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The effect of information-driven resource allocation on the propagation of epidemic with incubation period

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Abstract

In the pandemic of COVID-19, there are exposed individuals who are infected but lack distinct clinical symptoms. In addition, the diffusion of related information drives aware individuals to spontaneously seek resources for protection. The special spreading characteristic and coevolution of different processes may induce unexpected spreading phenomena. Thus we construct a three-layered network framework to explore how information-driven resource allocation affects SEIS (Susceptible-Exposed-Infected-Susceptible) epidemic spreading. The analyses utilizing microscopic Markov chain approach reveal that the epidemic threshold depends on the topology structure of epidemic network, and the processes of information diffusion and resource allocation. Conducting extensive

Monte Carlo simulations, we find some crucial phenomena in the coevolution of information diffusion, resource allocation and epidemic spreading. Firstly, when E-state (exposed state, without symptoms) individuals are infectious, long incubation period results in more E-state individuals than I-state (infected state, with obvious symptoms) individuals. Besides, when E-state individuals have strong or weak infectious capacity, increasing incubation period have an opposite effect on epidemic propagation. Secondly, the short incubation period induces the first-order phase transition. But enhancing the efficacy of resources would convert the phase transition to a second-order type. Finally, comparing the coevolution in networks with different topologies, we find setting the epidemic layer as scale-free network can inhibit the spreading of the epidemic.

Keywords: Epidemic spreading, Exposed state, Multiplex network, Information-driven resource allocation, Microscopic Markov chain

1 Introduction

Infectious diseases have been major threats to human society for centuries [1, 2], for example, SARS, Ebola, and Dengue. How to effectively contain and suppress the spreading of infectious diseases has been an important and urgent issue in the management of public health. Currently, COVID-19 is circulating around the world, disrupting the order of human life [3, 4]. It has been found that there are some individuals who test positive for nucleic acid but are asymptomatic or have delayed symptom onset in COVID-19 [5]. Moreover, asymptomatic infections also have the infectivity as symptomatic infections [6], and asymptomatic carrier transmission has been an underestimated and complicated problem due to its concealment [7]. Consequently, it's crucial to investigate the role of asymptomatic infections in epidemic spreading, which will provide insights into the formulation of the disease control policy.

Due to the infeasibility to conduct the actual experiments, mathematical modeling and simulation analysis have become effective tools to explore the dynamical characteristics, such as incidence, outbreak size, and phase transition, of the epidemic spreading. Actually, there have been plenty of studies focusing on modeling the propagation of the epidemic [8, 9]. Several classical mathematical models of infectious diseases have been proposed since the first mathematical approach by Bernoulli in 1760 [10], such as the SIS (susceptible-infected-susceptible) model and SIR (susceptible-infected-recovered) model [11, 12]. Based on these classic models, researchers can predict the epidemic size, critical threshold, and associated critical phenomena. Besides, a large amount of improved models which capture the observed features like network topology [13, 14], the state of the diseases [15], spatiality [16], and temporality [17, 18] have been proposed.

However, epidemic spreading rarely occurs in isolation since the real networks are much more complex. For example, it's not difficult to find that

the propagations of epidemics, information, and behaviors are not independent of each other, but they tend to coevolve with strong interplay [19, 20]. Specifically, when one epidemic spreads in the physical contact network, the associated information simultaneously spreads through various types of communication platforms [21], like WeChat, Weibo, Facebook, and phone calls, etc. Further, the susceptible individuals who are aware of the epidemic would take certain measures (e.g., wearing a mask, staying at home, disinfecting hands frequently, taking some medicine and so forth) to avoid being infected by the diseases [22, 23], which will effectively suppress the outbreak of the epidemic [24]. Funk et al. [25] formulated an epidemiological model to analyze the effects of awareness on the epidemic propagation, discovering that in a well-mixed population, reducing the susceptibility of the informed individuals would result in a lower size of the outbreak, but does not affect the epidemic threshold. On the other hand, it's more realistic to use multiplex networks to characterize the coevolution spreading of the epidemic and information [26–28]. As people contact different neighbors in the physical contact network and the virtual communication network, Granell et al. [21] studied the interrelation between the spread of an epidemic and the information awareness in a multiplex network by using a microscopic Markov chain (MMC) approach [29]. They revealed that the epidemic threshold depends on the topological structure of the multiplex and is related to the awareness diffusion. There are also studies exploring the influence of diffusion characteristics [30, 31]. In [30], Guo et al. proposed a local awareness-controlled contagion spreading model in multiplex networks to investigate the epidemic spreading with awareness cascade. The results showed that there exists a two-stage impact of local awareness ratio on the epidemic threshold and the prevalence of the infectious diseases.

Besides, some recent researches indicate that resources play an important role in the containment of epidemic spreading [32–35]. On the one hand, according to reference [32], the shortage of resource caused by the prevalence of epidemic would trigger the explosion of the epidemic. Chen et al. [33] found that the recovery rate has a strong positive correlation with the average resource allocated to the infected individuals and insufficient resource leads to an abrupt increase in the infected population size. On the other hand, how to effectively use limited resources for controlling the epidemic spreading is of prime significance in diverse contexts as well, and many scholars in different fields have proposed plenty of optimal strategies of resource allocation [36–39]. For example, Chen et al. suggested that resources need to meet a significant amount, which is proportional to the whole population size to ensure epidemic containment in reference [33].

Generally, there are two main classifications of resources: public and individual. Public resources are provided and allocated by public sectors, like governments and departments of health and hygiene, while individual-based resources are shared among friends and family in small social circles. Recently, some researchers pointed out that most people actually rely on resources from healthy neighbors in the social contact network rather than public resources

[34]. To this end, the above social support should be more considered into the model. In individual-based resource allocation, healthy individuals are generally expected to reproduce and share resources to infected individuals [40]. It is assumed that the recovery of sick individuals depends on the availability of healing resources that are generated by the healthy population in [32]. Meanwhile, individuals who are aware of the epidemic tend to actively seek for the resource from their neighbors in contact networks, such as family members and friends, to protect themselves. This indicates that there exists correlation between information spreading and resource allocation, which is called as information-driven resource allocation. Furthermore, the resource possessed by individuals in turn contains the propagation of the epidemic. Wang et al. [41] investigated the complex coevolution dynamics of information diffusion, resource allocation and epidemic spreading and found the anomalous effect of information diffusion. But to our knowledge, there are no studies exploring the epidemic spreading with exposed state incorporating both information diffusion and resource allocation, since the dynamical evolutions among them are extremely sophisticated.

