RESEARCH ARTICLE

Simulation study on the evolution of SIS, SIR, and SEIR disease transmission models based on double layer networks

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Infectious disease transmission dynamics is an important field at the intersection of complex network science and epidemiology, aiming to determine disease spread patterns within populations and the key factors influencing these processes. All traditional homogeneous mixing models such as susceptible-infected-susceptible (SIS), susceptible-infected-recovered (SIR), and susceptible-exposed-infected-recovered (SEIR) assume population contact to be random, yet the interaction structures in real-world societies present significant heterogeneity and hierarchy. This research aimed to delve into infectious disease transmission dynamics in complex networks by developing a more realistic social-family bi-layer network model. Social and family contact structures were integrated to explore infectious disease transmission dynamics. The upper network simulated social contact networks using scale-free, small-world, and regular network models, while the lower network simulated family community networks. SIS, SIR, and SEIR models were applied for the simulation of disease transmission and the impacts of infection rate, recovery rate, and immunization strategies of random and targeted immunization were investigated through sensitivity analyses. The obtained results indicated that scale-free networks exhibited rapid epidemic spread due to their highly connected node characteristics, while small-world networks presented slower and more stable transmission rates. Targeted immunization proved effective in scale-free networks, while random immunization was more effective in family community networks. This research not only provided a novel theoretical perspective to understand infectious disease transmission mechanisms in complex networks but also laid a scientific foundation for the formulation of targeted public health intervention measures..

Keywords: complex network; SIS model; SIR model; SEIR model; transmission threshold; evolutionary game.

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Introduction

Disease transmission models serve as essential tools in investigating disease transmission dynamics with the classic susceptible-infected-susceptible (SIS), susceptible-infected-recovered (SIR), and susceptible-exposed-infected-recovered (SEIR) models as widespread applications in epidemiological research. However, traditional disease transmission models generally assume random contacts within the population, overlooking complex network relationships among individuals in the real world. Recently, with the introduction of complex network theory, researchers have incorporated network structures into disease transmission

models for more accurate simulation of the spread of disease. Research on the evolutionary mechanisms of emergent infectious diseases mainly encompasses four categories including descriptive research [1], analytical research [2], experimental research [3], and theoretical research [4]. Regarding the evolutionary models of emergent infectious diseases, since the introduction of SIR compartment model by Kermack and McKendrick in 1926 [5], many researchers have proposed more realistic mathematical differential equations including the solutions and analyses of effective reproductive number, system dynamics models based on complex networks, and evolutionary game models. Studies on epidemics or epidemic processes are mainly based on compartment models [6, 7], which are commonly applied in epidemiology and modern health management systems. SIR model is among the most popular epidemiological models [6, 8], while other models such as exposed or asymptomatic agent models of SEIR and SEAIR are also employed [9-11].

Complex networks provide a more realistic environment for infectious disease transmission. To more precisely apply prevention and control strategies, it is essential to develop multilayer networks for evolution and discussion. By adjusting key parameter values, the performance under complex multilayer network structures can be explored and provided reference for auxiliary prevention and control. Transmission dynamics on complex networks are important research topic in network science [12, 13]. A wide range of transmission trajectories are possible in systems such as infectious diseases, information, and rumors and can be affected by several factors that rely on the transmission characteristics of the transmitted items themselves as well as global network structural properties. Generally, classical network models including random [14], small-world, and scale-free networks [15] can be employed to evaluate the impacts of single-layer network structures on transmission processes. Currently, infectious disease transmission in human contact networks remains a key research topic in complex network transmission dynamics [16-18]. Complex network is a research concept to simplify network models of complex systems. Multi-layer networks are continuously updated based on the properties of individual network nodes and network edges. These networks can be divided into multidimensional multi-layer networks and dependency multi-layer networks based on the type of the network. Multidimensional multi-layer networks were designed to address the problem of duplicate edges in multiple networks arising due to the homogeneity of network nodes and edges in complex networks. Similarly, dependency multi-layer networks were developed to address the issue that network nodes in complex networks might possess different attributes. Although dependency multi-layer networks do not allow edges among nodes in different networks, they allow nodes in different networks to have interdependent relationships. Most individual networks can be considered as dependency multi-layer networks. In multi-layer networks, selecting various types of networks for each layer results in multi-layer networks with different complexities [19]. With the introduction of the Internet to complex network theory, the research paradigm of complex networks can be employed in research in a wide variety of fields such as cooperative propagation model threshold study in a three-layer scale-free associative network [20].

This research aimed to explore infectious disease transmission dynamics in complex networks by constructing a dual-layer network consisting of social and household communities. The upper layer network simulated social contacts using scale-free, small-world, and regular networks, while the lower layer network simulated household communities. SIS, SIR, and SEIR models were applied to simulate disease transmission and the impacts of infection rate, recovery rate, and immunization strategies including random immunization and targeted immunization. This study provided a new perspective to understand infectious disease transmission mechanisms in complex networks

as well as a theoretical basis for formulating effective strategies for infectious disease prevention and control, which not only enriched the application of complex network theory in the field of infectious disease transmission but also provided a scientific basis for public health decision-making. By simulating the impacts of various network structures and immunization strategies on infectious disease spread, the epidemic development trends could be better predicted, and the resource allocation could be well optimized, which would formulate more targeted prevention and control measures.

