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Research Article

SELHR: A Novel Epidemic-Based Model for Information Propagation in Complex Networks

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The study of information spreading based on the complex network theory and topological structure has become an important issue in complex networks. Plenty of infectious disease models are widely used for information diffusion research in complex networks. Based on these state-of-the-art models, a new epidemic dynamic model with dynamic evolution equations is proposed and performed on the homogeneous and heterogeneous networks, respectively, in this paper. Meanwhile, we divide the propagation states into two states: L and H (low propagation ability groups and high propagation ability groups) and consider the transformation of these two states in our model. Then, the equilibria and stability of the model are analyzed for both homogeneous and heterogeneous networks to verify the validity of the proposed model. Finally, simulation results illustrate that the proposed model and information propagation dynamic evolution equations are reasonable and effective. Experiments with effect factors also reveal the interaction mechanism and the diffusion process of the proposed model in complex networks.

1. Introduction

Information dissemination [1, 2] represents the process by which information is transmitted from the original communicator to other receptors in social networks. Information can be news, rumors, opinions, diseases, and computer viruses in real life. Society provides people with different ways to exchange information through various channels. At present, the research on information dissemination in social networks mainly focuses on studies of the information dissemination process [3–6] and the studies of information dissemination prediction [7, 8]. Information propagation dynamic models are mainly divided into three categories: infectious disease dynamics models [9, 10], computer virus dissemination models [11], and rumor dissemination models [12, 13]. The process of information dissemination in complex networks is similar to the spread of diseases. Many scholars have applied infectious disease models to complex social networks to address the problem of information dissemination.

Modeling and analyzing the spreading process of the epidemic is imperative for an in-depth analysis of the

internal mechanism of epidemic spreading and prediction of the spreading range, which can be applied to effectively prevent and control the transmission of diseases. The classic infectious disease models can be traced back to the SI [14] model proposed by Vazquez and the SIR and SIS [15] models proposed by Kermack. With these as a foundation, many scholars have studied the dynamics of epidemic propagation and have created many significant epidemic propagation models. In particular, the SEIR [16] model was proposed based on the SIR model by adding the latent node E to represent the proportion of latent nodes in the network. The SEIR model was widely used in the early research on the law of information dissemination. With the development of technology, the SEIR model has been unable to accurately describe the dissemination of information. Samuel et al. [17] proposed a modified susceptible-exposed-infectious-recovered (SEIR) model for predicting epidemic dynamics incorporating pathogens in the environment and interventions. He et al. [18] built an SEIR epidemic model according to control strategies. However, most infectious disease models are unidirectional. Infection is the most important factor in the spread of disease in populations. It is

of great practical significance to classify the infected and study the internal transformation of the infected.

In addition, individuals in the network are regarded as nodes, and connections between individuals are regarded as edges for studying the epidemic spreading. According to whether the nodes of the network are of the same degree, the model can be divided into a homogeneous network model and a heterogeneous network model [19, 20]. Classical models study the dynamics of homogeneous networks and ignore the connections between individuals. The diseases will spread more rapidly than with fewer links, where individuals have a complex connection. A heterogeneous network is more realistic than a homogeneous network because of the different links between each individual. Xia et al. [21] proposed an improved susceptibility-exposure-infection-removal (SEIR) model with a hesitating mechanism, which considers the attractiveness and fuzziness of the content of rumors and verifies the dynamics of SEIR models both on homogeneous and heterogeneous networks. El-Saka studied the dynamics of the fractional model in the homogeneous network of the (SIRS) model [22] and improved their study of the dynamics of the fractional model in a heterogeneous network of the (SIRS) model [23].

Motivated by the above analysis, we propose a novel epidemic dynamic model called SELHR, which is improved from the epidemic SEIR model. For the SEIR model, there exists only one type of propagation state, which cannot greatly displays the real situations of disease dissemination. We divide the propagation state into two types: low-risk states and high-risk states, and add the transformation process of these two states, which can better match real-life situations. Furthermore, the proposed model SELHR is introduced for both homogeneous networks and heterogeneous networks. The main contributions to this paper are listed as follows.

(1) A new epidemic dynamic model (SELHR) is proposed based on the SEIR infectious disease model. We consider the different types of propagation states and add the transformed rate between the two types of propagation states.

(2) Dynamic evolution an equation of information propagation is constructed in both homogeneous and heterogeneous networks. Meanwhile, the equilibria and stability of the model are analyzed to verify the validity of the proposed model.

(3) Effect factors of the information propagation process both in homogeneous and heterogeneous networks are investigated. In addition, a series of simulation experiments are conducted to prove the superiority of the proposed model.

The rest of this paper is organized as follows. In Section 2, we introduce some related research on an information dissemination model based on the infectious disease model. In Section 3, we introduce the propagation rules of SELHR and drive the dynamic equations on both the homogeneous and heterogeneous networks. In Section 4, the equilibria and stability of the model are analyzed both for the homogeneous and heterogeneous networks. Section 5 is the simulation analysis of the above model. We finish the paper with Section 6, where the conclusion and future research work are discussed.

2. Related Work

The new generation of information technology includes but is not limited to blockchain [24], Big data [25], cloud computing [26, 27], Internet of Things [28–30], deep learning [31, 32], etc., which is a new state of full utilization of the information resources. Information dissemination has been studied and applied in various fields. For a 5G network, to improve the computational efficiency and to address the privacy and security of IoT (Internet of Things) data, Jin et al. [28] proposed a Multiple-Strategies Differential Privacy Framework on STF (MDPSTF) for HOHDST network traffic data analysis. For the Internet of medical things (IoMT), Mohammad et al. [29] reviewed the trust challenges in cloud computing and analyzes how blockchain technology can address these challenges. Computer virus propagation can also simulate the process of information propagation. Mohammad et al. [30] suggested a collection of strategies for preventing virus propagation in the computer population.

Studies based on the classic SIR model can be divided into three categories: variant infectious disease models, hesitated infectious disease models, and improved infectious disease models based on the classic model. In fact, in the process of virus spreading, not all infected people can recover after being infected. New viruses have a high chance of invading when people are in an infected state. Elena and Zhu [33] established the SIVR (susceptible infective variant recovery) model, where V represents the variant after infecting a new virus. Xu et al. [34] proposed a novel SIVRS mathematical model for infectious diseases spreading, where virus variation factors are considered to describe different contact statuses for different agents, including the susceptible, the infectious, the variant, and the recovery in a network.

Considering the mutual influence of forgetting and remembering mechanisms, Zhao et al. [35] proposed the SIHR (susceptible infected hibernator removed) model. By adding direct links from infected to stiflers, this model examines the final size of the rumor spreading under various spreading rates, stifling rates, forgetting rates, and average degree of the network. Considering individuals' opinion divergences and differentiations in online social media, Liu et al. [36] proposed a susceptible hesitated infected removed (SHIR) model to study the dynamics of competitive dual information diffusion.

An important purpose of information dissemination research based on the infectious disease model is to predict and control the information dissemination behavior of a system. Rui et al. [37] proposed a susceptible potential infective removed (SPIR) model, which analyzes the diffusion process based on discrete time. Considering the counter-attack mechanism of rumor spreading, Zan et al. [38]

introduced two new models: the susceptible infective counterattack refractory (SICR) model and the adjusted-SICR model, and the self-resistance parameter τ was introduced to study the influence of this parameter in rumor spreading. Wang et al. [39] analyzed that information dissemination in online social networks not only includes substantive news but also emotional expressions. They proposed an emotion-based spreader–ignorant–stifler (ESIS) model to simulate the process of information diffusion, which categorizes information cascades into fine-grained classes.

In reality, the interaction between individuals in networks is connected to the structure of heterogeneous distribution but not of homogeneous distribution. Xueyu et al. [40] proposed an epidemic SEIRV (susceptible exposed infected removed vaccinated) model and an evolutionary game model to analyze the difference between the mandatory vaccination method and voluntary vaccination method on heterogeneous networks. Kabir et al. [41] presented a modified susceptible vaccinated infected recovered (SIR/V) with the unaware-aware (UA) epidemic model in heterogeneous networks to study the effect of information spreading in the spatial structure of the vaccination game on epidemic dynamics. Taking two susceptible groups into account, Gui and Guo [42] developed a modified sub-healthy-healthy-infected- recovered (SHIR) model with time delay and a nonlinear incidence rate in networks with different topologies.

We can see in Table 1 that in the process of developing the model, the variables of the model are constantly increasing. More and more models take the effects of time lag, variation, and isolation into account. The attributes and characteristics of the information itself can also be used as part of the model parameters.

3. SELHR Spreading Model

The mutation of the new coronavirus has accelerated the expansion of the epidemic, which has had a huge impact on social and economic development. We proposed a new epidemic spreading model and applied it to simulate the process of information propagation on social networks. An individual in a complex social network is considered a node and the relationship between users is regarded as an edge. The population in complex social networks is divided into five groups: S, E, L, H, and R. Moreover, S represents the uninfected population. E refers to the group infected by the virus but still in the incubation period of the infection. L and H stand for the population with low propagation ability and the population with high propagation ability, respectively. R is the group of people who have been cured after contracting the virus. To simplify the calculation, assuming that the total population of the model is constant, the population entry rate and exit rate are the same, that is μ. The state transition diagram of the SELHR model is shown in Figure 1. The notations used in the proposed model are listed in Table 2.

3.1. Principle of the Information Propagation Process.
The rules of state transformation of the SELHR model can be summarized as follows:

(1) μ is the population entry and exit rates, ρ is the proportion of susceptible people in the new entry population.

(2) When an uninfected node S_i is linked to a propagation node L or H, the uninfected node S_i transforms to a latent node E with probability α_1 or α_2.

(3) For the latent node E, it can become a propagation node L or H with speed p_1 or p_2. In addition, it can also transform into an immune node R with speed v.