In this paper, we consider that once healthy individual is infected, he or she won't show obvious clinical symptoms immediately but firstly enter incubation period. In other words, infected individuals are divided into E-state (exposed state, without clinical symptoms) and I-state (infected state, with evident clinical symptoms). Thereinto, one parameter is designated to measure the incubation period. Then we formulate SEIS (Susceptible-Exposed-Infected-Susceptible) epidemic propagation model and articulate a three-layered network to explore the coevolution dynamics of epidemic propagation, information diffusion and resource allocation. Meanwhile, the therapeutic efficiency of resources and the individual's dependence on resources are considered. Based on MMC approach, the analytical expression of the epidemic size and epidemic threshold can be derived. Comparing MMC predictions and results obtained by Monte Carlo simulations, we find some crucial phenomena in the coevolution. Firstly, information diffusion notably inhibits the spread of diseases. Further, with the same information propagation probability, diseases more dependent on resources spread more widely. Secondly, the incubation period induces some interesting findings in coevolution. The results show that if E-state individuals have infectious ability, long incubation period or high self-healing ability would result in more E-state individuals than I-state individuals. Moreover, increasing incubation period would have an opposite effect on the propagation of the epidemic when E-state individuals have strong or weak infectious ability. Besides, when exploring epidemic size versus transmission probability, we find for long incubation period, epidemic size grows continuously with the increase of transmission probability. But for short incubation period, the growth of epidemic size exhibits a first-order phase transition with the emergence of hysteresis loop. However, if the efficacy of resources is enhanced, this first-order phase transition would turn into the second-order type. Lastly, comparing the coevolutionary processes in networks with different topologies, we

find that setting the epidemic layer as scale-free network would induce inhibition of the spreading dynamics. These findings would provide insights into controlling the epidemic with the incubation period, like COVID-19.

The remainder of this article is organized as follows. In section 2, we give the detailed description of the proposed model. In section 3, we analyze the model by employing MMC approach and derive the theoretical expression of the epidemic size and epidemic threshold. In section 4, extensive simulations are carried out to verify the accuracy of the MMC approach and explore how information-driven resource allocation affects epidemic spreading with exposed state. Finally, we conclude our work in 5.

2 SEIS Epidemic Model with Information-Driven Resource Allocation

Fig. 1 (a) Framework of proposed three-layered multiplex network, where UAU (unaware-aware-unaware) process takes place in the information layer ($\mathcal{A}$), resource allocation takes place in the resource layer ($\mathcal{B}$) and SEIS (susceptible-exposed-infected-susceptible) process takes place in the epidemic layer ($\mathcal{C}$). The orange arrows represent that S-state and E-state individuals allocate resources to aware individuals. The solid lines between two nodes represent the online (or offline) social relationships in the information (or resource and epidemic) layer. And the vertical dashed lines indicate the one-to-one correspondence among nodes on three-layered networks. (b) State evolution diagram of the epidemic propagation. $I(\beta)$ and $E(\alpha\beta)$ represent I-state individuals and E-state individuals infect susceptible individuals with probability β and $\alpha\beta$.

In our model, there exists an interplay among information diffusion, resource allocation and epidemic spreading. The emergence of the epidemic tends to stimulate the diffusion of related information. When individuals are aware of the epidemic, they will spontaneously seek resources for self-protection. In other words, information drives resource allocation. In the outbreak of an epidemic, the resources are always limited and the aware individuals are more likely to gain supports from their family members and friends (our work focuses on this type of resource allocation). Meanwhile, resources

are assumed to have the cure effect which can be used to recover the infected individuals. Therefore, the recovery rate of individuals who possess resources is enhanced, which in turn suppresses the outbreak of the epidemic.

To model this interplay among information diffusion, resource allocation and epidemic spreading, a three-layered multiplex network is employed. As is shown in Figure 1 (a), there are three layers where information diffusion, resource allocation and epidemic spreading occur in the $\mathcal{A}$, $\mathcal{B}$, and $\mathcal{C}$ layers, respectively. For simplicity, we focus on the case where the networks in all three layers are unweighted and undirected. Suppose there are N individuals in the system, and an individual is assumed to simultaneously appear in the three layers, i.e., there is a one to one correspondence among the nodes (individuals) in the three layers. In the information layer, the edges characterize the online social relationships, like friend relationships on Facebook or WeChat. In the epidemic layer, the edges represent the offline social relationships, such as with friends, family members and neighbors of the same community. Considering people tend to contact with different individuals online and offline, these two layers are with different connectivity, as is shown in Figure 1 (a). Consistent with the reality, resources are physically allocated via offline relationships, which indicates the topology of the resource and epidemic layer are identical.

To quantitatively characterize the dynamics of information diffusion, standard UAU (Unaware-Aware-Unaware) process [21] are employed. Specifically, there are two states of individuals in the information layer: 1) unaware; 2) aware of the epidemic. Aware individuals have information to initiatively seek resources from neighbors for self-protection, while unaware individuals don't take any actions. Furthermore, the individuals who are aware of the information would spread the information related to the epidemic to their unaware neighbors via information layer with a probability λ , which stimulates the diffusion of the information. Note that there are two incentives causing the individuals become aware of the epidemics: 1) Once individuals are infected and show clinical symptoms, they are aware of the existence of the epidemic immediately; 2) The unaware individuals get information from their aware neighbors. Considering the seasonality of the epidemic, there is a certain probability δ of an aware individual to forget the information or neglect the existence of the epidemic anymore so that he or she becomes unaware again.

In the epidemic layer, exposed state is introduced and we formulate the SEIS (Susceptible-Expose-Infected-Susceptible) epidemic propagation model. The individuals in the epidemic layer are classified into 3 compartments: 1)susceptible (S); 2) exposed (E); and infected (I). We designate a parameter $\gamma \in (0, 1]$ to represent the probability of one UE-state (unaware and exposed state) individual becoming I-state (infected state) individual. In other words, $\frac{1}{\gamma}$ represents the length of incubation period for UE-state individual, and a larger value of γ means the shorter incubation period of the disease. At the initial moment, most individuals are S-state (susceptible state) and few individuals are E-state (exposed state) or I-state (infected state), and the latter are

generally called seeds. The I-state and E-state individuals would infect their S-state neighbors with probability β and $\alpha\beta$, respectively. Thereinto, $\alpha \in [0, 1]$. When $\alpha = 1$, the infectious ability of the E-state individuals is the same as that of the I-state individuals. In our model, once the S-state individual is infected by its I-state or E-state neighbors, it turns into the exposed state firstly. Considering the coupling of information diffusion and epidemic propagation, at any time during the process, an individual in the multiplex network can be in one of five states: 1) aware and susceptible (AS); 2) unaware and susceptible (US); 3) aware and exposed (AE); 4) unaware and exposed (UE); 5) aware and infected (AI). Note that the unaware and infected (UI) state is impossible cause the individual is aware of the epidemic immediately once he or she shows clinical symptoms.