Materials and methods

Selection and design of the upper-level network

A dual-layer network with the upper layer network represented the social network of individuals and the lower layer network represented their family community network was adopted in this research. Three typical complex network models were applied for the upper-level social network including Barabási–Albert scale-free network (BA model), Watts-Strogatz small-world network (WS model), and regular network. The scale-free network was established *via* a preferential attachment mechanism, reflecting "super-spreaders" phenomenon in real-life social interactions, while small-world network introduced random rewiring based on the regular network, combining high clustering and short path features, and the regular network served as a control, adopting a uniform connection pattern. These three networks corresponded to various social contact scenarios, providing a foundation for the analysis of network heterogeneity impacts on disease transmission. A BA scale-free network was constructed, equally dividing the total number of nodes (N) based on the set edge number (m) for each node, ultimately presenting a single-layer network with each node having m edges. The initial number of nodes was set as m_0 = 5 and the number of edges for newly added nodes was adjusted as m = 4, constructing a network with a power-law degree distribution. A WS small-world network was constructed, where the total number of nodes (N) was set based on the degree of each node (k), and random reconnection probability (cp) was equally distributed among each node and other unconnected node. A single-layer network was established, where each node had a degree of k and a random reconnection probability of cp to connect with other unconnected nodes. Node number was set as N = 200, the initial number of neighbours was set as k = 4, and reconnection probability was p = 0.3. A regular network was constructed by equally dividing the total number of nodes (N) based on the preset degree of each node (k), ultimately generating a single-layer network with each node having a degree of k. Node number was set as N = 200 with each node fixedly connected to its nearest k = 4 neighbours. Python's networkx library for BA/WS model and custom algorithms for regular networks were implemented. Pymnet package was used for multilayer network visualization, multilayer network processing, scalable implementation of sparse networks, and various network analysis transformations, reading, and writing tasks.

Design of the lower family community network

To generate a family community network, it was necessary to create a total node number (N) representing households of 3, 5, and 6 members. By setting different probabilities, fixed nodes were randomly assigned to households with different sizes and household number was not fixed. During family community network construction, there might be instances that the formed final total number of households was less than the fixed total number set. Therefore, it was necessary to determine whether the set value had been reached. If it had, nodes were sorted for each household. The network edges were eventually set for the nodes that made up the households, establishing family community network. Family cluster network referred to the entire social group, where each family was an independent entity and the nodes of each family were interconnected. The number of nodes in a family corresponded to the number of people in that family and the number of nodes in different

families might be different or equal, meaning that the number of people in each family might vary or be the same. No connection was observed among families, therefore, epidemic spread could only occur within the family. Family cluster network resembled the population distribution formed by people in the real world at night.

Basic evolutionary model of infectious diseases

The evolutions of SIS, SIR, and SEIR on complex bipartite networks were investigated using a fundamental mathematical model that was developed by solving differential equations with the premise of a regular and homogeneous network with contact probability among individuals. This model presented an evolutionary pattern on the double layer networks by settings the developed mathematical model. Its parameters still used the parameters of the mathematical model listed below, but the evolution was no longer obtained by solving mathematical equations. Instead, it implemented nonlinear evolution process *via* the rules of a simulation program.

(1) SIS mathematical model

In SIS model, the population was divided into two groups including susceptible individuals and infected individuals. Infected individuals could be transformed into susceptible individuals through treatment or other external interventions. However, they might not be immune to the specific infectious disease. There was no death throughout the entire process. An infectious disease model possessing the above characteristics was considered as the SIS model and was mathematically defined as below.

$$\frac{dS}{dt} = \frac{-\beta * I}{N} * S + \gamma I \quad (1)$$

$$\frac{dI}{dt} = \frac{\beta * I}{N} * S - \gamma I \quad (2)$$

where equation 1 was the variation in the number of susceptible individuals. Equation 2 was the variation in the number of infected individuals. N was the total number of individuals. S was the initial number of susceptible individuals. I was the initial number of infected individuals. β was infection rate. γ was recovery rate.

(2) SIR mathematical model

SIR model divided the population into three categories as susceptible individuals (S), infected individuals (I), and the recovered individuals who had antibodies after recovery. An infectious disease model with the above characteristics was an SIR model mathematically defined as follows.

$$\frac{dS}{dt} = \frac{-\beta * I}{N} * S \quad (3)$$

$$\frac{dR}{dt} = \gamma * I \quad (4)$$

$$\frac{dI}{dt} = \frac{\beta * I}{N} * S - \gamma * I \quad (5)$$

where equation 3 was the changes in the number of susceptible individuals. Equation 4 was the changes in the number of recovered individuals. Equation 5 was the changes in the number of infected individuals. N was the total number of individuals. S was the initial number of susceptible individuals. I was the initial number of infected individuals. R was the initial number of recovered individuals.

(3) SEIR model

SEIR model divided the population into four categories including susceptible individuals (S), latent individuals (E), infected individuals, and recovered individuals. An infectious disease model exhibiting these characteristics was referred to as SEIR model and was mathematically represented as follows.