(4) For the propagation nodes L and H, when they are adjacent to an immune node R, the propagation nodes L and H will become immune node R with probability r_1 and r_2. Meanwhile, propagation nodes L and H can be converted to each other at speeds t_1 and t_2.

(5) For the immune node R, its state has not changed.

3.2. Node State Transition Probability.
For the node i in the complex social network, its state can be transformed among five different states (S, E, L, H, and R). The probability of node state transition in the period $[t, t + \Delta t]$ is shown in Table 3.

3.2.1. S ⟶ E.
If the node i is in S state at time t, it is obvious that:

$$P_{SS}^i + P_{SE}^i = 1. \tag{1}$$

The number of neighbors of a node i is k and states of its neighbors can be divided into , H and other states. m_1 and m_2 represent the number of propagation nodes L and H respectively. So, we can have as follows:

$$m_1 + m_2 + m_3 = k$$
$$P_{SS}^i = \left(1 - \Delta t \alpha_1\right)^{m_1} \left(1 - \Delta t \alpha_2\right)^{m_2} \tag{2}$$

It is supposed that node i has k edges and m_1, m_2 are random variables, which are subject to polynomial distribution:

$$P\left(k_1 = m_1, k_2 = m_2, k_3 = m_3\right)$$

$$= \frac{k!}{m_1! m_2! m_3!} P_{I_L S}(k, t)^{m_1} P_{I_H S}(k, t)^{m_2} \tag{3}$$

$$\left(1 - P_{I_L S}(k, t) - P_{I_H S}(k, t)\right)^{k - m_1 - m_2}.$$

$P_{I_L S}(k, t)$ is the probability from the susceptible node with k edges to a propagation node L. Likewise, $P_{I_H S}(k, t)$ is the probability of the susceptible node with k edges to a propagation node H.

TABLE 1: Analysis of different propagation dynamics models.

| Model	Node states	Parameters	Heterogeneous	Structure	Advantage
SIVR [33]	4	4	N	Unidirectional	Individuals can be exposed to new information in the dissemination of information influence.
SIVRS [34]	5	9	N	Unidirectional	Considering the influence of network node degree and mortality.
SIHR [35]	4	5	N	Bidirectional	Infectious disease model with forgetting mechanism H.
SHIR [36]	5	3	N	Unidirectional	A dynamic model of competitive dual information diffusion.
SPIR [37]	3	2	N	Unidirectional	The concept of potential spreader set is presented.
SICR [38]	4	5	N	Unidirectional	Present a counterattack group in rumor propagation model.
ESIS [39]	3	3	N	Unidirectional	Information disseminated in online social networks includes expressions of emotion.
SEIRV [40]	4	7	Y	Unidirectional	The impact of the cost of vaccination and disease treatment on the vaccination coverage rate is analyzed.
SIR/V [41]	7	5	Y	Unidirectional	Two layers SIR/V epidemic model is considered, and individuals are parted as an unaware and an aware state
SHIR [42]	4	10	Y	Unidirectional	Two dynamical systems are designed in homogeneous and heterogeneous networks by utilizing mean field equations.
SELHR	5	9	Y	Bidirectional	Transform rate between different types of propagation nodes is considered.
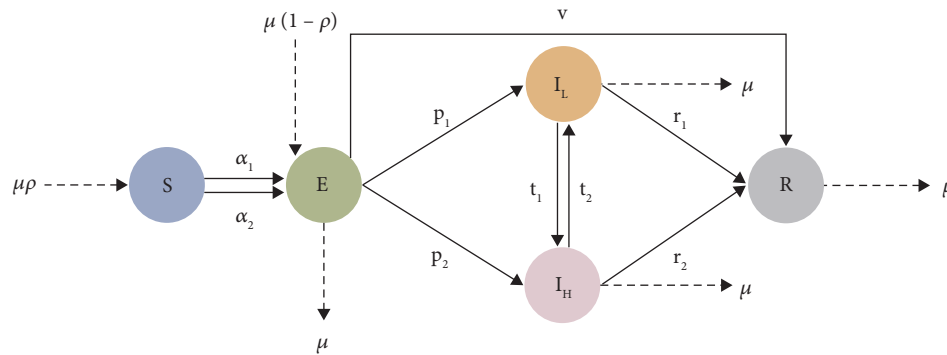
FIGURE 1: State transition diagram of the proposed SELHR model.

TABLE 2: Notations.

Symbol	Description	Value range
$S(k,t)$	The density of susceptible individuals with degree k at time t	[0, 1]
$E(k,t)$	The density of exposed individuals with degree k at time t	[0, 1]
$I_L(k,t)$	The density of infected individuals (L) with degree k at time t	[0, 1]
$I_H(k,t)$	The density of infected individuals (H) with degree k at time t	[0, 1]
$R(k,t)$	The density of recovery individuals with degree k at time t	[0, 1]
μ	The population entry and exit rates	[0, 1]
ρ	The proportion of susceptible person of entry population	[0, 1]
α_1	Probability of transforming to a latent node linked with node L	[0, 1]
α_2	Probability of transforming to a latent node linked with node H	[0, 1]
p_1	Speed of transforming to a propagation node L	[0, 1]
p_2	Speed of transforming to a propagation node H	[0, 1]
t_1	Probability of node L converted to node H	[0, 1]
t_2	Probability of node H converted to node L	[0, 1]
r_1	Probability of node L transforming to an immune node	[0, 1]
r_2	Probability of node H transforming to an immune node	[0, 1]
v	Speed of a latent node transforming to an immune node	[0, 1]
$\langle k \rangle$	Average degree of the network	Finite positive integer
$P(k)$	Degree distribution function	[0, 1]

$$P_{I_L S}(k,t) = \sum_{k_1} P(k_1|k) P\left(I_{Lk_1}|S_k\right),$$

$$P_{I_H S}(k,t) = \sum_{k_2} P(k_2|k) P\left(I_{Hk_2}|S_k\right), \tag{4}$$

where $P(k_1|k)$ is the degree correlation function, $P(I_{Lk_1}|S_k)$ represents the probability of a propagation node L whose degree is k_1 linked to a susceptible node whose degree is k. $P_{I_H S}(k,t)$ is the same as $P_{I_L S}(k,t)$.

Then, the average probability $\overline{P_{SS}(k,t)}$ of remaining susceptible state during the period $[t, t+\Delta t]$ can be expressed as follows:

$$\overline{P_{SS}(k,t)} = \sum_{m_1,m_2} \left(1-\Delta t\alpha_1\right)^{m_1} * \left(1-\Delta t\alpha_2\right)^{m_2}$$

$$* \frac{k!}{m_1! m_2! (k-m_1-m_2)!} P_{I_L S}(k,t)^{m_1} P_{I_H S}(k,t)^{m_2} \tag{5}$$

$$\left(1-P_{I_L S}(k,t)-P_{I_H S}(k,t)\right)^{k-m_1-m_2}$$

$$\approx \left(1 - \alpha_1 \Delta t P_{I_L S}(k,t) - \alpha_2 \Delta t P_{I_H S}(k,t)\right)^k$$

Hence, the average probability $\overline{P_{SE}(k,t)}$ of a node from a susceptible state to a latent state can be derived during the period $[t, t+\Delta t]$,

$$\overline{P_{SE}(k,t)} = 1 - \overline{P_{SS}(k,t)}$$

$$= 1 - \left(1 - \alpha_1 \Delta t P_{I_L S}(k,t) - \alpha_2 \Delta t P_{I_H S}(k,t)\right)^k. \tag{6}$$

3.2.2. $E \longrightarrow I$. It is supposed that node i is in a latent state at the time t, then,

$$P^i_{EE} + P^i_{ER} + P^i_{EI_L} + P^i_{EI_H} = 1, \tag{7}$$

and

$$\begin{cases} P^i_{ER} = \Delta t\nu, \\ P^i_{EI_L} = \Delta t P_1, \\ P^i_{EI_H} = \Delta t P_2. \end{cases} \tag{8}$$

3.2.3. $L \longrightarrow R$. It is supposed that the node i is in the propagation state L at time t. We can know that,

$$P^i_{I_L I_L} + P^i_{I_L I_H} + P^i_{I_L R} = 1. \tag{9}$$

The number of neighbors of a node i is k and the state of its neighbors can be divided into H, R and other states. n_1 and n_2 represent the number of propagation nodes H and immune nodes R, respectively. So, we can have as follows:

$$P^i_{I_L I_L} = \left(1-\Delta t t_1\right)^{n_1} * \left(1-\Delta t r_1\right)^{n_2} \tag{10}$$

It is supposed that node i has k edges, and n_1, n_2 are random variables, which are subject to polynomial distribution:

$$P\left(k_1 = n_1, k_2 = n_2, k_3 = n_3\right)$$

$$= \frac{k!}{n_1! n_2! n_3!} P_{I_H I_L}(k,t)^{n_1} P_{RI_L}(k,t)^{n_2} \tag{11}$$

$$\left(1-P_{I_H I_L}(k,t)-P_{RI_L}(k,t)\right)^{k-n_1-n_2}.$$

where $P_{I_H I_L}(k,t)$ is the probability of the propagation node L with k edges to a propagation node H. $P_{RI_L}(k,t)$ is the probability of the propagation node L with k edges to an immune node R.

$$P_{I_H I_L}(k,t) = \sum_{k_1} \frac{P(k_1|k) P\left(I_{Hk_1}|I_{Lk}\right)}{P_{RI_L}(k,t)} = \sum_{k_2} P(k_2|k) P\left(R_{k_2}|I_{Lk}\right) \tag{12}$$

where $P(I_{Hk_1}|I_{Lk})$ represents the probability of a propagation node H whose degree is k_1 linked to a propagation node L whose degree is k, $P_{RI_L}(k,t)$ is the probability of an immune node R whose degree is k_2 linked to a propagation node L which degree is k.