As is mentioned above, once individuals are informed the existence of the epidemic, they will positively seek resources from their neighbors out of self-protection. It's appropriately assumed that the resource is generated by individuals who appear to be healthy, i.e., S-state (AS or US) and E-state (AE or UE) individuals, and sick individuals, i.e., I-state individuals can't generate resource by themselves. Meanwhile, we consider that all aware individuals will actively seek resources from their S-state and E-state neighbors. For the sake of epidemic control, S-state and E-state individuals will generously distribute the generated resource equally to themselves for self-protection and each of their aware neighbors for cure. In other words, the resources of aware individuals are obtained from their S-state and E-state neighbors, and affect the recovery probabilities of the E-state and I-state individuals. To measure the impact of resources, especially the effect of medical resources on the recovery probabilities, we introduce the dependence of recovery on medical resources and the therapeutic efficacy of medical resources, which are presented by $\mu \in (0, 1]$ and $\varepsilon \in (0, 1]$, respectively. Thereinto, a larger value of μ means that the recovery of infected individuals is extremely dependent on medical resources, which means the weaker resilience to the disease of the individual. It's worth noting that the definition of μ in our work is different from the general one where μ represents the recovery probability. Besides, a larger value of ε indicates more efficient therapeutic of the medical resource, which is conducive to recovery of the disease.

To concisely describe how information-driven resource allocation affects epidemic spreading, we use discrete-time model for exploring coevolution of information-resource-epidemic dynamics. At the beginning of each time step t , S-state and E-state individuals will generate r units of resources. For simplicity, we set $r = 1$. Then resource allocation is performed in the resource layer based on the distribution principle. Aware individuals seek resources from neighbors and S-state and E-state individuals distribute one unit of resources averagely to each of their aware neighbors and themselves. After resource allocation at time t , assuming the resource amount possessed by on individual is $R_i(t)$. Next in the epidemic layer, the epidemic spreads among individuals. I-state and E-state individuals infect their S-state neighbors with probability β and $\alpha\beta$,

Table 1 Description of symbols

Symbol	Description
N	Number of individuals in network
$\langle k_1 \rangle$	Mean degree of the epidemic layer
$\langle k_2 \rangle$	Mean degree of the information layer
λ	Information transmission rate
δ	Information recovery rate
γ	The probability of UE-state individuals transforming into I-state individuals
β	Epidemic transmission rate of I-state individuals
$\alpha\beta$	Epidemic transmission rate of E-state individuals
μ	The dependence of recovery on resources
ε	The therapeutic efficacy of resources
$\mathbb{A}$	The adjacency matrix for the information layer
$\mathbb{B}$	The adjacency matrix for the epidemic and resource layer
$R_i^{AE}(t)$	The resource amount possessed by AE-state individual i at time t
$R_i^{AI}(t)$	The resource amount possessed by AI-state individual i at time t
$r_i(t)$	The probability that individual i is not informed by any neighbor at time t
$q_i(t)$	The probability that individual i is not infected by any neighbor at time t
$p_i^X(t)$	The probability that individual i is in state X
$\rho^X(t)$	The total proportion of X-state individuals at time t
ρ^X	The final proportion of X-state individuals

respectively. Once an S-state individual is infected, the state of this individual will become E-state at time $t + 1$. Furthermore, there are chances for AE-state and I-state individuals at time t to recover to be S-state at time $t + 1$ owing to medical resources. Specifically, for AE-state individual, he would recover with a probability $1 - \mu(1 - \varepsilon)^{R_i(t)}$, or he would become I-state with a probability $\gamma\mu(1 - \varepsilon)^{R_i(t)}$, or just remain to be E-state at time $t + 1$. And for AI-state individual, he would either recover with a probability $1 - \mu(1 - \varepsilon)^{R_i(t)}$ or just remain to be I-state at time $t + 1$. But for E-state individual which is unaware of the corresponding information, he could either turn into I-state with a probability γ or remain to be E-state at time $t + 1$. The above state transition of the epidemic propagation is shown in Figure 1 (b). To better suppress the outbreak of the epidemic, it's appropriately assumed that all their resources have been consumed completely, i.e., $R_i(t) = 0$ for each individual i at the end of each time step t . Finally in the information layer, UAU process occurs under the stimulation of epidemic spreading, which generates a new set of aware and unaware individuals. These new aware individuals will positively seek resources from neighbors at the next time step. In conclusion, there are three processes in total at each time step in our model, i.e., resource generation and allocation, epidemic spreading, information diffusion, orderly. The above three processes take place repeatedly until the system reaches the steady state.

3 Analytical Results Based on Microscopic Markov Chain Approach

In this section, microscopic Markov chain (MMC) approach [21] is utilized to analyze the coupled spreading dynamics in the three-layered networks. Specifically, the probabilities of network individuals in any state at any time can be calculated based on MMC approach. Through iterative calculations, we can predict the spreading size and critical transmission probability of the epidemic.

Let $\mathbb{A} = [a_{ij}]$ denotes the adjacency martrix for the information layer. If there is an online communication relationship between individual i and j , we have $a_{ij} = 1$ in the information layer; otherwise $a_{ij} = 0$. Likewise, let $\mathbb{B} = [b_{ij}]$ denotes the adjacency martrix for the resource and epidemic layer. If there exists a physical contact relationship between individual i and j , we have $b_{ij} = 1$ in the resource and epidemic layer; otherwise $b_{ij} = 0$. Based on the proposed model, each individual i has a certain probability of being in one of five states at time t , denoted by $p_i^{AS}(t)$, $p_i^{AE}(t)$, $p_i^{AI}(t)$, $p_i^{US}(t)$, and $p_i^{UE}(t)$ respectively, where $p_i^A(t) = p_i^{AS}(t) + p_i^{AE}(t) + p_i^{AI}(t)$, $p_i^S(t) = p_i^{US}(t) + p_i^{AS}(t)$ and $p_i^E(t) = p_i^{UE}(t) + p_i^{AE}(t)$. Descriptions of all symbols are listed in Table 1.