$$\frac{dS}{dt} = \frac{-\beta * I}{N} * S \quad (6)$$

$$\frac{dR}{dt} = \gamma * I \quad (7)$$

$$\frac{dE}{dt} = \frac{\beta * I}{N} * S - q * E \quad (8)$$

$$\frac{dI}{dt} = q * E - \gamma * I \quad (9)$$

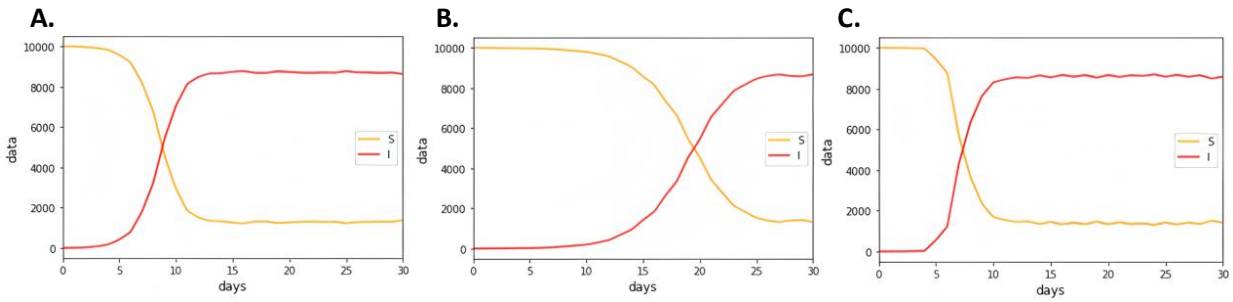

Figure 1. The development trend chart of the model where the lower network was a home area network and the upper-layer network could be varied over time. **A.** The upper layer is a regular network. **B.** The upper layer is a small-world network. **C.** The upper layer is a scale-free network. The Y-axis was the number of individuals.

where equation 6 was the change in the number of susceptible individuals. Equation 7 was the change in the number of recovered individuals. Equation 8 was the change in the number of latent individuals. Equation 9 was the change in the number of infected individuals. N was the total number of individuals. S was the initial number of susceptible individuals. E was the initial number of latent individuals. I was the initial number of infected individuals. R was the initial number of recovered individuals, q was the probability of a latent individual transitioning to an infected individual.

Results and discussion

Implementation of a two-layer network evolution model

The upper and lower mathematical models adopted SIS model, which involved susceptible and infected individuals. After encountering an infected individual, a susceptible individual had a certain probability of being infected and the infected individual had a certain probability of recovering to become a susceptible individual. The lower network model selected a household community network, where the distribution of population was in the form of households with nodes within a household being interconnected and no connections among households. This feature aligned with the distribution of population in real-life nighttime scenarios. The upper network could vary including regular with degree of 4, small-world, and scale-free networks. In regular networks, all nodes had a degree of 4. In small-world networks, most nodes were not connected to other nodes, and two nodes might be connected through multiple nodes, resulting in an association. In scale-free networks, most nodes were connected to other nodes, and the degrees of nodes could be varied significantly. Epidemic spreads between the upper and lower networks and population state update method for the two-layer network was that the upper layer influenced the lower layer. That is, if a neighbor of a node in the upper layer was an infected individual, there was a certain probability for the infection of the node. After being infected, the corresponding node in the lower network would also be infected (Figure 1). The upper- and lower-level network models remained unchanged as scale-free and household community networks, respectively. The reason for choosing scale-free network for the upper-level was that it was closer to reality and the data simulated was more referential. The lower level adopted household community network, which aligned with the distribution of people in real-life nighttime scenarios. Mathematical models could be either SIS, SIR, or SEIR. SIS included two individual types in the model as susceptible and infected individuals. Susceptible individuals had a certain probability of being infected by contact with infected individuals and infected individuals had a certain probability of recovering to become susceptible individuals. In this model, the equilibrium state

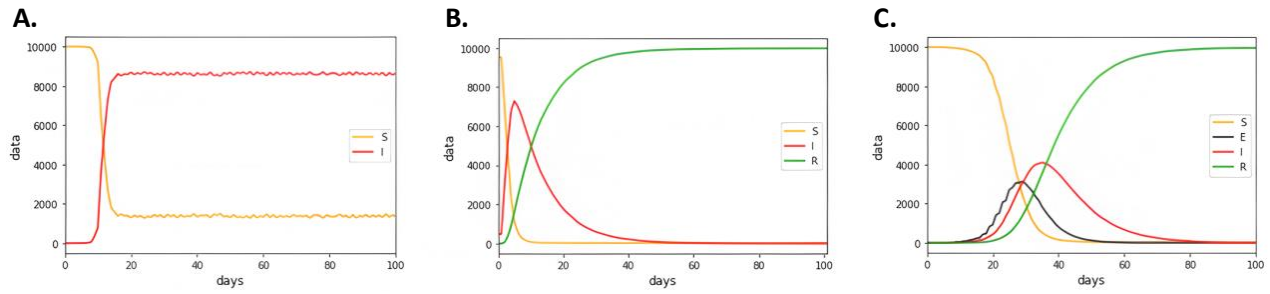

Figure 2. Variation trend diagrams of the invariant mathematical model of the double-layer network model over time. **A.** SIS model. **B.** SIR model. **C.** SEIR model. The vertical axis was the number of individuals.

was related to the infection rate (β) and recovery rate (γ). When β < γ, the system eventually stabilized with a majority of the population being susceptible. Conversely, when β > γ, infected individuals made up the majority at equilibrium. The SIR model had three individual types including susceptible, infected, and recovered individuals. Recovered individuals gained permanent immunity to the infectious disease. At the final equilibrium of this model, the recovered population typically constituted the majority. The final proportions of susceptible and infected individuals depended on the infection rate (β) and recovery rate (γ). When β > γ, the infected population would ultimately exceed the susceptible population with susceptible population possibly reaching zero. Otherwise, susceptible population would outnumber infected individuals. However, if β < γ, the number of susceptible individuals was greater than that of infected individuals. The SEIR model constituted of four individual types including susceptible, latent, infected, and recovered individuals. After being infected by an infected individual, a susceptible individual was not directly converted into an infected individual but was instead converted into a latent individual. With a certain probability, latent individuals could convert into infected individuals, and infected individuals had a certain probability of recovering to become permanently immune recovered individuals. Transmission mode of this model was that both the upper- and lower-level networks transmitted the epidemic with the upper-level influencing the lower-level. If a neighbour of a node in the upper-level was an infected individual and if that node was infected by an infected individual, the corresponding node in the lower-level network would also be infected (Figure 2).