The average probability $\overline{P_{I_L I_L}(k,t)}$ of the remaining propagation state L during the period $[t, t+\Delta t]$ can be expressed as follows:

$$\overline{P_{I_L I_L}(k,t)} = \sum_{n_1,n_2} \left(1-\Delta t t_1\right)^{n_1} * \left(1-\Delta t r_1\right)^{n_2}$$

$$* \frac{k!}{n_1! n_2! (k-n_1-n_2)!} P_{I_H I_L}(k,t)^{n_1} P_{RI_L}(k,t)^{n_2} \tag{13}$$

$$\left(1-P_{I_H I_L}(k,t)-P_{RI_L}(k,t)\right)^{k-n_1-n_2}$$

$$\approx \left(1 - t_1 \Delta t P_{I_H I_L}(k,t) - r_1 \Delta t P_{RI_L}(k,t)\right)^k.$$

The propagation nodes L will be converted to propagation nodes H with speed t_1,

$$P^i_{I_L I_H} = \Delta t t_1. \tag{14}$$

Then,

$$P^i_{I_L R} = 1 - \overline{P_{I_L I_L}(k,t)} - \Delta t t_1 \tag{15}$$

3.2.4. $H \longrightarrow R$. It is supposed that node i is in the propagation state H at time t. We can know that,

$$P^i_{I_H I_H} + P^i_{I_H I_L} + P^i_{I_H R} = 1. \tag{16}$$

The same as $P^i_{I_L I_L}$, we can know,

$$P^i_{I_H I_H} = \left(1-\Delta t t_2\right)^{l_1} * \left(1-\Delta t r_2\right)^{l_2}, \tag{17}$$

and

$$P(k_1 = l_1, k_2 = l_2, k_3 = l_3)$$

$$= \frac{k!}{l_1! l_2! l_3!} P_{I_L I_H}(k,t)^{l_1} P_{R I_H}(k,t)^{l_2} \tag{18}$$

$$\left(1 - P_{I_L I_H}(k,t) - P_{R I_H}(k,t)\right)^{k - l_1 - l_2},$$

where

$$P_{I_L I_H}(k,t) = \sum_{k_1} P(k_1|k) P\left(I_{Lk_1} \mid I_{Hk}\right),$$
$$P_{R I_H}(k,t) = \sum_{k_2} P(k_1 \mid k) P\left(R_{k_2} \mid I_{Hk}\right). \tag{19}$$

Therefore, the average probability $\overline{P_{I_H I_H}(k,t)}$ of the remaining propagation state L during the period $[t, t + \Delta t]$ can be derived as follows:

$$\overline{P_{I_H I_H}(k,t)} = \sum_{l_1, l_2} \left(1 - \Delta t t_2\right)^{l_1} * \left(1 - \Delta t r_2\right)^{l_2}$$

$$* \frac{k!}{l_1! l_2! (k - l_1 - l_2)!} P_{I_L I_H}(k,t)^{l_1} P_{R I_H}(k,t)^{l_2} \tag{20}$$

$$\left(1 - P_{I_L I_H}(k,t) - P_{R I_H}(k,t)\right)^{k - l_1 - l_2}$$

$$\approx \left(1 - t_2 \Delta t P_{I_L I_H}(k,t) - r_2 \Delta t P_{R I_H}(k,t)\right)^{k}.$$

The propagation nodes H will be converted to propagation nodes L with speed t_2,

$$P^i_{I_H I_L} = \Delta t t_2. \tag{21}$$

Similarly,

$$P^i_{I_H R} = 1 - \overline{P_{I_H I_H}(k,t)} - \Delta t t_2. \tag{22}$$

3.3. Dynamic Equations of Information Propagation.

In the complex social network, we consider that the ratio of different states can be represented as $S(k,t)$, $E(k,t)$, $I_L(k,t)$, $I_H(k,t)$, $R(k,t)$ respectively; then,

$$S(k,t) + E(k,t) + I_L(k,t) + I_H(k,t) + R(k,t) = 1 \tag{23}$$

In the period $[t, t + \Delta t]$, the ratio changes of various nodes are as follow:

(1) Susceptible nodes
$$S(k, t + \Delta t) = \mu\rho + S(k,t)\overline{P_{SS}(k,t)} - \mu S$$

$$= \mu\rho + S(k,t)$$

$$\left(1 - \alpha_1 \Delta t P_{I_L S}(k,t) - \alpha_2 \Delta t P_{I_H S}(k,t)\right)^{k} - \mu S. \tag{24}$$

(2) Latent nodes
$$E(k, t + \Delta t) = \mu(1 - \rho)$$

$$+ E(k,t) + S(k,t)\left(1 - \overline{P_{SS}(k,t)}\right)$$

$$- E(k,t)\left(P_{ER} + P_{EI_L} + P_{EI_H}\right) - \mu E$$

$$= \mu(1 - \rho) + E(k,t) + S(k,t) \tag{25}$$

$$\left(1 - \left(1 - \alpha_1 \Delta t P_{I_L S}(k,t) - \alpha_2 \Delta t P_{I_H S}(k,t)\right)^{k}\right)$$

$$- E(k,t)\left(\Delta t v + \Delta t P_1 + \Delta t P_2\right) - \mu E.$$

(3) Propagation nodes

$$I_L(k, t + \Delta t) = I_L(k,t) + E(k,t)P_{EI_L} + I_H(k,t)P_{I_H I_L} - I_L(k,t)P_{I_L I_H} - I_L(k,t)\overline{P_{I_L R}(k,t)} - \mu I_L = I_L(k,t) + E(k,t)\Delta t P_1$$

$$+ I_H(k,t)P_{I_H I_L} - I_L(k,t)\Delta t t_1 - I_L(k,t)\left(1 - \left(1 - t_1 \Delta t P_{I_H I_L}(k,t) - r_1 \Delta t P_{R I_L}(k,t)\right)^{k} - \Delta t t_1\right) -$$

$$\mu I_L$$

$$I_H(k, t + \Delta t) = I_H(k,t) + E(k,t)P_{EI_H} + I_L(k,t)P_{I_L I_H} - I_H(k,t)P_{I_H I_L} - I_H(k,t)\overline{P_{I_H R}(k,t)} - \mu I_H$$

$$= I_L(k,t) + E(k,t)\Delta t P_2 + I_L(k,t)P_{I_L I_H} - I_H(k,t)\Delta t t_2 - I_H(k,t)\left(1 - \left(1 - t_2 \Delta t P_{I_L I_H}(k,t) - r_2 \Delta t P_{R I_H}(k,t)\right)^{k} - \Delta t t_2\right) - \mu I_H \tag{26}$$

(4) Immune nodes

$$R(k, t + \Delta t) = R(k,t) + E(k,t)P_{ER}$$

$$+ I_L(k,t)\overline{P_{I_L R}(k,t)} + I_H(k,t)\overline{P_{I_H R}(k,t)} - \mu R = R(k,t) + E(k,t)\Delta t v$$

$$+I_L(k,t)\left(1-\left(1-t_1\Delta t P_{I_H I_L}(k,t)-r_1\Delta t P_{RI_L}(k,t)\right)^k-\Delta t t_1\right)$$
$$+I_H(k,t)\left(1-\left(1-t_2\Delta t P_{I_L I_H}(k,t)-r_2\Delta t P_{RI_H}(k,t)\right)^k-\Delta t t_2\right)-\mu R. \tag{27}$$

For susceptible nodes:

$$S(k,t+\Delta t)-S(k,t)=\mu\rho+S(k,t)$$

$$\left(\left(1-\alpha_1\Delta t P_{I_L S}(k,t)-\alpha_2\Delta t P_{I_H S}(k,t)\right)^k-1\right) \tag{28}$$

$$-\mu S=\mu\rho+S(k,t)$$

$$\left(-\alpha_1\Delta t k P_{I_L S}(k,t)-\alpha_2\Delta t k P_{I_H S}(k,t)\right)-\mu S,$$

$$\frac{\partial P^S(k,t)}{\partial t}=\mu\rho-P^S(k,t)$$

$$\left(\alpha_1 k P_{I_L S}(k,t)+\alpha_2 k P_{I_H S}(k,t)\right)-\mu S\mu\rho-S_k(t)$$

$$\left(\alpha_1 k\sum_{k'}I_{Lk'}(t)P(k'|k)+\alpha_2 k\sum_{k'}I_{Hk'}(t)P(k'|k)\right) \tag{29}$$

$$-\mu S_k(t)$$

Similarly:

$$\frac{\partial P^E(k,t)}{\partial t}=\mu(1-\rho)+P^E(k,t)\left(-\nu-P_1-P_2\right)$$

$$+P^S(k,t)\left(\alpha_1 k P_{I_L S}(k,t)+\alpha_2 k P_{I_H S}(k,t)\right)-\mu E, \tag{30}$$

$$\frac{\partial P^{I_L}(k,t)}{\partial t}=P_1 P^E(k,t)+t_2 P^{I_H}(k,t)-t_1 P^{I_L}(k,t)$$

$$-P^{I_L}(k,t)\left(t_1 k P_{I_H I_L}(k,t)+r_1 k P_{RI_L}(k,t)-t_1\right)-\mu I_L, \tag{31}$$

$$\frac{\partial P^{I_H}(k,t)}{\partial t}=P_2 P^E(k,t)+t_1 P^{I_L}(k,t)$$

$$-t_2 P^{I_H}(k,t)-P^{I_H}(k,t)\left(t_2 k P_{I_L I_H}(k,t)\right.$$
$$\left.+r_2 k P_{RI_H}(k,t)-t_2\right)-\mu I_H, \tag{32}$$

$$\frac{\partial P^R(k,t)}{\partial t}=\nu P^E(k,t)+P^{I_L}(k,t)\left(t_1 k P_{I_H I_L}(k,t)\right.$$
$$\left.+r_1 k P_{RI_L}(k,t)-t_1\right)+P^{I_H}(k,t)$$
$$\left(t_2 k P_{I_L I_H}(k,t)+r_2 k P_{RI_H}(k,t)-t_2\right)-\mu R. \tag{33}$$

By further simplifying and improving the above model, we can apply it to homogeneous and heterogeneous networks, respectively.