At the beginning of each time step t , each S-state or E-state individual generates one unit of resource and then the resource is averagely allocated to aware neighbors and themselves in the resource layer. After resource allocation has been finished, the resource amount possessed by AE-state and AI-state individuals at time t can be computed as:

$$R_i^{AE}(t) = \frac{1}{\sum_j b_{ij} p_j^A(t) + 1} + \sum_j b_{ij} [p_j^S(t) + p_j^E(t)] \frac{1}{\sum_{l \neq i} b_{jl} p_l^A(t) + 2}, \quad (1)$$

$$R_i^{AI}(t) = \sum_j b_{ij} [p_j^S(t) + p_j^E(t)] \frac{1}{\sum_{l \neq i} b_{jl} p_l^A(t) + 2}. \quad (2)$$

Thereinto, the resources of AE-state individuals are composed of two parts. As is shown in equation (1), the first term denotes the remaining amount after allocating one unit of resource to all aware neighbors, while the second term is the resources received from its S-state and E-state neighbors. The resources of AI-state individuals only come from its S-state and E-state neighbors, as is shown in equation (2).

Then epidemic propagation and information diffusion takes place in the epidemic layer and information layer respectively. For individual i , the probabilities of not being informed by any aware neighbors $r_i(t)$, and not being infected by any infected neighbors $q_i(t)$ are

$$r_i(t) = \prod_j [1 - a_{ij} p_j^A(t) \lambda], \quad (3)$$

$$q_i(t) = \prod_j \{1 - b_{ij}[p_j^E(t)\alpha\beta + p_j^{AI}(t)\beta]\}. \quad (4)$$

Thereinto, the term $a_{ij}p_j^A(t)\lambda$ on the right-hand side of equation (3) is the probability that individual i is informed by its aware neighbor j . Likewise, the term $b_{ij}[p_j^E(t)\alpha\beta + p_j^{AI}(t)\beta]$ on the right-hand side of equation (4) denotes the probability that individual i is infected by its E-state or I-state neighbor j .

Fig. 2 Transition probability trees for the states (a) AS, (b) US, (c) AE, (d) UE and (e) AI, of the SEIS-UAU coevolution dynamics in the multiplex at each time step.

The transition probability trees are employed to reveal the possible states of each individual i and their transitions, as is shown in Figure 2. Combining Eqs. (1) ~ (4), MMC equations are developed for the coupled process for each individual i as

$$\begin{aligned}
p_i^{AI}(t+1) &= p_i^{AE}(t)\gamma\mu(1-\varepsilon)^{R_i^{AE}(t)} + p_i^{UE}(t)\gamma + p_i^{AI}(t)\mu(1-\varepsilon)^{R_i^{AI}(t)}, \\
p_i^{AE}(t+1) &= p_i^{AS}(t)[1-q_i(t)](1-\delta) + p_i^{US}(t)[1-q_i(t)][1-r_i(t)] \\
&\quad + p_i^{AE}(t)(1-\gamma)\mu(1-\varepsilon)^{R_i^{AE}(t)}(1-\delta) + p_i^{UE}(t)(1-\gamma)[1-r_i(t)], \\
p_i^{UE}(t+1) &= p_i^{AS}(t)[1-q_i(t)]\delta + p_i^{US}(t)[1-q_i(t)]r_i(t) \\
&\quad + p_i^{UE}(t)(1-\gamma)r_i(t) + p_i^{AE}(t)(1-\gamma)\mu(1-\varepsilon)^{R_i^{AE}(t)}\delta, \\
p_i^{AS}(t+1) &= p_i^{AS}(t)q_i(t)(1-\delta) + p_i^{US}(t)q_i(t)[1-r_i(t)] \\
&\quad + p_i^{AE}(t)[1-\mu(1-\varepsilon)^{R_i^{AE}(t)}](1-\delta) \\
&\quad + p_i^{AI}(t)[1-\mu(1-\varepsilon)^{R_i^{AI}(t)}](1-\delta), \\
p_i^{US}(t+1) &= p_i^{AS}(t)q_i(t)\delta + p_i^{AE}(t)[1-\mu(1-\varepsilon)^{R_i^{AE}(t)}]\delta \\
&\quad + p_i^{US}(t)q_i(t)r_i(t) + p_i^{AI}(t)[1-\mu(1-\varepsilon)^{R_i^{AI}(t)}]\delta.
\end{aligned} \tag{5}$$

Therefore, the proportions $\rho^A(t)$, $\rho^E(t)$, $\rho^I(t)$ and $\rho^{total}(t)$ of susceptible, aware, E-state, I-state and total infected individuals at time t can be computed as

$$\begin{aligned}
\rho^S(t) &= \rho^{AS}(t) + \rho^{US}(t) = \frac{\sum_{i=1}^N [p_i^{AS}(t) + p_i^{US}(t)]}{N}, \\
\rho^A(t) &= \rho^{AS}(t) + \rho^{AI}(t) + \rho^{AE}(t) = \frac{\sum_{i=1}^N [p_i^{AS}(t) + p_i^{AE}(t) + p_i^{AI}(t)]}{N}, \\
\rho^I(t) &= \rho^{AI}(t) = \frac{\sum_{i=1}^N p_i^{AI}(t)}{N}, \\
\rho^E(t) &= \rho^{AE}(t) + \rho^{UE}(t) = \frac{\sum_{i=1}^N [p_i^{AE}(t) + p_i^{UE}(t)]}{N}, \\
\rho^{total}(t) &= \rho^I(t) + \rho^E(t) = \frac{\sum_{i=1}^N [p_i^{AE}(t) + p_i^{UE}(t) + p_i^{AI}(t)]}{N},
\end{aligned} \tag{6}$$

where N is the total number of individuals.

When there are enough iterations, final proportions of individuals in different states can be computed as a set of fixed points satisfying $p_i^{AI}(t+1) = p_i^{AI}(t) = p_i^{AI}$ and, similarly, for (AE), (AS), (US) and (UE). We use ρ^S , ρ^A , ρ^E , ρ^I and ρ^{total} to represent final proportions of susceptible, aware, E-state, I-state and total infected individuals, respectively.

