AN SICR RUMOR SPREADING MODEL IN HETEROGENEOUS NETWORKS

JINXIAN LI*
School of Mathematical Sciences, Shanxi University, Taiyuan 030006, China
Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention
Taiyuan 030006, China

NING REN
School of Mathematical Sciences, Shanxi University, Taiyuan 030006, China

ZHEN JIN
Complex System Research Center, Shanxi University, Taiyuan 030006, China
Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention
Taiyuan 030006, China

(Communicated by Hao Wang)

ABSTRACT. This article discusses the spread of rumors in heterogeneous networks. Using the probability generating function method and the approximation theory, we establish an SICR rumor model and calculate the threshold conditions for the outbreak of the rumor. We also compare the speed of the rumors spreading with different initial conditions. The numerical simulations of the SICR model in this paper fit well with the stochastic simulations, which means that the model is reliable. Moreover the effects of the parameters in the model on the transmission of rumors are studied numerically.

1. Introduction. Since ancient times, a rumor is an important form of social interaction. With the development of the times and the progress of science and technology, rumors spread faster and wider. Sometimes rumors may play a positive role, for example, leading people to notice something [16]. However most rumors induce damage [3]. We do not want to see the panic and losses caused by rumors. Therefore, the characteristics of the process of rumor dissemination become a very important research topic.

The study of the rumor models began in the 1960s with the work of Daley and Kendall [2]. They established the classical DK rumor spreading model based on SIR epidemic model. In this model, people were divided into three groups: people who never heard the rumor, people who know the rumor and spread it, people who know the rumor but never spread it. Since then, this model has been widely extended. Moreno et al. [11] derived the mean-field equations characterizing the

2010 Mathematics Subject Classification. Primary: 05C80, 37N25; Secondary: 92D25.
Key words and phrases. Rumor spreading, SICR model, heterogeneous networks, counterattack, probability generating function.

* Corresponding author: lijinxian@sxu.edu.cn.
dynamics of a rumor process in a heterogeneous network. Wang et al. [21] proposed a novel susceptible-infected-removed (SIR) model by introducing the trust mechanism and analyzed the critical threshold and the final size. Qian et al. [14] described the phenomenon that individuals could obtain rumors from different sources like online social networks rather than only getting them from neighbors by adopting independent spreaders in classic ignorant-spreaders-stiflers (SIR) model. Zhu and Wang [28] took the variation of connectivity into consideration and assumed the variation as noise which was described as standard Brownian motion. Based on it, they presented a modified susceptible-infected-removed (SIR) model to explore rumor diffusion in complex social networks. Ma et al. [9] introduced the independent spreaders in a susceptible-infectious-recovered contagion process to overcome the negligence of the possible interactions between nodes beyond the underlying network.

In order to refine the process of rumor propagation, more compartments are introduced into rumor spreading model. Huo et al. [4] established a dynamic model for the rumor spreading called $I_2SR$, in which he considered the activity of nodes and divided spreaders into spreaders with the high rate of the active state and the low rate of active spreaders. More than one rumor spreading in the complex internet at the same time has been studied in [19, 5]. Zhao et al. [27, 26] developed a new rumor spreading model: susceptible-infected-hibernator-removed (SIHR) model with forgetting and remembering mechanisms in homogeneous networks and inhomogeneous networks. Wang et al. [20] thought that a person could decide whether to accept the rumor and to continue to spread it or not when he heard about the rumor. So Wang et al. established $SIRaRu$ rumor spreading model in which classified stiflers divided into two groups: Ra and Ru. Ra and Ru expressed respectively the people who accepted the rumor but lost interest to spread it and who did not accept the rumor at all. Xia et al. [22] considered that some people would become an exposed state (hesitating state) after knowing a rumor topic, instead of spreading the rumor immediately. So Xia et al. proposed a modified susceptible-exposed-infected-removed (SEIR) model. Considering that the exposed nodes may become the removed nodes at a rate, Liu et al. [8] presented a novel rumor propagation SEIR model in heterogeneous network. Since there may be some people do not agree with the rumor and they may persuade others to resist the rumor propagation, Wan et al. [18] presented a susceptible-infected-counter-susceptible (SICS) rumor spreading model with immigration and emigration. In [18], people who had an ambiguous attitude about the rumor were collectively called susceptible individuals. Zan et al. [23] distinguished the susceptible into the unknown people and the silent people. Zan et al. established the susceptible-infective-counterattack-refractory (SICR) rumor spreading model in homogeneous network. In this paper, the unknown people and the silent people were called the susceptible and the refractory, respectively.