For homogeneous networks, $k=\langle k\rangle$, let S stands for $P^S(k,t)$. Hence, the differential equation of homogeneous networks can be denoted as follows:

$$\left\{\begin{array}{l}\dfrac{dS}{dt}=\mu\rho-S\left(\alpha_1 k I_L+\alpha_2 k I_H\right)-\mu S\\[2mm]\dfrac{dE}{dt}=\mu(1-\rho)+S\left(\alpha_1 k I_L+\alpha_2 k I_H\right)\\[2mm]\qquad\qquad-E\left(\nu+P_1+P_2\right)-\mu E\\[2mm]\dfrac{dI_L}{dt}=EP_1+t_2 I_H-t_1 k I_L I_H-r_1 k I_L R-\mu I_L\\[2mm]\dfrac{dI_H}{dt}=EP_2+t_1 I_L-t_2 k I_L I_H-r_2 k I_H R-\mu I_H\\[2mm]\dfrac{dR}{dt}=E\nu+t_1 k I_L I_H+r_1 k I_L R+t_2 k I_L I_H\\[2mm]\qquad\qquad+r_2 k I_H R-t_1 I_L-t_2 I_H-\mu R\end{array}\right. \tag{34}$$

For heterogeneous networks, $S(t)=\sum S_k(t)P(k)$, $P(k)$ is the degree distribution function. We can conclude as follows:

$$\frac{dS_k(t)}{dt}=\mu\rho-S_k(t)$$

$$\left(\alpha_1 k\sum_{k'}I_{Lk'}(t)P(k'|k)+\alpha_2 k\sum_{k'}I_{Hk'}(t)P(k'|k)\right) \tag{35}$$

$$-\mu S_k(t),$$

$$\frac{dE_k(t)}{dt}=\mu(1-\rho)+S_k(t)$$

$$\left(\alpha_1 k\sum_{k'}I_{Lk'}(t)P(k'|k)+\alpha_2 k\sum_{k'}I_{Hk'}(t)P(k'|k)\right) \tag{36}$$

$$-E_k(t)\left(\nu+P_1+P_2\right)-\mu E_k(t),$$

$$\frac{dI_{Lk}(t)}{dt} = E_k(t)P_1 + t_2 I_{Hk}(t)$$
$$- t_1 k I_{Lk}(t) \sum_{k'} I_{Hk'}(t)P(k'|k)$$
$$- r_1 k I_{Lk}(t) \sum_{k'} R_{k'}(t)P(k'|k) - \mu I_{Lk}(t), \tag{37}$$

$$\frac{dI_{Hk}(t)}{dt} = E_k(t)P_2 + t_1 I_{Lk}(t)$$
$$- t_2 k I_{Hk}(t) \sum_{k'} I_{Lk'}(t)P(k'|k)$$
$$- r_2 k I_{Hk}(t) \sum_{k'} R_{k'}(t)P(k'|k) - \mu I_{Hk}(t), \tag{38}$$

$$\frac{dR_k(t)}{dt} = E_k(t)v + t_1 k I_{Lk}(t) \sum_{k'} I_{Hk'}(t)P(k'|k)$$
$$+ r_1 k I_{Lk}(t) \sum_{k'} R_{k'}(t)P(k'|k) + t_2 k I_{Hk}(t)$$
$$* \sum_{k'} I_{Lk'}(t)P(k'|k) + r_2 k I_{Hk}(t) \sum_{k'} R_{k'}(t)P(k'|k) \tag{39}$$
$$- t_1 I_{Lk}(t) - t_2 I_{Hk}(t) - \mu R_k(t).$$

Let,

$$\begin{cases} \Theta_{I_L}(k) = \sum_{k'} I_{Lk'}(t)P(k'|k), \\ \Theta_{I_H}(k) = \sum_{k'} I_{Hk'}(t)P(k'|k), \\ \Theta_R(k) = \sum_{k'} R_{k'}(t)P(k'|k), \end{cases} \tag{40}$$

where $\Theta_i(k)$ represents the probability that a node with degree k points to the infected or immune node. The differential equation of heterogeneous networks can be obtained as follows:

$$\begin{cases} \dfrac{dS_k(t)}{dt} = \mu\rho - S_k(t)\left(\alpha_1 k\Theta_{I_L}(k) + \alpha_2 k\Theta_{I_H}(k)\right) - \mu S_k(t), \\[2mm] \dfrac{dE_k(t)}{dt} = \mu(1-\rho) + S_k(t)\left(\alpha_1 k\Theta_{I_L}(k) + \alpha_2 k\Theta_{I_H}(k)\right) - E_k(t)(v + P_1 + P_2) - \mu E_k(t), \\[2mm] \dfrac{dI_{Lk}(t)}{dt} = E_k(t)P_1 + t_2 I_{Hk}(t) - t_1 k I_{Lk}(t)\Theta_{I_H}(k) - r_1 k I_{Lk}(t)\Theta_R(k) - \mu I_{Lk}(t), \\[2mm] \dfrac{dI_{Hk}(t)}{dt} = E_k(t)P_2 + t_1 I_{Lk}(t) - t_2 k I_{Hk}(t)\Theta_{I_L}(k) - r_2 k I_{Hk}(t)\Theta_R(k) - \mu I_{Hk}(t), \\[2mm] \dfrac{dR_k(t)}{dt} = E_k(t)v + t_1 k I_{Lk}(t)\Theta_{I_H}(k) + r_1 k I_{Lk}(t)\Theta_R(k) + t_2 k I_{Hk}(t)\Theta_{I_L}(k) + r_2 k I_{Hk}(t)\Theta_R(k), \\[2mm] \qquad - t_2 I_{Lk}(t) - t_2 I_{Hk}(t) - \mu R_k(t), \end{cases} \tag{41}$$

4. Equilibrium Analysis

4.1. Homogeneous Networks. For the real world, the equation satisfied the conditions that $S(t) \geq 0$, $E(t) \geq 0$, $I_L(t) \geq 0$, $I_H(t) \geq 0$, $R(t) \geq 0$. Let the equation equals to 0. We can calculate the disease-free equilibrium point $E_0(\rho, 1-\rho, 0, 0, 0)$. According to the next-generation matrix method, we can obtain as follows:

TABLE 3: Definition of node state transition probability.

Transition probability	Description
P_{SS}^i	The probability that node i remains inactive state
P_{SE}^i	The probability that node i transfers from inactive state to latent state
P_{EE}^i	The probability that node i remains latent state
$P_{EI_L}^i$	The probability that node i transfers from latent state to L propagation state
$P_{EI_H}^i$	The probability that node i transfers from latent state to H propagation state
P_{ER}^i	The probability that node i transfers from latent state to immune state
$P_{I_L I_L}^i$	The probability that node i remains L propagation state
$P_{I_L I_H}^i$	The probability that node i transfers from L propagation state to H propagation state
$P_{I_L R}^i$	The probability that node i transfers from L propagation state to immune state
$P_{I_H I_H}^i$	The probability that node i remains H propagation state
$P_{I_H I_L}^i$	The probability that node i transfers from H propagation state to L propagation state
$P_{I_H R}^i$	The probability that node i transfers from high propagation state to immune state

$$F = \begin{bmatrix} 0 & \alpha_1 k\rho & \alpha_2 k\rho \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} v + P_1 + P_2 + \mu & 0 & 0 \\ -P_1 & \mu & -t_2 \\ -P_2 & -t_1 & \mu \end{bmatrix}. \tag{42}$$

The basic reproductive number R_0 is equal to the spectral radius $\rho(FV^{-1})$ of the next-generation matrix, that is, the maximum value of the eigenvalue modulus of the next-generation matrix V^{-1}; hence,

$$R_0 = \frac{\alpha_1 k\rho(\mu P_1 + t_2 P_2) + \alpha_2 k\rho(\mu P_2 + t_1 P_1)}{(t_1 t_2 - \mu^2)(v + P_1 + P_2 + \mu)}, \tag{43}$$

Theorem 1. *If $0 < R_0 < 1$, the system is asymptotically stable at the disease-free equilibrium point.*

Proof. The Jacobi matrix of the system at the disease-free equilibrium point is as follows:

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\alpha_1 k\rho & -\alpha_2 k\rho \\ 0 & -(v + P_1 + P_2 + \mu) & \alpha_1 k\rho & \alpha_2 k\rho \\ 0 & P_1 & -\mu & t_2 \\ 0 & P_2 & t_1 & -\mu \end{bmatrix}. \tag{44}$$

We can calculate the eigenvalue of the Jacobi matrix, that is,

$$\begin{cases} \lambda_1 = -\mu, \\ \lambda_2 = M - \dfrac{A}{B} - B, \\ \lambda_3 = M - \dfrac{A}{(-(1/2) + (\sqrt{3}/2)i)B} - \left(-\dfrac{1}{2} + \dfrac{\sqrt{3}}{2}i\right)B, \\ \lambda_4 = M - \dfrac{A}{(-(1/2) + (\sqrt{3}/2)i)B} - \left(-\dfrac{1}{2} - \dfrac{\sqrt{3}}{2}i\right)B, \end{cases} \tag{45}$$

where

$$\begin{cases} M = -\mu - \dfrac{1}{3}(v + P_1 + P_2), \\ A = 3(k\rho\alpha_1 P_1 + k\rho\alpha_2 P_2 + t_1 t_2) + (v + P_1 + P_2)^2, \\ B = \dfrac{1}{3}\left(-\dfrac{27}{2}C + \dfrac{1}{2}\sqrt{D} + (3\mu + v + P_1 + P_2)^3 + \dfrac{3}{2}(3\mu + v + P_1 + P_2)\left(A - (3\mu + v + P_1 + P_2)^2\right)\right)^3, \\ C = \alpha_1 k\rho(\mu P_1 + t_2 P_2) + \alpha_2 k\rho(\mu P_2 + t_1 P_1) - (t_1 t_2 - \mu^2)(v + P_1 + P_2 + \mu), \\ D = -4A^3 + \left(-\dfrac{27}{2}C + 2(3\mu + v + P_1 + P_2)^3 - 3(3\mu + v + P_1 + P_2)\left(A - (3\mu + v + P_1 + P_2)^2\right)\right)^2, \end{cases} \tag{46}$$

We can easily obtain that $\lambda_1, \lambda_2 < 0$. When $0 < R_0 < 1$, $\lambda_3, \lambda_4 < 0$. Therefore, all the eigenvalues of the Jacobi matrix $J(E_0)$ are less than 0. According to the Lyapunov criterion, the system is asymptotically stable at the disease-free equilibrium point.