Since S-state individuals firstly go to E-state after being infected by E-state or I-state individuals in our model, we choose ρ^E as crucial parameter to derive epidemic threshold. Near the critical transmission probability β_c , the final probability which individual i is in the exposed state satisfies the relation $p_i^E = \theta_i \ll 1$. Since all AI-state individuals are transformed from E-state, p_i^{AI} can be rewritten using p_i^{AE} and p_i^{UE} as

$$p_i^{AI} = \frac{\gamma\mu(1-\varepsilon)^{R_i^{AE}} p_i^{AE} + \gamma p_i^{UE}}{1 - \mu(1-\varepsilon)^{R_i^{AI}}}. \quad (7)$$

Neglecting high-order terms of θ_j , Eq.(4) can be approximated as

$$\begin{aligned} q_i &\approx 1 - \sum_j b_{ij} [p_j^E \alpha \beta + \frac{\gamma\mu(1-\varepsilon)^{R_i^{AE}} p_i^{AE} + \gamma p_i^{UE}}{1 - \mu(1-\varepsilon)^{R_i^{AI}}} \beta] \\ &= 1 - \sum_j b_{ij} \beta \eta_j, \end{aligned} \quad (8)$$

$$\text{where } \eta_j = p_j^E \alpha + \frac{\gamma\mu(1-\varepsilon)^{R_i^{AE}} p_i^{AE} + \gamma p_i^{UE}}{1 - \mu(1-\varepsilon)^{R_i^{AI}}}.$$

In the steady state, based on MMC equations, we can get

$$\begin{aligned} p_i^E &= p_i^{AS}(1 - q_i) + p_i^{US}(1 - q_i) + p_i^{UE}(1 - \gamma) + p_i^{AE}(1 - \gamma)\mu(1 - \varepsilon)^{R_i^{AE}} \\ &= (p_i^A - p_i^{AE} - p_i^{AI})(1 - q_i) + (p_i^U - p_i^{UE})(1 - q_i) \\ &\quad + (1 - \gamma)[p_i^{UE} + p_i^{AE}\mu(1 - \varepsilon)^{R_i^{AE}}]. \end{aligned} \quad (9)$$

Neglecting high-order terms of θ_i , p_i^E can be simplified as

$$\begin{aligned} p_i^E &\approx (p_i^A + p_i^U) \sum_j b_{ij} \beta \eta_j + (1 - \gamma)[p_i^{UE} + p_i^{AE}\mu(1 - \varepsilon)^{R_i^{AE}}] \\ &= \sum_j b_{ij} \beta \eta_j + (1 - \gamma)[p_i^{UE} + p_i^{AE}\mu(1 - \varepsilon)^{R_i^{AE}}]. \end{aligned} \quad (10)$$

Since the networks of epidemic and information layers possess homogeneous structure, i.e., random-regular networks in our work, the probability that individual i is in the aware (or susceptible) state satisfies $p_i^A \approx \rho^A$ (or $p_i^S \approx \rho^S$) in the steady state. The resource amount R_i^{AE} and R_i^{AI} of AE-state and AI-state individual i can be simplified as

$$R_i^{AE} \approx R^{AE} = \frac{1}{\langle k_1 \rangle \rho^A + 1} + \langle k_1 \rangle (\rho^S + \rho^E) \frac{1}{(\langle k_1 \rangle - 1) \rho^A + 2}, \quad (11)$$

$$R_i^{AI} \approx R^{AI} = \langle k_1 \rangle (\rho^S + \rho^E) \frac{1}{(\langle k_1 \rangle - 1) \rho^A + 2}, \quad (12)$$

where $\langle k_1 \rangle$ is the mean degree of the epidemic layer. Near the critical transmission probability β_c , there are extremely few E-state individuals in the system. Then we can assume the resources are generated mainly by S-state individuals. R_i^{AE} and R_i^{AI} can be further simplified as

$$R_i^{AE} \approx R^{AE} = \frac{1}{\langle k_1 \rangle \rho^A + 1} + \langle k_1 \rangle \rho^S \frac{1}{(\langle k_1 \rangle - 1) \rho^A + 2}, \quad (13)$$

$$R_i^{AI} \approx R^{AI} = \langle k_1 \rangle \rho^S \frac{1}{(\langle k_1 \rangle - 1) \rho^A + 2}. \quad (14)$$

It's appropriately assumed that $p_i^{UE} \approx (1 - \rho^A) p_i^E$ and $p_i^{AE} \approx \rho^A p_i^E$. Inserting these expressions and $p_i^E = \theta_i$ into Eq.(10), we can get

$$\begin{aligned} \theta_i = \sum_j b_{ij} \beta [\theta_j \alpha + \frac{\gamma \mu (1 - \varepsilon)^{R^{AE}} \rho^A \theta_j + \gamma (1 - \rho^A) \theta_j}{1 - \mu (1 - \varepsilon)^{R^{AI}}}] \\ + (1 - \gamma) [(1 - \rho^A) \theta_i + \mu (1 - \varepsilon)^{R^{AE}} \rho^A \theta_i]. \end{aligned} \quad (15)$$

Let $Q_1 = 1 - (1 - \gamma) [(1 - \rho^A) + \mu (1 - \varepsilon)^{R^{AE}} \rho^A]$ and $Q_2 = \alpha + \frac{\gamma \mu (1 - \varepsilon)^{R^{AE}} \rho^A + \gamma (1 - \rho^A)}{1 - \mu (1 - \varepsilon)^{R^{AI}}}$. Eq.(15) can be rewritten as

$$\sum_j (Q_2 \beta b_{ij} - Q_1 \delta_{ij}) \theta_j = 0, \quad (16)$$

where δ_{ij} is the element of the identity matrix $\mathbb{E}$. Together with all individual i , we obtain

$$(\mathbb{B} - \frac{Q_1}{\beta Q_2} \mathbb{E}) \theta = 0, \quad (17)$$

where $\theta = (\theta_1, \theta_2, \dots, \theta_N)^T$. The nontrivial solution of Eq.(17) is eigenvectors of $\mathbb{B}$, whose real eigenvalues are equal to $\frac{Q_1}{\beta Q_2}$. The critical transmission probability β_c is the minimum value β for the onset of the epidemic, which can be given by the largest eigenvalue of adjacency matrix $\mathbb{B}$. That is

$$\beta_c = \frac{Q_1}{Q_2 \Lambda_{max}(\mathbb{B})}, \quad (18)$$

where $\Lambda_{max}(\mathbb{B})$ is the largest eigenvalue of adjacency matrix $\mathbb{B}$. Since the epidemic layer possesses homogeneous network structure, the largest eigenvalue of adjacency matrix $\mathbb{B}$ is close to the mean degree of the epidemic network, i.e., $\Lambda_{max}(\mathbb{B}) \approx \langle k_1 \rangle$. It's worth noting that the theoretical expression of β_c in Eq.(18) is based on the assumption that the network structures of epidemic and information layers are homogeneous. From Eq.(18), it can be found that critical transmission probability β_c is related to the dynamics of information diffusion (i.e., ρ^A) and resource allocation (i.e., ε, μ, R^{AE} and R^{AI}).