Sensitivity analysis of model evolution

The upper networks were regular network, small-world network, and scale-free network, respectively. When the infection rates (β) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, each model was run 20 times to calculate the average value. The results showed that the infection rate (β) was gradually increased, and all three network-based epidemic models experienced significant surges in the number of infected individuals. However, comparison showed that scale-free network was the first to react with a very strong outbreak intensity, where the initial case growth process was close to exponential growth, leading to a rapid infection of most people. The small-world network was the last to experience an outbreak with the smallest outbreak intensity, but its growth process remained relatively balanced throughout the experiment, which might be caused by the characteristics of the network. When the upper-layer network was a small-world network, most nodes were not directly connected, i.e., direct contact was not possible, which significantly slowed down outbreak speed. However, when the upper-layer network was a scale-free network, all nodes were generally connected to other nodes with some nodes having very high degrees, meaning that they were connected to several other nodes. If such

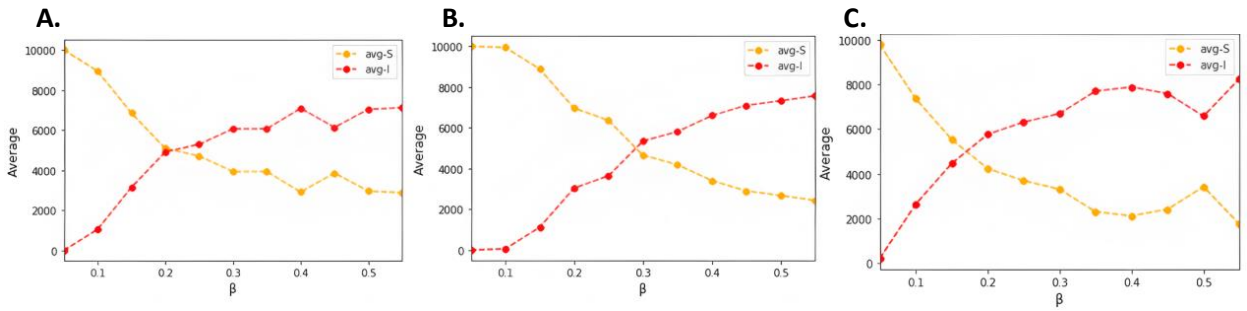

Figure 3. The development trend diagram of the model where the lower network was a family cluster network and the upper network changed with the infection rate (β). **A.** regular network. **B.** small-world network. **C.** scale-free network. The vertical axis was the number of people.

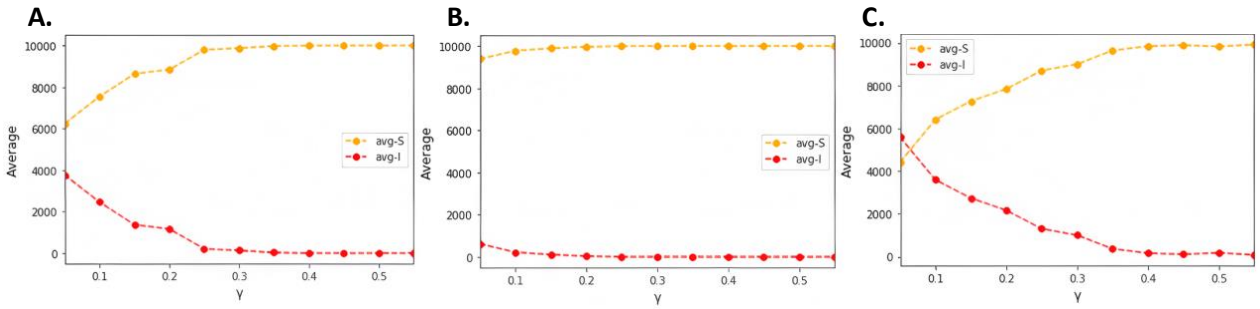

Figure 4. Trend chart of the development of the model where the lower network was a family cluster network and the upper network varied with the recovery rate (γ). **A.** regular network. **B.** small-world network. **C.** scale-free network. The vertical axis was the number of people

individuals became infected, the spread occurred extremely rapid, resulting in very high outbreak speeds for the scale-free network with growth process being far from stable (Figure 3). Small differences were observed among the three network types in terms of growth recovery rate. The equilibrium point in scale-free network was relatively unstable, which was also caused by the characteristics of the scale-free network. Because some nodes had sufficiently high degrees, if this group of individuals had not recovered, they were connected to several other nodes, making it easy for the nodes connected to these high-degree nodes to be reinfected. Therefore, the curves exhibited unstable variations during the change process. Similarly, if high-degree nodes were not infected and low-degree nodes were infected, the probability of other nodes being infected was relatively small and their infection rates were low. The variation trend of the regular network was relatively stable with no significant fluctuations occurring during the change process (Figure 4).