4.2. Heterogeneous Networks. The same as the homogeneous networks, the disease-free equilibrium point is $E_0(\rho, 1 - \rho, 0, 0, 0)$,

Theorem 2. *In a heterogeneous network, the basic reproductive number* R_0 *is related to the topological structure of the network.*

Proof. For the latent nodes,

$$\frac{dE_k(t)}{dt} = \mu(1 - \rho) + S_k(t)\left(\alpha_1 k \Theta_{I_L}(k) + \alpha_2 k \Theta_{I_H}(k)\right)$$ (47)

$$-E_k(t)(\nu + P_1 + P_2) - \mu E_k(t),$$

when $E_k = 0$, the Jacobi matrix is $L = \{l_{kk'}\}$ at $k = 1, 2, \cdots, n$.

$$l_{kk'} = -\delta_{kk'}(\nu + P_1 + P_2 + \mu) + (\alpha_1 k \rho + \alpha_2 k \rho) P(k'|k).$$ (48)

where $\delta_{kk'} = \begin{cases} 1, k = k' \\ 0, k \neq k' \end{cases}$ *is the Kronecker Delta function. If* all the eigenvalues of the matrix L are less than 0, the system is locally and asymptotically stable at $E_k = 0$; we define the connection matrix $C = \{c_{kk'}\}$, $c_{kk'} = kP(k'|k)$. Let the largest eigenvalue of the matrix C be Λ_m. Then, the largest eigenvalue of the matrix L is $-(\nu + P_1 + P_2 + \mu) + (\alpha_1 \rho + \alpha_2 \rho)\Lambda_m < 0$.

Similarly, for $I_{Lk} = 0$, $I_{Hk} = 0$, there exist $-\mu - (\theta_1 + \beta_1)\Lambda_m < 0$ and $-\mu - (\theta_2 + \beta_2)\Lambda_m < 0$, respectively. Therefore, the system is locally and asymptotically stable at $I_{Lk} = 0$ and $I_{Hk} = 0$, and we can get the basic reproductive number R_0 for heterogeneous networks.

$$R_0 = \frac{(\alpha_1 \rho + \alpha_2 \rho)}{(\nu + P_1 + P_2 + \mu)}\Lambda_m.$$ (49)

5. Simulation Results

In this section, we study the propagation dynamic characteristics by simulating a complex network. We apply the proposed model to an artificial network and to real-world networks to present the information propagation process and verify the rationality of the proposed model.

5.1. Homogeneous Networks. In homogeneous networks, we use the Runge–Kutta method to investigate the dynamics of the SELHR model on the Watts–Strogatz network [43]. The size of the WS network is $N = 10000$, and the initial settings are as follows: $\alpha_1 = 0.3$, $\alpha_2 = 0.1$, $P_1 = P_2 = 0.05$, $\nu = 0.1$, $t_1 = t_2 = 0.05$, $r_1 = 0.05$, $r_2 = 0.01$, and the $\langle k \rangle$ is 10. All parameters are set to satisfy the system's stability. In the beginning, the number of susceptible nodes is 9998 and the
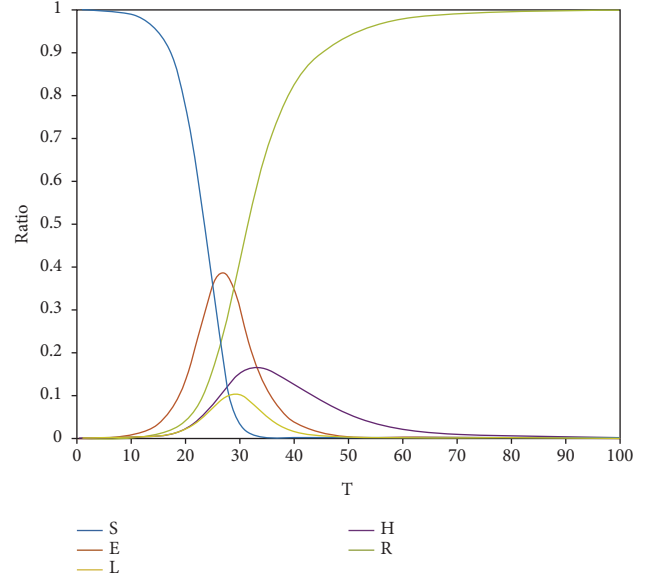

FIGURE 2: The evolution process of groups in different states (homogeneous network).
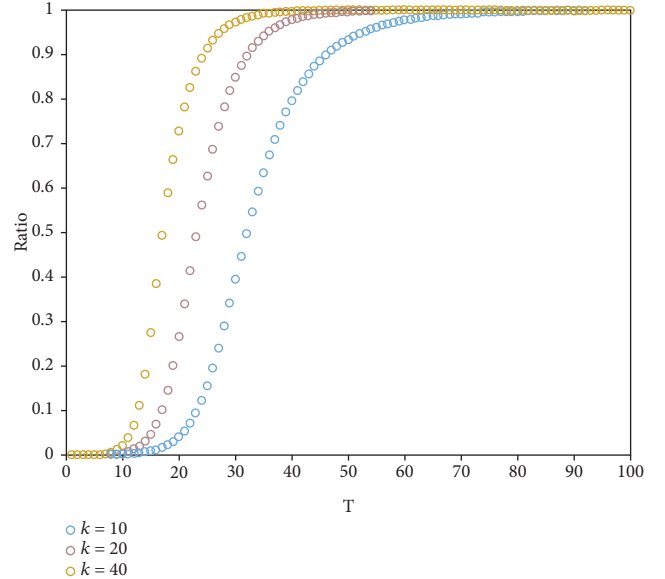

FIGURE 3: Influence of different k on information propagation (homogeneous network).

propagation states L and H have one node for each. In terms of population entry rate and population exit rate μ, according to the 2019 population survey of India, the annual population growth rate of India is 1.01%, which is regarded as the population growth rate of 1.01%. [44] The value of the entrance entry rate and the population exit rate is a reference, so the value is $\mu = \sqrt[365]{1.01} - 1 \approx 0.000027$.

5.1.1. Densities of Different Nodes over Time. There are five states in the proposed model. The changes in the five states over time are presented in Figure 2. With the propagation of information, the ratio decreases deeply to 0. On the contrary,
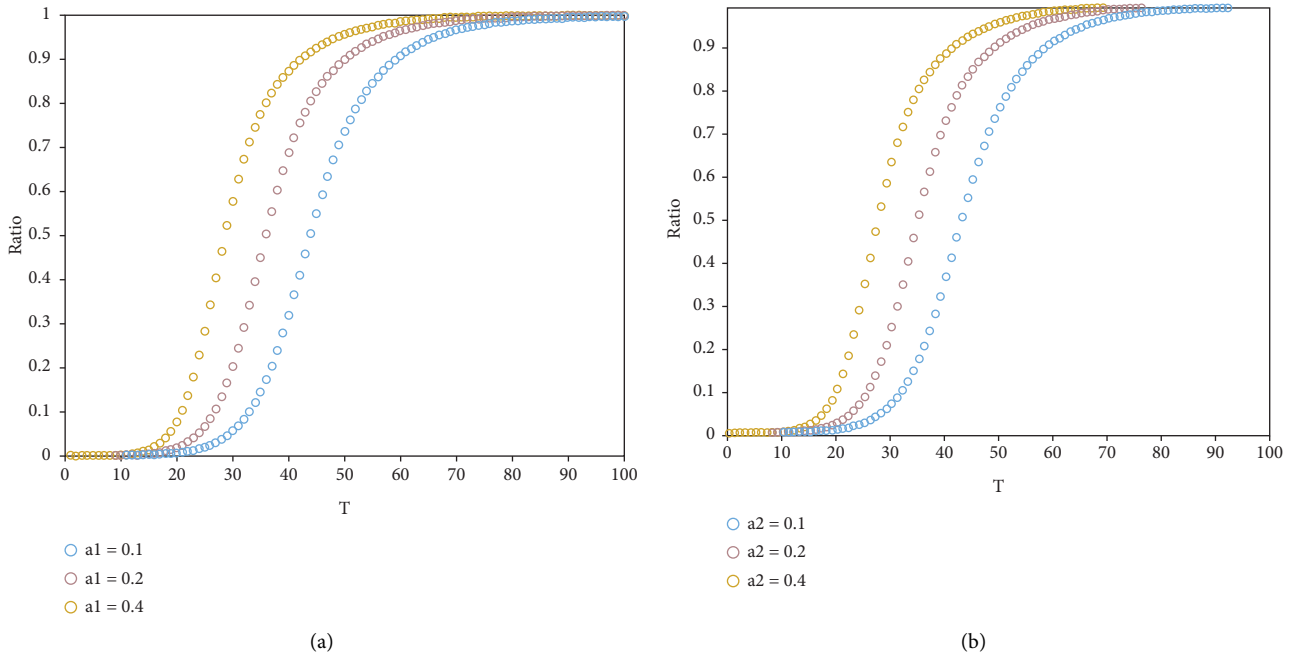
(a)