We verify the accuracy of Eq.(18) in Figure 3 where the asterisks on X-axis mark the corresponding values of critical transmission probability β_C . It can be clearly found that theoretical thresholds accurately predict the onset of the epidemic. When $\beta < \beta_c$, we can see $\rho^E \approx 0$. But for $\beta > \beta_c$, we can see $\rho^E > 0$, which indicates the spreading of the epidemic in the system.

Fig. 3 Epidemic threshold predicted by MMC theory for different ε . Colored curves represent ρ^E versus β and the asterisks on X-axis mark the critical transmission probability β_c . Initial density of AI-state individuals is set to be 0.01. To solve β_c , initial transmission probability β is set to be 0.01. The specific parameters for the simulations are $\alpha = 0.5$, $\delta = 1$, $\lambda = 0.2$, $\mu = 0.5$, $\gamma = 0.2$. Information and epidemic networks have different random-regular (RR) topology, each of size $N = 1000$ and average degree $\langle k_1 \rangle = \langle k_2 \rangle = 10$.

4 Numerical and Simulations Results

In this section, we carry out extensive numerical simulations to study how information-driven resource allocation affects epidemic spreading with exposed state. Specifically, the experimental results are obtained by Monte Carlo simulations and MMC theory. We employ two different networks to describe the epidemic layer and information layer, respectively. Based on the assumption in Section 2, the epidemic and resource layers have the same network topology. Thereinto, there are 1000 nodes (individuals) of each network and mean degree of the epidemic layer $\langle k_1 \rangle$ and information layer $\langle k_2 \rangle$ satisfy $\langle k_1 \rangle = \langle k_2 \rangle = 10$. As is mentioned earlier, the individuals on the three networks have one-to-one correspondence.

Firstly, we analyze time evolutions of epidemic spreading for different combinations of α , γ , μ in Figure 4. Specifically, the densities ρ^E , ρ^I and ρ^{total} of E-state, I-state and total infected individuals are utilized to analyze the spread size of the epidemic. For each experiment, time evolution step T is set to be 30, and it can be found that spread size has already reached a stationary value during T . To reduce the statistical fluctuations to more accurately predict

Fig. 4 Time evolutions of the spread size of the epidemic for (a) $\alpha = 0.2, \gamma = 1, \mu = 0.9$; (b) $\alpha = 0.8, \gamma = 1, \mu = 0.9$; (c) $\alpha = 0.8, \gamma = 0.5, \mu = 0.9$; (d) $\alpha = 0.8, \gamma = 1, \mu = 0.5$. The spread size is characterized by the densities of I-state individuals, E-state individuals and total infected individuals, denoted by ρ^I , ρ^E and ρ^{total} , respectively. At the initial stage, $\rho^{AI}(0)$ and $\rho^{AE}(0)$ are set to be 0.01, and the remaining nodes are in US state. The other parameters for the simulations are $\lambda = 0.2, \delta = 1, \beta = 0.4, \varepsilon = 0.5$. The value of time evolution steps is set to be $T = 30$. Each symbol is obtained by averaging 500 independent Monte Carlo realizations, and colored curves are calculated from MMC theory. Information and epidemic networks have different random-regular (RR) topology, each of size $N = 1000$ and average degree $\langle k_1 \rangle = \langle k_2 \rangle = 10$.

the spread size of the epidemic, all Monte Carlo simulation results are obtained by averaging 500 independent runs.

In Figure 4, it can be found Monte Carlo simulations (symbols) match well with MMC based numerical iterations (colored curves), which validates the accuracy of MMC theory. As is shown in Figure 4 (a), when the incubation period is relatively short (e.g., $\gamma = 1$) and the recovery of the E-state and I-state individuals significantly depends on the resources ($\mu = 0.9$), there are more I-state individuals than E-state individuals in the stationary state. This is mainly because E-state individuals will transform into I-state individuals quickly due to the short incubation period, but I-state and E-state individuals

Fig. 5 Effect of information transmission probability λ on the final epidemic size with different system parameters. (a) The final epidemic size ρ_{total} versus λ for three different values of μ for $\gamma = 1$, $\alpha = 0.5$. With three different values of γ , the final epidemic size ρ_{total} versus λ for (b) $\alpha = 0$, $\mu = 0.5$ (c) $\alpha = 1$, $\mu = 0.5$. For (a)-(c), each symbol is obtained by Monte Carlo simulations, and colored curves are calculated from MMC theory. (d) Color-coded value of ρ_{total} in the parameter plane (α, γ) by performing MMC iterations for $\mu = 0.5$. For these simulations, the initial values of ρ^{AI} are set to be 0.01. The other parameters for the simulations are $\delta = 1$, $\beta = 0.4$, $\varepsilon = 0.5$. Information and epidemic networks have different random-regular (RR) topology, each of size $N = 1000$ and average degree $\langle k_1 \rangle = \langle k_2 \rangle = 10$.

depend on resources so seriously that induces them difficult to recover within a short period. In other words, the rapid transition of E-state individuals and the infeasibility of recovery result in the accumulation of I-state individuals. Even if E-state individuals have strong infectious ability ($\alpha = 0.8$), the results remain consistent, as is shown in Figure 4 (b). However, if the incubation period becomes longer ($\gamma = 0.5$ in Figure 4 (c)) or self-healing ability of infected individuals is enhanced ($\mu = 0.5$ in Figure 4 (d)), there will be exactly the opposite result, i.e., more E-state individuals than I-state individuals in system. The qualitative understandings of phenomena in Figure 4 (c) and Figure 4 (d) are as follows. For the former case, E-state individuals which

have long incubation period will gradually transform into I-state after a long period of accumulation. For the latter case, AE-state and AI-state individuals with high recovery capacity will directly transform into S-state individuals by using resources, and just few E-state individuals transform into I-state individuals. To this end, the above results suggest that as for those epidemics with a long incubation period or less dependent on resources in recovering, we not only need to care about I-state individuals, E-state individuals that are possible to induce the second outbreak of the epidemic are equally critical.