When lower network was a household community network and the upper network was a scale-free network, the results showed that the infection rate was gradually increased. The results obtained from all three models indicated that the epidemic became more severe, which could be inferred from the average number of infected individuals. Higher infection rates indicated higher average numbers of infected individuals at equilibrium in the model. When infection rate reached a certain level, the equilibrium state of SIS model was changed with almost all individuals being infected. However, the equilibrium states of SIR and SEIR models remained unaffected with the increase of infection rate, ultimately dominated by recovered individuals in both models. Comparison of SIR and SEIR models showed that variations in the number of infected individuals were basically consistent with slight differences

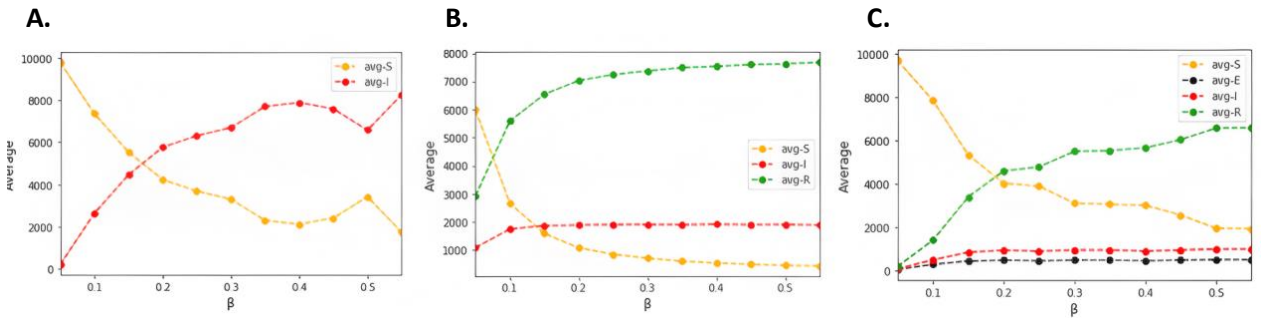

Figure 5. Variation trend diagrams of the invariant mathematical double-layer network models with different infection rates. **A.** SIS model. **B.** SIR model. **C.** SEIR model. The vertical axis was the number of people

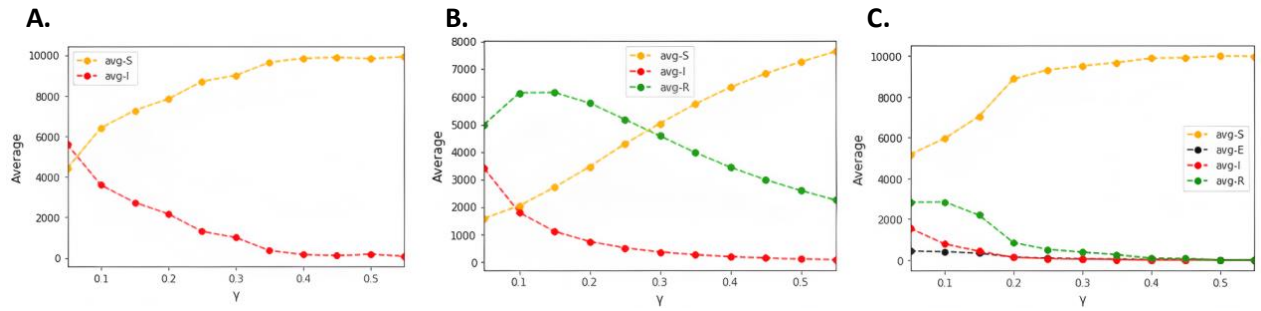

Figure 6. Variation trend diagrams of the invariant mathematical double-layer network model for different recovery rates. **A.** SIS model. **B.** SIR model. **C.** SEIR model. The vertical axis was the number of people

in variations in the numbers of recovered and susceptible individuals. In SIR model, when infection rate reached a certain level, the number of recovered individuals was consistently higher than that of susceptible individuals. In SEIR model, the numbers of recovered and susceptible individuals crossed multiple times as the infection rate increased, which was the presence of latent individuals in SEIR model. If the probability of converting latent individuals into infected individuals was too small, the number of infected individuals was not changed significantly in a short period of time. Also, the fact that latent individuals did not have infectious capability also reduced the probability of other susceptible individuals contacting with infectious individuals. Therefore, the number of susceptible individuals might be decreased after the infection rate reached a certain level (Figure 5).

When the recovery rates (γ) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6 and dynamic change mathematical models were SIS, SIR, and SEIR, the results showed that, with the gradual increase of recovery rate, the epidemic situations of the three models were improved. During the change process, SIS model changed relatively smoothly with no significant fluctuations. When recovery rate reached a certain level, the final number of infected individuals was decreased to below that of susceptible individuals. As recovery rate was increased, the final number of infected individuals was decreased, eventually approaching 0. At this point, the corresponding recovery rate represented the practical significance that such an infectious disease could not break out or spread and the epidemic could be rapidly controlled. In the change process of SIR model, the number of both infected and recovered individuals showed downward trends and higher recovery rates resulted in smaller numbers of infected and recovered individuals. In the change process of SEIR model, the number of infected individuals was slightly changed with the increase of recovery rate. When recovery rate reached a certain level, the number of