(b)

FIGURE 4: Influence of infection rate on information propagation (homogeneous network).
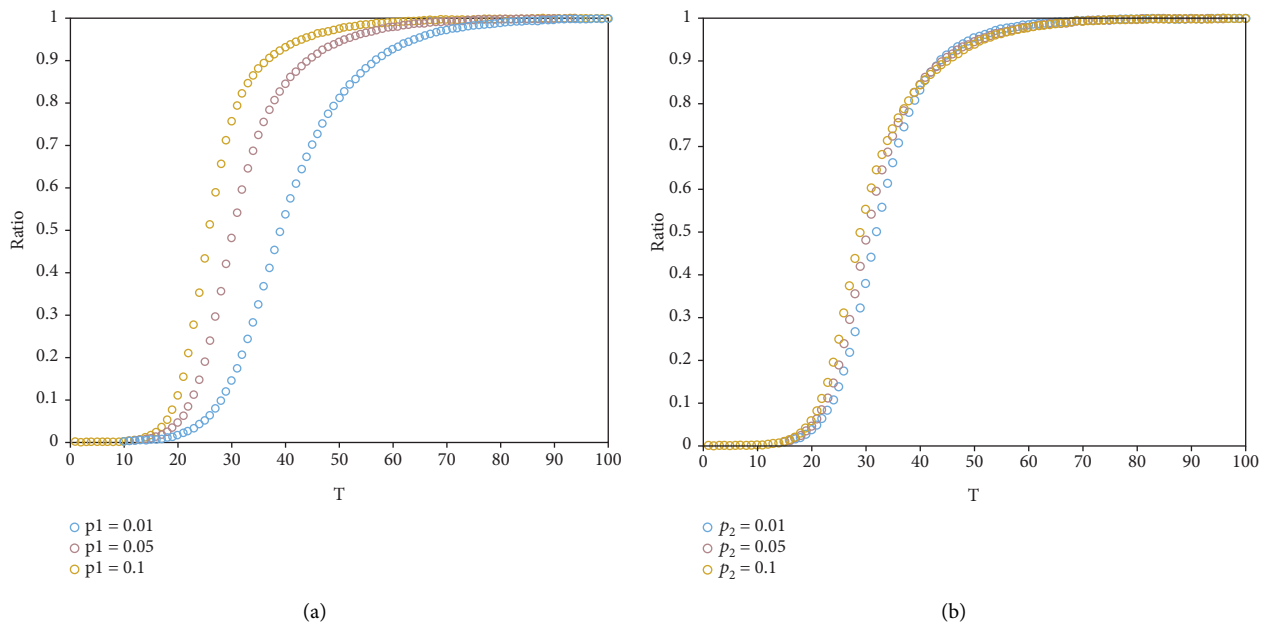

(a)

(b)

FIGURE 5: Influence of exposure rate on information propagation (homogeneous network).

the ratio of immune nodes increases slowly at the beginning and then increases quickly and reaches a stable state. However, due to the attributes of the low-risk propagation nodes and high-risk propagation nodes, the density of low-risk propagation nodes drops to zero at a faster speed during the process.

5.1.2. Influence of Different ⟨k⟩ on Information Propagation.
In homogeneous networks, the average degree decides the speed of information spread. When the total number of

nodes in a network remains constant, the higher the average degree is, the faster the information spreads. When ⟨k⟩ is 10, 20, and 40, respectively, the influence of the ⟨k⟩ value on the number of immune nodes is studied, as shown in Figure 3. Figure 3 shows that the average degree is positively correlated with the number of immune nodes. The higher the average degree value, the faster the information spreads. That is because a node can transmit information to more neighboring nodes if the average degree is higher.
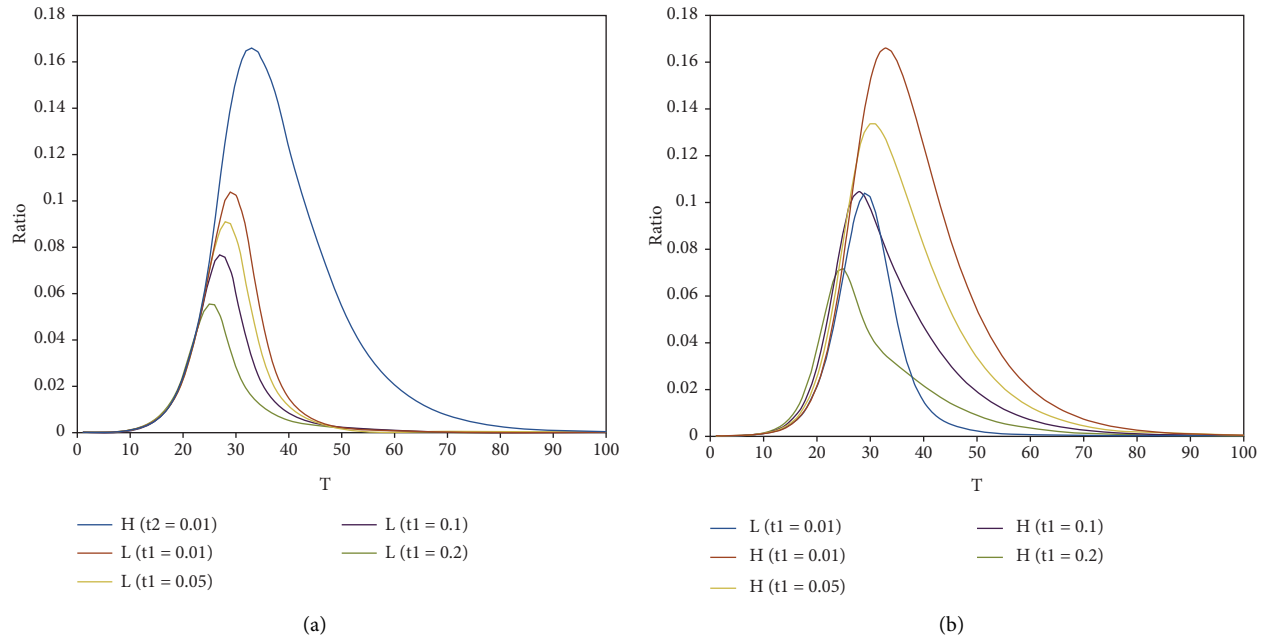
(a)

(b)

FIGURE 6: Influence of transform rate on information propagation (homogeneous network).

5.1.3. Influence of Different Transmission Rates on Information Propagation.
In our proposed information propagation model, there are four types of transmission rates: infection rate α from uninfected nodes to latent nodes, exposure rate p from latent nodes to propagation nodes (nodes with high propagation ability or low propagation ability), transform rate t between two types of propagation nodes and recovery rate r from propagation nodes to immune nodes. In Figure 4, we present the final immune node rate for three different values of α_1 and α_2, where we set $\alpha_1 = 0.1, 0.2, 0.4, \alpha_2 = 0.1, 0.2, 0.4$, and other parameters are set as the initial. With the increasing of the infection rate, nodes are more likely to be infected and the earlier the system becomes stable. Figure 5 shows the general trends of the immune node ratio under different values of p_1 and p_2, where we set $p_1 = 0.01, 0.05, 0.1, p_2 = 0.01, 0.05, 0.1$.

In general, the higher the exposure rate, the faster the immune nodes increase. However, the difference in immune node trend among different values of p_2 was slightly compared with the exposure rate p_1. That is the transmission rate from latent nodes to highpropagation ability nodes has little influence on the evolution of the immune nodes. For one reason, the number of high propagation ability nodes is smaller than the low propagation ability nodes. For another, high propagation ability nodes and lowpropagation ability nodes can be transformed from each other. Figure 6 describes how the transform rate affects the evolution of the high propagation ability nodes and the low propagation ability nodes. When $t_2 = 0.01$, we set $t_1 = 0.01, 0.05, 0.1, 0.2$, and when $t_1 = 0.01$, we set $t_2 = 0.01, 0.05, 0.1, 0.2$. From Figure 6, we can see that with the increasing of the transform rate t_1, the peak of the lowpropagation ability nodes ratio gradually becomes lower. That is because more and more low propagation ability nodes are being transformed into high propagation ability nodes. Similarly, the trends of

different high propagation ability nodes can also indicate that the higher the transform rate t_2 is, the faster the high propagation ability nodes transform into low propagation ability nodes. Figure 7 illustrates how different recovery rates affect the evolution of immune nodes, where we set $r_1 = 0.01, 0.1, 0.2, r_2 = 0.01, 0.1, 0.2$. When $r_1 = 0.01$ and $r_2 = 0.01$, the corresponding trends of immune nodes have a lower slope than $r_1 = 0.1, 0.2$, and $r_2 = 0.1, 0.2$. However, when $r_1 = 0.1$, the system stabilizes faster than $r_1 = 0.2$. That is because the neighbors of a propagation node have different states. The transformation from propagation nodes to immune nodes is not only decided by a single immune node.

5.2. Heterogeneous Networks.
In heterogeneous networks, we investigate the dynamics of the SELHR model on an interaction network—Ia-fb-messages [45]. The dataset includes the users who sent or received at least one message. The number of nodes and edges of the Ia-fb-messages network is $n = 1266, m = 6451$. The average degree $\Box k \Box$ is 10 and the clustering coefficient C is 0.0683. According to the structure features of Ia-fb-messages, the initial settings are as follows: $\alpha_1 = 0.002, \alpha_2 = 0.001, P_1 = 0.3, P_2 = 0.1, v = 0.01, t_1 = t_2 = 0.05, r_1 = 0.05, r_2 = 0.01$. In the beginning, the number of susceptible nodes is 1262 and the propagation states L and H are nodes {2,3}, {4,6} for each. In terms of population entry rate and population exit rate μ, μ is too small to have an effect on the dynamics of the Ia-fb-messages network. Therefore, we neglect the influence of the population entry rate and population exit rate during the simulation process.