Then we explore how the spreading probability λ of information affects final epidemic size ρ^{total} for different combinations of α , γ , μ . As is shown in Figures 5 (a) $\sim$ (c), the final epidemic size ρ^{total} decreases continuously as the value of λ increases. This overall descending trend shows that the diffusion of the related information would be helpful to suppress the propagation of the epidemic, which is consistent with our common sense. Figure 5 (a) shows that epidemic size ρ^{total} decreases in sequence in the case of $\mu = 1$, $\mu = 0.6$, $\mu = 0.2$, demonstrating that diseases excessively dependent on resources are more harmful to human society.

In Figures 5 (b) $\sim$ (d), we find combinations of infection ability (α) and incubation period ($\frac{1}{\gamma}$) of E-state individuals have a dual effect on the spreading of the epidemic. As is shown in Figure 5 (b), if E-state individuals have no infectious capacity, i.e., $\alpha = 0$, final epidemic size ρ^{total} decreases with the increase of the incubation period. However, if E-state individuals have strong infectious capacity, like $\alpha = 1$, the longer incubation period will induce more infected individuals in the system, as is shown in Figure 5 (c). To further investigate this dual effect, we analyze the epidemic size by theoretically predicting final epidemic size in the parameter plane (α, γ), as shown by the color-coded value of ρ^{total} in Figure 5 (d). It can be observed that for small value of α , ρ^{total} decreases as $\frac{1}{\gamma}$ increases, but for large value of α , ρ^{total} increases as $\frac{1}{\gamma}$ increases, which apparently exhibits a dual effect as well. The qualitative understanding of such dual effect is that when the incubation period is long, E-state individuals without infectious ability would neither easily transform into I-state individuals nor infect other susceptible individuals, but infectious E-state individuals will infect more S-state individuals during long incubation period, resulting in more infected individuals in the system. The dual effect suggests us to pay more attention to diseases with long and infective incubation period.

When exploring how infection probability β affects epidemic spreading, our theory predicts that variations of incubation period can induce a remarkable change in the phase transition in Figure 6. In epidemiology, the spreading size will remain zero and an outbreak will not arise until the infection probability β exceeds the critical threshold β_c , which leads to a phase transition. If spreading size abruptly increases from zero to a positive large value at β_c , the phase transition is of the first-order type. In contrast, if spreading size continuously increases as β passes through β_c , the phase transition is of the second-order type.

Fig. 6 Effect of incubation period $\frac{1}{\gamma}$ on phase transition. Shown are the final epidemic size ρ^{total} versus the transmission probability β for (a) $\alpha = 0.5$ and (b) $\alpha = 0.8$. Thereinto, each symbol is obtained by Monte Carlo simulations, and the meanings of specific symbols are shown in legend. The solid and dotted curves represent the MMC prediction results when initial densities of AI-state individuals are 0.01 and 0.95, respectively. Color-coded value of ρ^{total} in the parameter plane (γ, β) by performing MMC iterations for (c) $\alpha = 0.5$ and (d) $\alpha = 0.8$. At the initial stage, the values of ρ^{AI} are set to be 0.01. The other parameters for the simulations are $\delta = 1$, $\lambda = 0.2$, $\varepsilon = 0.2$, $\mu = 1$. Information and epidemic networks have different random-regular (RR) topology, each of size $N = 1000$ and average degree $\langle k_1 \rangle = \langle k_2 \rangle = 10$.

Figures 6 (a) and (b) show final epidemic size ρ^{total} versus infection probability β with two different values of γ for $\alpha = 0.5$ and $\alpha = 0.8$ respectively. Thereinto, we use solid and dashed curves to represent ρ^{total} obtained by MMC theory versus β when $\rho^{AI}(0) = 0.01$ and 0.95 respectively. We find that, in both Figures 6 (a) and (b), for $\gamma = 1$, which indicates short incubation period, the growth of ρ^{total} exhibits a first-order phase transition as β exceeds β_c . Meanwhile, it can be seen that black ($\gamma = 1$) solid curve and dashed curve don't overlap each other completely, signifying the emergence of hysteresis loop. But blue ($\gamma = 0.2$) solid curve overlaps with dashed curve perfectly and ρ^{total} shows a continuous increase. Thus for $\gamma = 0.2$, corresponding to long

incubation period, the hysteresis loop vanishes and the phase transition is of the second-order type.

Fig. 7 Effect of efficacy of resources ϵ on phase transition. Shown are the final epidemic size ρ^{total} versus the transmission probability β for (a) $\alpha = 0.5$ and (b) $\alpha = 0.8$. Thereinto, each symbol is obtained by Monte Carlo simulations, and the meanings of specific symbols are shown in legend. The solid and dotted curves represent the MMC prediction results when initial densities of AI-state individuals are 0.01 and 0.95, respectively. Color-coded value of ρ^{total} in the parameter plane (ϵ, β) by performing MMC iterations for (c) $\alpha = 0.5$ and (d) $\alpha = 0.8$. At the initial stage, the values of ρ^{AI} are set to be 0.01. The other parameters for the simulations are $\delta = 1$, $\lambda = 0.2$, $\gamma = 1$, $\mu = 1$. Information and epidemic networks have different random-regular (RR) topology, each of size $N = 1000$ and average degree $\langle k_1 \rangle = \langle k_2 \rangle = 10$.

Furthermore, we analyze the epidemic size by theoretically predicting final epidemic size in the parameter plane (γ, β), as shown by the color-coded value of ρ^{total} in Figures 6 (c) and (d). Likewise, there exists a prominent change in the phase transition induced by different incubation period $\frac{1}{\gamma}$ in both (c) $\alpha = 0.5$ and (d) $\alpha = 0.8$. For large value of γ , in the vertical direction, there is a sudden change in the color from dark blue to light yellow, denoting a first-order phase transition. For small value of γ , in the vertical direction, the color gradually changes from dark blue to green, orange, and lastly to yellow,

signifying a continuous phase transition. In the case of first-order transition, the system is more vulnerable to the epidemic cause a tiny infection probability can result in the sudden and large-scale outbreak of the epidemic. Thus, the epidemic with short incubation period should be paid exceptional attention and take fast coping strategies.