Furthermore, the importance of network structure to rumor spreading is revealed by Lefevre and Picard [6] in 1994. The topology of social networks shows highly complex connectivity in which each has a random number of connections to other individuals [13]. A series of simulations in both static and small-world networks are done to show the existence of a critical threshold of rumor spreading by Zanette [24, 25] and Buzna et al. [1]. So the influence of network structure on rumor spreading has been paid more and more attention. Nekovee et al. [12] extended the former dynamic model to scale-free networks with assortative degree correlations.
and have noticed the effect of degree correlation on rumor spreading. Roshani and Naimi [15] introduced the generalized rumor spreading model and analytically investigate the epidemic spreading for this model on scale-free networks.

This paper also focuses on the impact of network structure on the rumor spreading. Since the models proposed in the above literature are all mean-field equations, they ignore the heterogeneity in the locality of rumor spreading along the edges. So we try to find a better way to describe rumor spreading, rather than mean field theory. As two types of transmission problems in complex networks, rumor spreading has many similarities with disease spreading. Thus, we can learn from the modeling process of disease transmission. Probability generating function method and pairwise approximation method are two classic methods to model the propagation process. Probability generating function (PGF) method was presented by Volz [17] to model the SIR disease spreading in random networks with heterogeneous connectivity. Pairwise approximation method was presented by Matsuda [10] to derive the lattice Lotka-Volterra model with intrinsic birth, death and migration. This paper combines the generating function method with the approximation method. We here follow the rumor spreading mechanism in [23] and establish the SICR model in heterogeneous networks. Then we do some stochastic simulations to confirm the rationality of the model. We also give the threshold and investigate the influences of degree on the speed of rumor spreading.

The remainder of this paper is organized as follows. In Section 2, we establish the SICR rumor spreading model and analyze the threshold. In section 3, we do some simulations to confirm the accuracy of the model. Furthermore, more simulations are done to study the effect of parameters on the spread of the rumor.

2. SICR models in random networks. In this section, we will build an SICR rumor spreading model. Firstly, we introduce the rumor propagation mechanism.

2.1. Rumor propagation mechanism. In this paper, we use the same rumor spreading mechanism in [23] and individuals are divided into four groups: people who never hear the rumor (susceptible); people who hear the rumor and spread it (infective); people who hear the rumor but disagree it (counterattack); people who hear the rumor but have no interest to spread it (refractory). We use $S$, $I$, $C$ and $R$ to denote the fraction of the susceptible, the infective, counterattack and refractory nodes.

The rules of the SICR model are shown in Figure 1. Moreover, the dynamics of the SICR model can be summarized as follows: (1)When a susceptible node contacts an infective node, the susceptible node may become a new infective node with probability $\alpha$; may become a new refractory node with probability $\beta$; may become a new counterattack node with probability $\delta$. (2)When an infective node contacts a refractory node or another infective node, the infective node will become a new refractory node with probability $\gamma$. (3)When an infective node contacts a counterattack node which refutes the rumor, the infective node will become a new refractory node with probability $\eta$.

2.2. Complex networks. To model the above rumor spreading progress in a heterogeneous network, we consider a closed and mixed population $G = (V, E)$ with $N$ individuals, where $V = \{v_1, v_2, \cdots, v_N\}$ is the set of vertices representing the individuals and $E$ is the set of edges representing the connections between the individuals. We also assume that contacts are symmetric, that is, if an edge $(v_1, v_2) \in E$
connects $v_1$ to $v_2$, then an edge also connects $v_2$ to $v_1$. Although the network is undirected (i.e., any two neighboring vertices can spread each other), we wish to keep track of who spread who. Therefore, for each edge $(v_1, v_2) \in E$, we define two arcs as the ordered pairs $(v_1, v_2)$ and $(v_2, v_1)$. The first and second elements in the ordered pair $(v_1, v_2)$ are frequently called the ego and the alter, respectively [17]. Let $p_k$ be the degree distribution. Then $p(k)$ is the proportion of nodes having $k$ network contacts, which can reveal the structure of this network. Then the probability generating functions (PGFs) of the degree distribution is given by

$$g(x) = \sum_k p_k x^k.$$  

Let $\bar{k}$ and $\bar{k}^2$ be the averaged degree and averaged second-moment. Then they are $g'(1)$ and $g''(1) + g'(1)$, respectively.