5.2.1. Densities of Different Nodes over Time.
Figure 8 describes the evolution of five states in the Ia-fb-messages network. The evolution curves have a similar dynamic
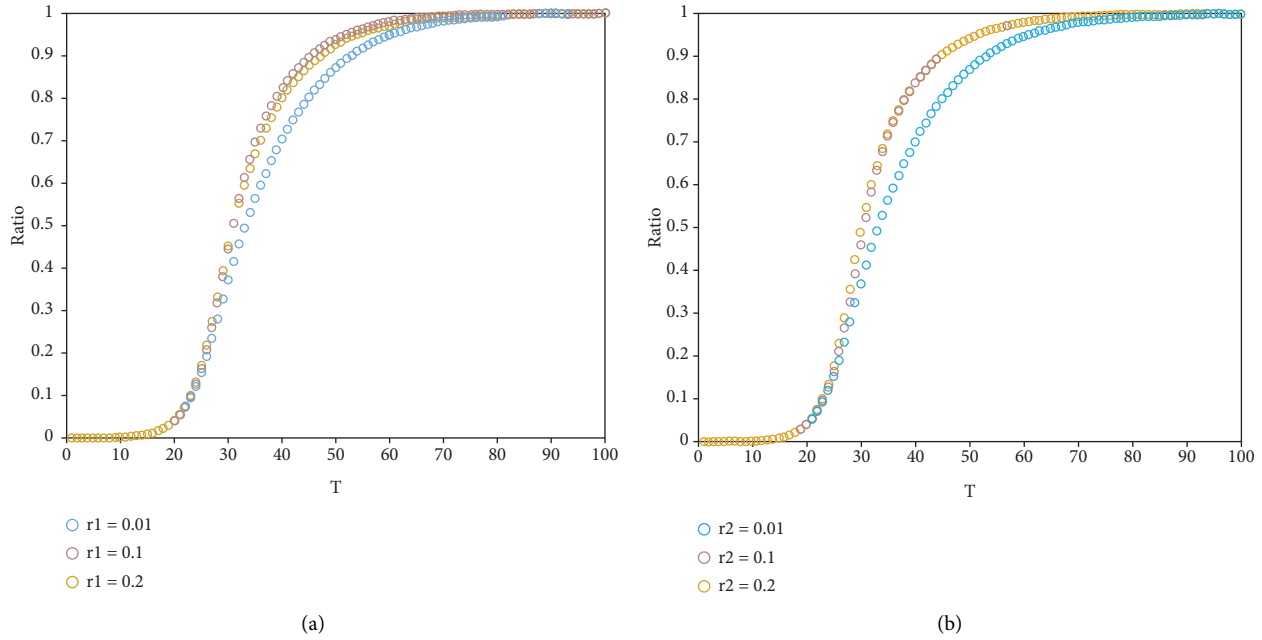
(a)

(b)

FIGURE 7: Influence of recovery rate on information propagation (homogeneous network).
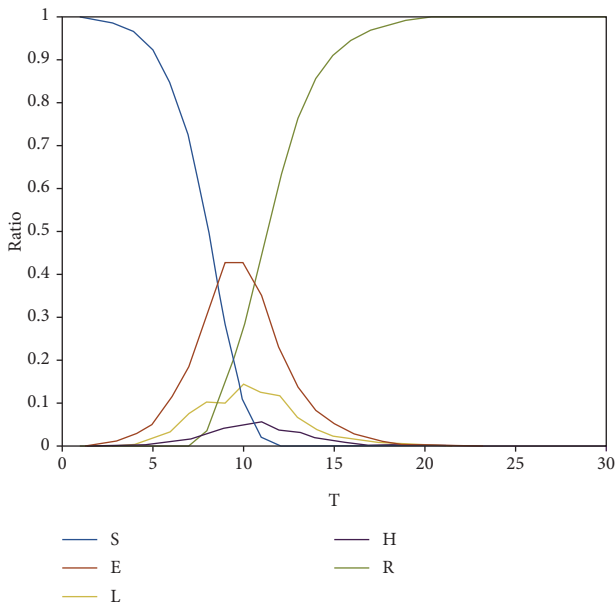

FIGURE 8: The evolution process of groups in different states (heterogeneous network).

FIGURE 9: Degree distribution $P(k)$ of Ia-fb-messages network.

tendency as the simulated results in Figure 2. In Figure 9, we plot the degree distribution of the Ia-fb-messages network. Obviously, $P(k)$ depicted in the log scale exhibits a power-law form and, thus, this network can be considered a heterogeneous network.

5.2.2. Influence of Different Transmission Rates on Information Propagation. In heterogeneous networks, the transmission rates are set according to the basic
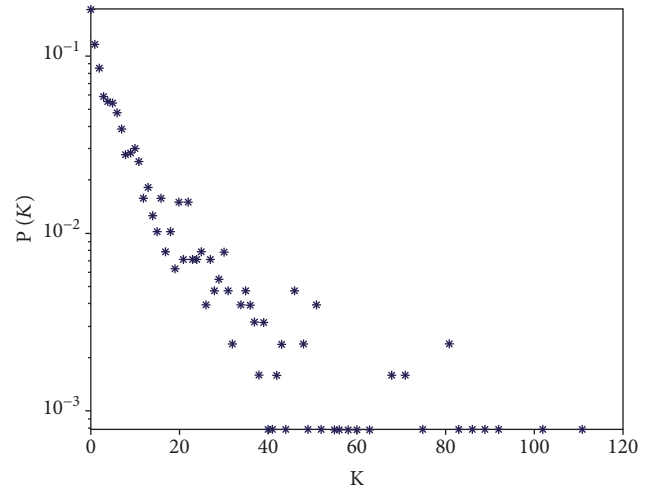
reproduction number. Figure 10 displays the immune node ratio for four different values of α_1 and α_2, where we set $\alpha_1 = 0.001, 0.002, 0.005, 0.01\alpha_2 = 0.001, 0.002, 0.005, 0.01$. The evolution curves are similar to those in homogeneous networks. The higher the infection rate is, the faster the spread of information, and the wider the spread of information. Figure 11 shows the immune nodes ratio for three different values of p_1 and p_2, where we set $p_1 = 0.1, 0.2, 0.5$, $p_2 = 0.1, 0.2, 0.5$. Figure 12 describes how the transform rate affects the evolution of the highpropagation ability nodes and the low propagation ability nodes in heterogeneous networks. When $t_2 = 0.05$, we set $t_1 = 0.01, 0.02, 0.05$, and when $t_1 = 0.05$, we set $t_2 = 0.01, 0.02, 0.05$. The curves of the transform rate in heterogeneous networks are not as smooth
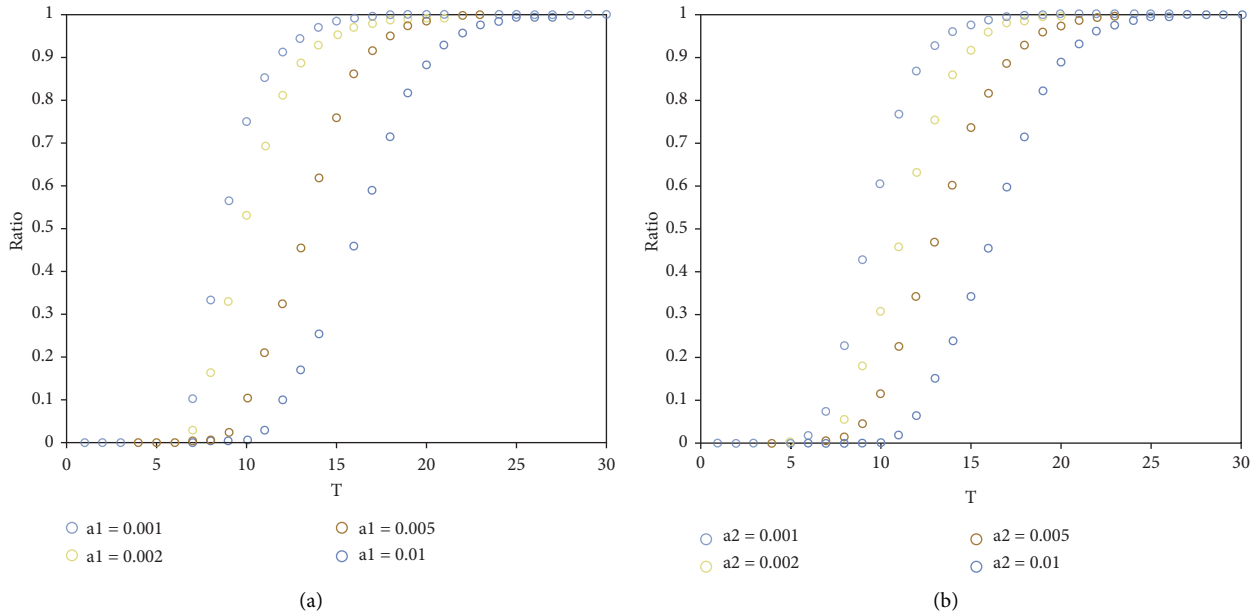
FIGURE 10: Influence of infection rate on information propagation (heterogeneous network).
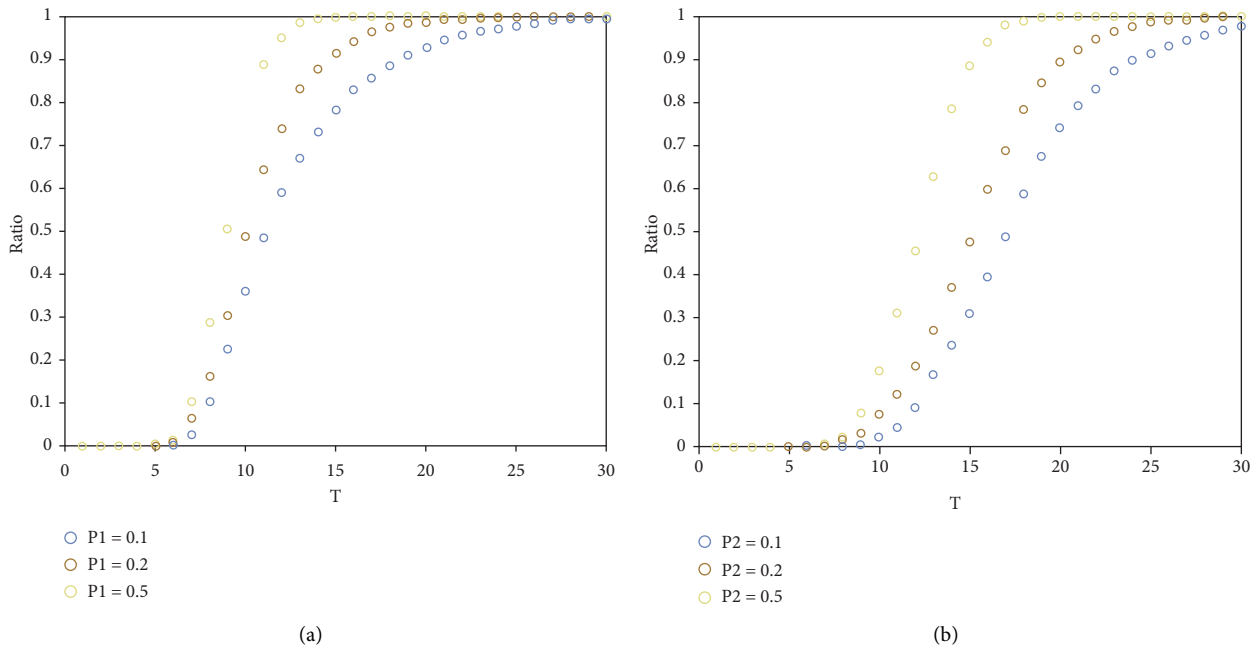

