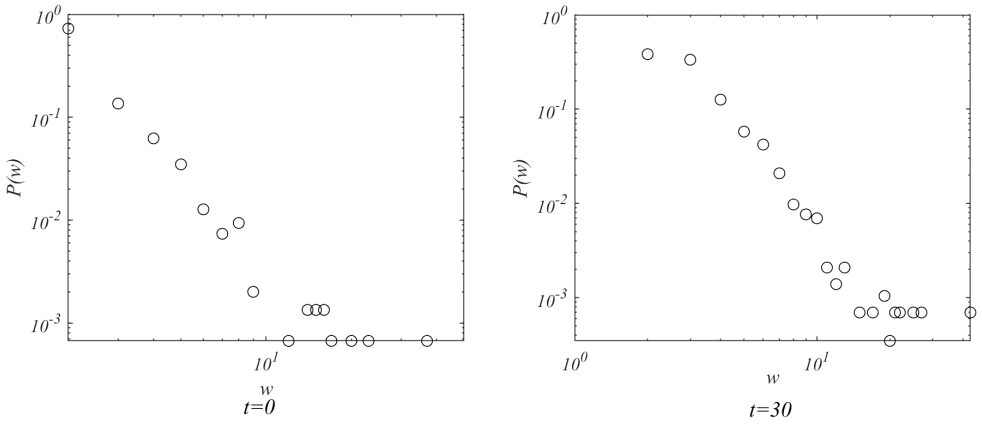


Figure 8. Thefinal infection $I(p_2)$ under different rewiring parameters p_2.



(a) The degree distribution of nodes at different time.



(b) The weight distribution of links at different time.

Figure 9. Theimpact of rewiring behaviors on network structure.

5. Conclusions

Information diffusion is an inevitable influencing factor in the process of epidemic spread, and in turn, epidemic spreading also affects information diffusion. This paper uses a two-layer network model to describe and analyze the interaction between information dissemination and epidemic transmission. Our model considers two co-evolutionary processes: the co-evolving of information diffusion and epidemic spreading between two layers and the co-evolving of epidemic spreading and network structure in the physical contact network. Considering the difference in the connections between individuals, the heterogeneous rewiring rate, which is proportional to the strength of the connection, is introduced in our model. Simulation results show that the epidemics spreading is closely related to the information diffusion and rewiring strategy. The maximum infection scale decreases as the information acceptance probability grows, and the final infection decreases as the rewiring behaviors increase. Interestingly, an infection peak appears in our model due to the interaction between information diffusion and epidemic spread.

The weighted co-evolving multiplex network model we propose is used to describe the dynamic interaction between information diffusion and epidemic spreading, which is more diverse in real life. Therefore, we hope to have a more realistic model based on our model to deepen the research on spreading dynamic interactions in real-world systems. In fact, besides social networks, a significant number of real-world systems, e.g., communication networks, transportation networks, and power networks, own multiplex network structures. For example, information diffusion can help drivers better understand road conditions and avoid traffic congestion in transportation networks. The information exchange in the communication network can help decision makers to appropriately load redistribution in a timely manner, thereby avoiding cascading failures in the power grid. The model we propose in this paper can be extended to more scenarios and will hopefully be used to study problems of multiplex network coexistence and cooperative evolution in these scenarios.

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Data Availability Statement: https://github.com/hm-harry/Epidemic-Spreading-on-Weighted-Co-evolving-Multiplex-Networks.

Conflicts of Interest: The authors declare no conflict of interest.

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