Fig. 8 Comparison of the propagation of the epidemic under RR-RR, SF-RR, SF-SF networks. Shown are the final epidemic size ρ^{total} versus λ for (a) $\alpha = 0$ and (b) $\alpha = 0.5$. Thereinto, $\beta = 0.4$. Shown are the final epidemic size ρ^{total} versus β for (c) $\alpha = 0$ and (d) $\alpha = 0.5$. Thereinto, $\lambda = 0.2$. Each symbol is obtained by Monte Carlo simulations, while colored curves represent the results calculated by MMC theory. The initial density of AI-state individuals is set to be 0.01. The other parameters for the simulations are $\delta = 1$, $\gamma = 1$, $\varepsilon = 0.5$, $\mu = 0.2$.

However, if the therapeutic efficacy of resources is enhanced, the first-order phase transition induced by short incubation period would transform into the second-order phase transition. Under the premise of $\gamma = 1$, Figures 7 (a) and (b) show final epidemic size ρ^{total} versus infection probability β with two different values of ε for $\alpha = 0.5$ and $\alpha = 0.8$ respectively. For $\varepsilon = 0.2$,

corresponding to low efficacy of resources, the growth of ρ^{total} exhibits a first-order phase transition with the emergence of hysteresis loop. But for $\varepsilon = 0.8$, it can be found hysteresis loop vanishes and ρ^{total} shows a continuous increase, which indicates a second-order phase transition. Further, we explore how ε affects phase transition in the parameter plane (ε, β) , as shown by the color-coded value of ρ^{total} in Figures 7 (c) and (d). It can be found the first-order phase transition apparently turns into the second-order phase transition with the increase of ε . This finding reminds it crucial to improve the efficacy of resources in disease control, which are helpful to slow down the outbreak of diseases and reduce the final epidemic size.

Lastly, we consider how the epidemic evolves in different kinds of multiplex networks. Specifically, scale-free (SF) networks with 1000 nodes and degree exponent 3 (i.e., $P(k) \sim k^{-3}$) are introduced. As is shown in the legend of Figure 8, RR-SF(RR-RR/SF-SF) represents that RR (RR/SF) network and SF (RR/SF) network is employed to describe the information layer and epidemic layer, respectively. Figures 8 (a) and (b) show final epidemic size ρ^{total} versus λ in different networks for (a) $\alpha = 0$ and (b) $\alpha = 0.5$. Comparing with the RR-RR network, we find that merely changing the structure of the information layer to the SF network has little effect on the propagation of the epidemic. However, if the network structure of the epidemic layer is SF network, final epidemic size is significantly smaller. When studying ρ^{total} versus β on different networks, the same phenomenon can be found, as is shown in Figures 8 (c) and (d). A potential explanation may be there are individuals with higher degrees who have more chances to get resources to recover themselves in SF network. Thus, the probability of these individuals infecting their neighbors is reduced, hindering the propagation of the epidemic.

5 Discussion

For some epidemics, like COVID-19, there are patients lacking distinct symptoms in the initial stage of infection. This asymptomatic infection of Exposed-state individuals may trigger unexpected effects on the evolutionary dynamics of the disease. Besides, epidemic spreading tends not to be an isolated process in the physical contact space, but it is always coevolving with the diffusion of related information and resource allocation. Specifically, when epidemic spreads on the physical contact networks, corresponding information disseminates through the virtual communication networks, which stimulates aware individuals to positively seek resources to protect themselves. The resources enhance the recovery probabilities of the infected individuals, thereby in turn affecting the epidemic spreading dynamics. In general, studying the interplay and coevolution among epidemic propagation, information diffusion and resource allocation is extremely complicated, but is crucial to accurately predict the future trend of the epidemic and develop effective control measures as well.

To this end, we have constructed a three-layered network model to better explore the spreading characteristics of epidemic with exposed state under the above coevolution. We have analyzed the proposed model and calculated the epidemic size and epidemic threshold based on microscopic Markov chain theory. The epidemic threshold has been proved to depend on the topology of the epidemic network, and processes of information diffusion and resource allocation. Then extensive Monte Carlo simulations have been conducted, validating the accuracy of the theoretical predictions. We have verified that information diffusion prominently suppresses the propagation of the epidemic. With the same information dissemination probability, epidemics more dependent on resources in recovering spread more widely. Meanwhile, a series of interesting phenomena involved in incubation period of the disease have been found. Firstly, we have found when epidemics are less dependent on resources in recovering or with a long incubation period, there are actually more E-state individuals than I-state individuals, awaking us to watch out for the second outbreak of the epidemic induced by E-state individuals. Secondly, there is a dual effect of combinations of incubation period and infection capacity on the epidemic propagation. If the infectious capacity of diseases is weak, final epidemic size decreases with incubation period increases. However, if diseases are with strong infectious capacity, long incubation period will exacerbate the spread of epidemic. This finding enlightens us to especially control the diffusion of the strong infectious diseases with long incubation period. In addition, it has been found a remarkable change in the phase transition caused by variations of incubation period. Long incubation period leads to second-order phase transition of the growth of ρ^{total} , but short incubation results in first-order transition, which means the system is more vulnerable to the epidemic. Accordingly, for the epidemic with short incubation period, the governments should quickly take effective measures to avoid an abrupt and large-scale explosion, such as improving the efficacy of resources. Based on our findings, enhancing the therapeutic efficacy of resources would convert this first-order phase transition to a second-order type. At last, we have studied the coevolution on different kinds of networks. Compared with changing the network structure of information layer to the SF network, setting epidemic layer as SF network will significantly hinder the propagation of the epidemic.

Our modeling framework and theory makes a substantial contribution to understanding the dynamical mechanisms and characteristics of epidemic spreading with exposed state under the influence of the information-driven resource allocation. The findings obtained in this paper will be of practical significance in developing more effective epidemic control strategies, especially for epidemic with exposed state, like COVID-19.

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Statements and Declarations

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Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

Author Contributions

The study conception was proposed by Xuzhen Zhu. The model was designed by Xuzhen Zhu and Xiaochen Wang. Theoretical derivation and analysis were conducted by Xiaochen Wang and Yuxin Liu. Simulation and results analysis were completed by all authors. The first draft of the manuscript was written by Yuxin Liu and all authors reviewed and revised the manuscript. All authors read and approved the final manuscript.

Data Availability

No real dataset was utilized in the current study. The networks (random-regular networks or scale-free networks) in this paper were randomly generated by computer program.