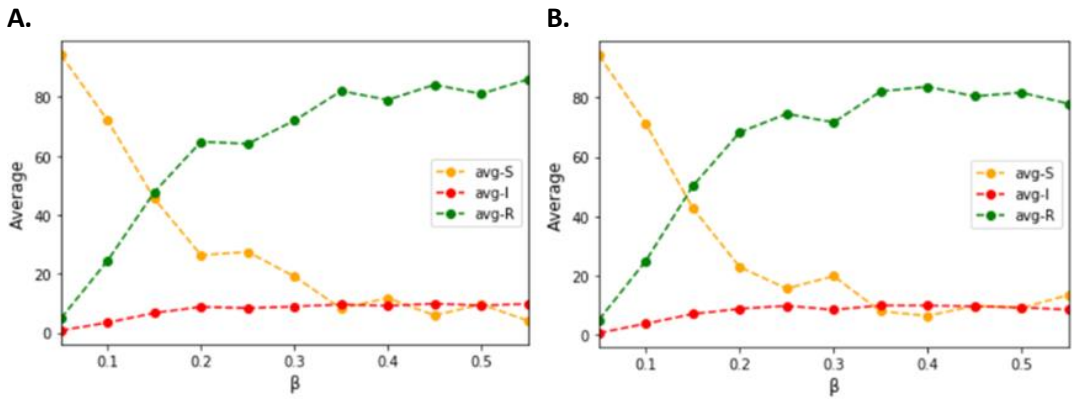

Figure 7. Evolution trend diagrams of the infection rate dynamic change model in the upper and lower network layers under random immunization. **A.** upper network. **B.** lower network.

susceptible individuals approached to 0 (Figure 6), which was because susceptible individuals were not immediately converted into infected individuals after being infected, but were instead converted into latent individuals. Latent individuals could not infect other individuals, therefore, when recovery rate was relatively high, many infected individuals were cured within the time required for latent individuals to convert into infected individuals. Therefore, the number of infected individuals continued to decrease. The results suggested that the common point among the three models was that, with the increase of recovery rate, the number of infected individuals was continuously decreased. When the recovery rate was excessively high, the number of susceptible individuals became the largest in the population.

Sensitivity analysis of random immune occurrence in upper and lower network models

Random immunity referred to a model in which each node had a certain probability of acquiring immunity, making it unable to be infected. This type of immunity could occur in both the upper and lower layers of the network. Implementation of random immunity in the model was reflected in the disconnection of the node from other nodes, preventing its infection by them. SIR mathematical model with the upper-layer network model being a scale-free network and the lower-layer network model being a household community network were adopted. Random immunization could occur in both the upper and lower networks. When random immunization occurred in the upper-layer network, each random immunization targeted a single node. When random immunization occurred in the lower-layer network, each random immunization targeted an entire household. Once a node within a household was immunized, its neighbours were considered as family members. Therefore, immunizing a single node was equivalent to immunizing an entire household. Model propagation occurred separately in the upper and lower networks with the upper network influencing the lower network. During model state updated, if a node in the upper network had an infected neighbour, after being infected by the infected neighbour, the corresponding node in the lower network would also be infected. To better reflect the effect of immunization on the model, population size was set to 60. When Infection rates (β) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, random immune responses occurred in both the upper and lower layers. The results demonstrated that, with the increase of infection rate, the impacts of random immunization occurring in both the upper and lower layers on the model were essentially the same. After infection rate reached a certain level, the impact of random immunization occurring in the upper network became more pronounced with the majority of individuals eventually

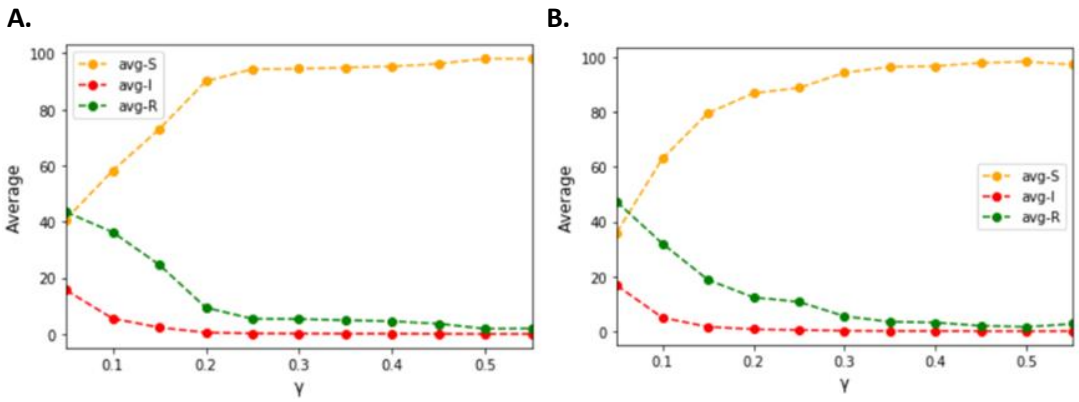

Figure 8. Evolution trend diagrams of recovery rate dynamic changes in random immune systems occurring in upper and lower network models. **A.** upper network. **B.** lower network.

recovering. However, when random immunization occurred in the lower network, ultimately the number of susceptible individuals exceeded that of infected individuals. The common point of random immunization in upper and lower networks was that the number of susceptible individuals was decreased with the increase of infection rate. The number of infected individuals was gradually increased, while the number of recovered individuals continued to increase (Figure 7). The difference lied in the fact that, when random immunization occurred in the upper network and the infection rate β was too high, the number of infected individuals exceeded that of susceptible individuals. On the other hand, when random immunization occurred in the lower network and infection rate was too high, the number of susceptible individuals exceeded that of infected individuals.