FIGURE 11: Influence of exposure rate on information propagation (heterogeneous network).

as those in homogeneous networks. That is because, in every dynamic turn, the number of propagation nodes converted from each other is decided by the location of the node in a network and the topological structures of a network. Figure 13 illustrates the immune node ratio for three different values of r_1 and r_2, where we set $r_1 = 0.01, 0.02, 0.05,$ $r_2 = 0.01, 0.02, 0.05$. From Figure 13, the curves of immune node ratio are similar under different values of recovery rate,

but they can also reflect the tendency that a high recovery rate will lead to faster propagation of information.

Overall, the correctness of the theoretical deduction is confirmed by sufficient simulations. According to the simulations for both homogeneous networks and heterogeneous networks, we verify the rationality of the proposed model and better understand the impacts of network structures.
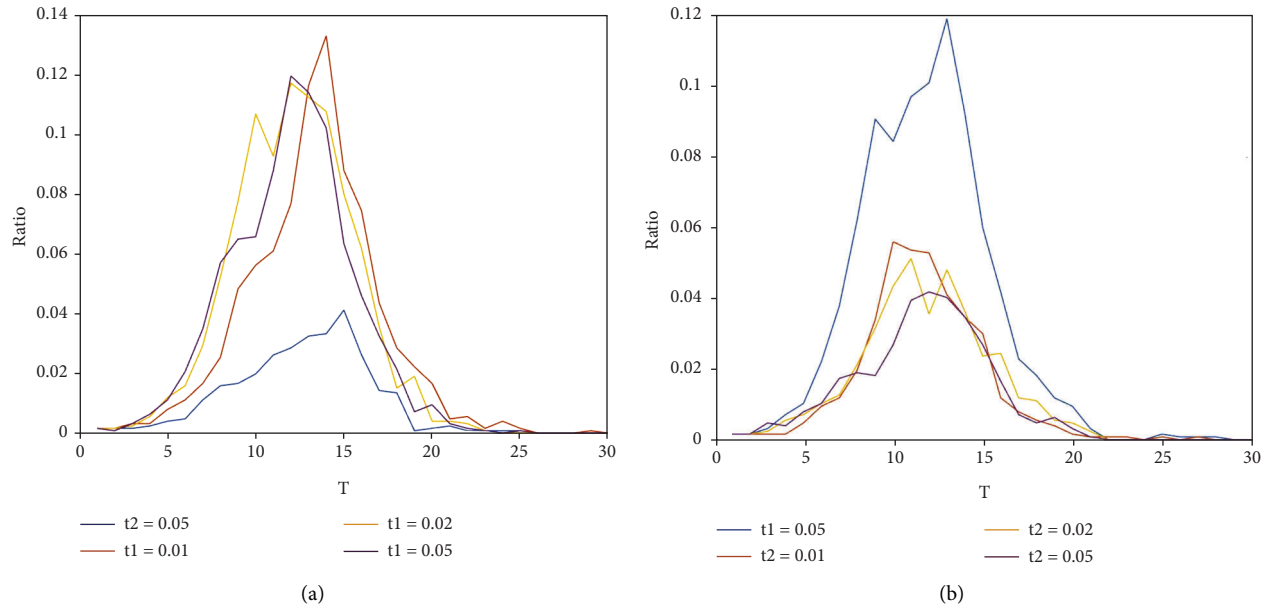
Figure 12: Influence of transform rate on information propagation (heterogeneous network).
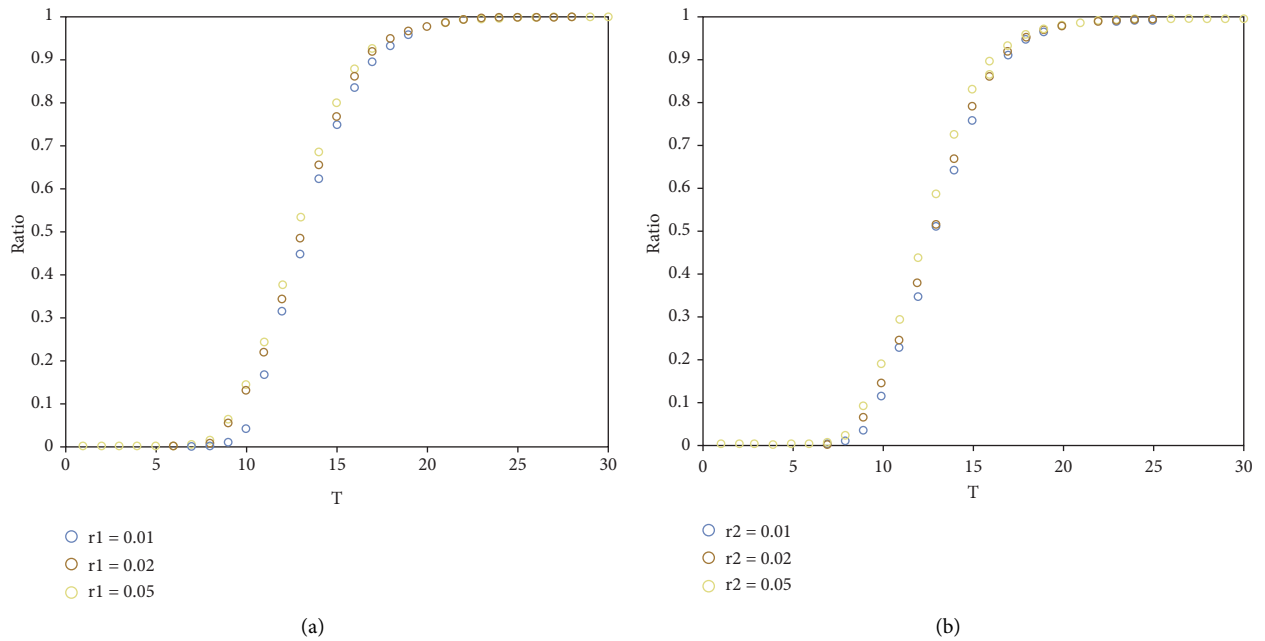

Figure 13: Influence of recovery rate on information propagation (heterogeneous network).

6. Conclusion

In summary, our work is mainly focused on the dynamics of information spreading on a complex social network. First, based on the classic SEIR model, we propose a new epidemic model, SELHR. Then, we construct the dynamic evolution equation of information propagation and analyze the equilibrium point and stability of the model from a dynamic perspective. In addition, simulations are carried out both on homogeneous networks and heterogeneous networks at the

epidemic equilibrium points to verify the rationality and validity of the proposed model. We conducted a sensitivity analysis of model parameters for the basic reproduction number. In particular, the heterogeneity of the network structure makes it significant in disease propagation. Different from the previous epidemic models, our model considers the two types of propagation nodes (L and H) and adds the transformed rate between these two types. The influence of key parameters on information propagation mainly reflects the speed of propagation and steady-state

densities of individuals. It reveals that considering the transformation of two propagation states, the dynamic behavior of information propagation is more realistic.

The next step is to further verify the feasibility of the proposed model. We will consider applying it to other information propagation studies in complex networks, like link prediction, influence maximization, identification of influential nodes, and so on. More specifically, driven by the practical significance of epidemic models, we desire to identify the super spreader to curb the spread of the disease. In our future work, we will seek to establish another epidemic model referring to two different social networks for disease and information spreading.

Data Availability

The network data used to support the findings of this study have been deposited in https://networkrepository.com/ia-crime-moreno.php.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

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