When recovery rates (γ) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, the random immune responses occurred in the upper and lower layers showed that, with the increase of recovery rate, random immunization occurred either in the upper or lower layer. In the equilibrium state of the model, the number of susceptible individuals was gradually increased. However, when random immunization occurred in the lower network layer, the change process of the model was relatively smooth, and the response was relatively rapid. When it occurred in the upper network layer, the change of the model was more extreme, which was because, when random immunization occurred in a community network, each immunization targeted one family, whereas in a scale-free network, nodes had different degrees and therefore, the impact of each immunization was different (Figure 8).

Sensitivity analysis of pinpoint immunization occurred in both the upper-layer and lower-layer network models

Site-specific immunization referred to selecting a specific point within a model to confer immunity, ensuring it remained uninfected. In this context, site-specific immunization specifically denoted high-level immunity, meaning that the point with the highest degree in the model was guaranteed to acquire immunity. Implementation method in the model involved traversing to identify the point with the highest degree and then, removing all its connections, turning it into an isolated point, thus achieving the effect of being uninfected. In the mathematical model SIR, the network model was consisted of an upper layer scale-free network where most nodes were connected and had different degrees and a lower layer family cluster network where the number of most family nodes varied, meaning most nodes had different degrees. When targeted immunization occurred within the cluster network, all nodes in the family of the selected node acquired corresponding immunity, that is,

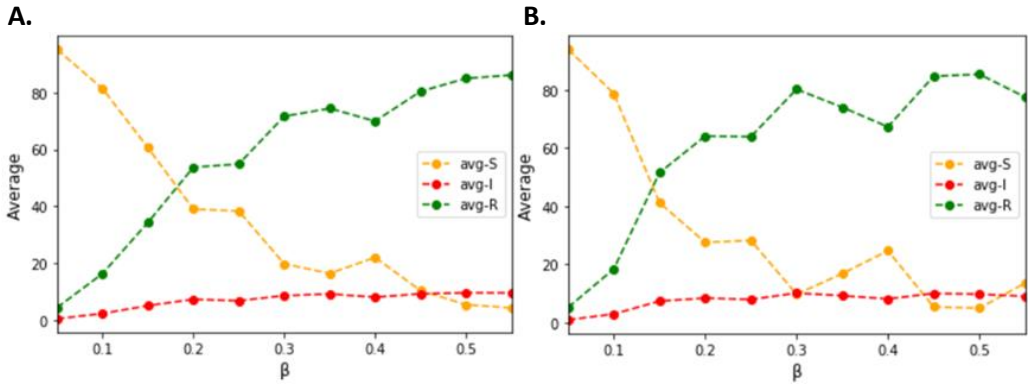

Figure 9. Trend diagrams of infection rate change model with targeted immunization occurring in both upper and lower network layers. **A.** fixed-point immunization in the upper network. B. fixed-point immunization in the lower network.

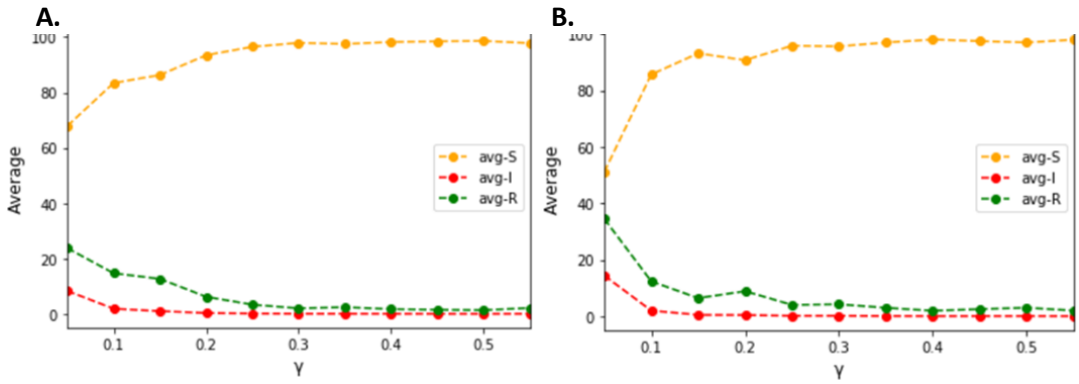

Figure 10. Trend diagrams of development for fixed-point immunization occurring in the upper and lower network models. **A.** random immunization in the upper network. **B.** random immunization in the lower network.

all nodes were converted into isolated points. When infection rates (β) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, targeted immunization occurred in the upper and lower layers, and the changing characteristics of infectious disease evolution models demonstrated that, with the increase of infection rate, targeted immunization occurred in both the upper and lower networks and model change trends were basically consistent. In addition, model changing process was not smooth, which was related to the nature of targeted immunization. When targeted immunization occurred in the lower network, once the infection rate reached a certain level, the number of susceptible individuals gradually exceeded that of infected individuals. However, when targeted immunization occurred in the upper network, once the infection rate reached a certain level, the number of susceptible individuals was always lower than that of infected individuals (Figure 9). When recovery rates (γ) were 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, targeted immunization occurred in the upper and lower layers, and the changing characteristics of infectious disease evolution models showed that, when recovery rate was equal to 0, a significant difference was observed in the occurrence of targeted immunization between the upper and lower layers. Recovery rate of 0 indicated that infected individuals could not recover and the nodes with the highest degrees could acquire immunity only through targeted immunization. After the model reached equilibrium, the infection level in scale-free networks was lower than that in home area networks. The reason for this was that, in scale-free network, some nodes

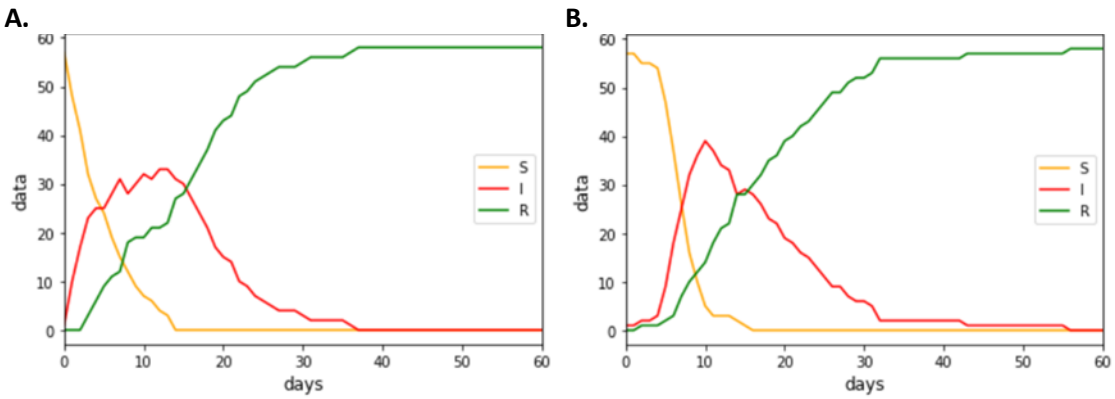

Figure 11. Development trend diagrams of the upper network model where targeted and random immunizations occurred, respectively. **A.** fixed-point immunization in the upper network. **B.** random immunization in the upper network.

had very high degrees. If the nodes with the highest degrees were immunized, this could have a significant positive effect on epidemic. However, in household community network, the number of individuals in each household ranged from 4 to 6 and the positive effect of targeted immunization was relatively smaller than that of scale-free network. Therefore, when the recovery rate equalled zero, the infection level in scale-free networks remained relatively low. As the recovery rate increased, both layers exhibited consistent trends, showing a gradual improvement in the epidemic situation (Figure 10).

Random and targeted immunizations occurring in the sensitivity analysis of the upper-layer and lower-layer network models

(1) Fixed-point and random immunizations occurring in the upper network

When random immunization occurred in the upper layer network, model change trend was more rapid, and epidemic outbreak duration was relatively longer than targeted immunization, making the time required for the model to reach equilibrium longer (Figure 11). The reason for this was that random immunization occurring in a scale-free network had a certain probability of encountering nodes with higher degrees. If such a node was immunized, it had a strong positive effect on the epidemic. However, targeted immunization occurring in a scale-free network definitely had a strong positive effect on the epidemic.

(2) Fixed-point and random immunizations occurring in the lower network layer

When random and targeted immunizations occurred within the family community network, network model change trend was more rapid and pronounced for random immunization, while that for targeted immunization was relatively slower and of shorter duration (Figure 12). The reason for this was that targeted immunization could achieve full immunization in households with the highest number of family members, while random immunization achieved full immunization in a household randomly. Compared with random immunization, targeted immunization resulted in a relatively lower peak infection level.

Conclusion

This research investigated the evolutionary processes of disease transmission models based on a dual-layer network and explored transmission dynamics of infectious disease in complex networks. The upper layer network simulated social contacts using scale-free, small-world, and regular networks, while the lower layer network simulated household communities. The research employed SIS, SIR,

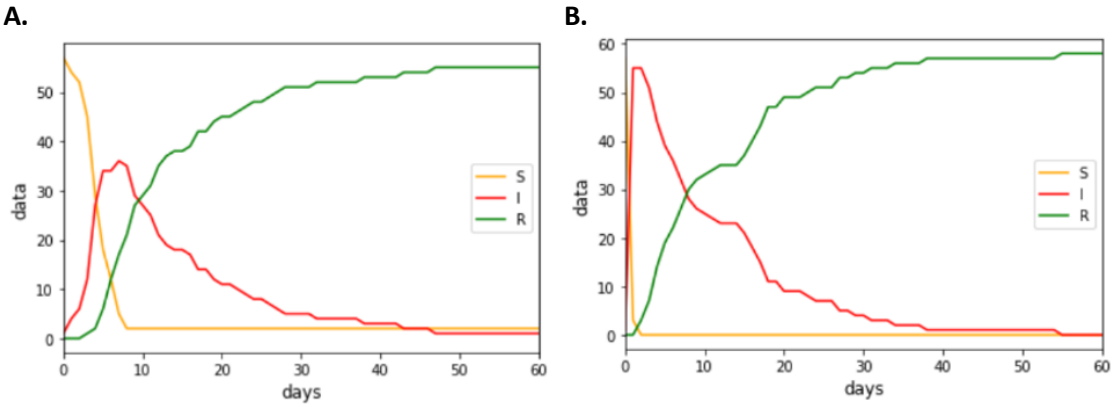

Figure 12. Development trend diagrams of random and targeted immunizations occurring in the lower-layer network model. **A.** random immunization in the lower network. **B.** targeted immunization in the lower network.

and SEIR models for the simulation of disease transmission and evaluated the influences of infection rate, recovery rate, and immunization strategies including random and targeted immunizations through sensitivity analyses. The results showed that scale-free networks exhibited rapid epidemic spread due to their highly connected node features with significantly lower transmission thresholds compared to traditional models, while small-world networks had slower and more stable transmission rates. Targeted immunization significantly inhibited epidemic spread in scale-free networks, while random immunization was more effective in household community networks. The findings of this research provided a new insight to understand infectious disease transmission mechanisms in complex networks and offered a theoretical basis to formulate effective strategies for the prevention and control of infectious diseases.

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