For example, for a poisson degree distribution $p_k = \frac{\lambda^k e^{-\lambda}}{k!}$, $k = 1, 2, \cdots$, the corresponding PGF is

$$g(x) = e^{\lambda(x-1)}.$$  

Furthermore, the averaged degree and averaged second-moment are

$$\bar{k} = \lambda, \quad \bar{k}^2 = \lambda^2 + \lambda,$$

respectively.

2.3. Our model. In this section, we will build an SICR rumor spreading model using methods of probability generating function (PGF) and pair approximations.

Since the network is heterogeneous, we use $S_k(t)$, $I_k(t)$, $C_k(t)$ and $R_k(t)$ to denote the density of the susceptible, the infective, counterattack and refractory with degree $k$ at time $t$ in the set of nodes with degree $k$. In addition, they satisfy the normalization condition: $S_k(t) + I_k(t) + C_k(t) + R_k(t) = 1$. Our model is an extension of the SIR model in [17], we also need some notations in [17]. Let $\theta$ represent the fraction of the nodes with degree one that remain ignorant at time $t$, and $P_A^B$ represent the probability that an arc with an ego in state $A$ has an alter in state $B$, where $A$ and $B$ may be $S$, $I$, $C$ or $R$. And $M_{AB}$ is the fraction in the set of the arcs with an ego in state $A$ and an alter in state $B$, where $A$ and $B$ may be $S$, $I$, $C$ or $R$. Given $\theta$, it is easy to determine the fraction of nodes which remain susceptible at time $t$, i.e.,

$$S = p_0 + p_1 \theta + p_2 \theta^2 + \cdots = g(\theta).$$

The summary of the nonlinear differential equations of SICR model can be described as follows.
\[
\begin{align*}
\dot{\theta} &= - (\alpha + \beta + \delta) P_S^I \theta; \\
P_S^I &= - \frac{P_S^I \sum_k k(k-1) I_k p_k \{\gamma \sum_k k I_k p_k - P_S^I \theta g'(\theta)\} + (\eta - \gamma) g'(1) M_{IC}}{(\sum_k k I_k p_k)^2} \\
&\quad + \frac{\alpha P_S^I P_S^C \theta g''(\theta)}{g'(\theta)} - (\alpha + \beta + \delta) P_S^I (1 - P_S^I); \\
P_S^C &= \frac{k P_C^I P_S^C \theta g'(\theta)}{g'(\theta)} + (\alpha + \beta + \delta) P_S^C P_S^I; \\
P_S^C &= \frac{(\alpha + \beta + \delta) P_C^I P_S^C [g'(\theta) - \theta g''(\theta)]}{g'(\theta)}; \\
M_{IC} &= \frac{\delta P_S^I \theta g'(\theta) + \theta^2 P_S^C \theta g'(\theta)(\alpha P_C^I + \delta P_C^I)}{g'(1)} - \frac{\eta g'(1)(M_{IC})^2 \sum_k k(k-1) I_k p_k}{(\sum_k k I_k p_k)^2} \\
&\quad - \eta M_{IC} - \frac{\gamma \sum_k k(k-1) I_k p_k M_{IC}}{\sum_k k I_k p_k} (1 - \frac{P_S^I \theta g'(\theta) + g'(1) M_{IC}}{\sum_k k I_k p_k}); \\
\dot{I}_k &= k \alpha \theta^k P_S^I - \frac{k \gamma I_k [\sum_k k I_k p_k - P_S^I \theta g'(\theta)] + k(\eta - \gamma) I_k g'(1) M_{IC}}{\sum_k k I_k p_k}. \\
\end{align*}
\]

(1)

The modeling process is given in detail in Appendix.

2.3.1. Initial conditions. If the probability of a node with degree \( k \) selected as an initially infected is \( \epsilon_k \), we have the following initial conditions:

1. \( I_k = \frac{\epsilon_k N_k}{N} = \epsilon_k, \quad M_I = \frac{\sum_k k p_k \epsilon_k}{\sum_k k p_k}, \quad M_{SI} \approx M_I = \frac{\sum_k k p_k \epsilon_k}{\sum_k k p_k}; \)
2. \( M_S = 1 - M_{SI} = 1 - \frac{\sum_k k p_k \epsilon_k}{\sum_k k p_k}, \quad M_{SS} = M_S - M_{SI} = 1 - \frac{2 \sum_k k p_k \epsilon_k}{\sum_k k p_k}; \)
3. \( P_S^I(t=0) = \frac{M_{SI}}{M_S} = \frac{\sum_k k p_k \epsilon_k}{\sum_k k p_k - \sum_k k p_k \epsilon_k}, \quad P_S^C(t=0) = \frac{M_{SS}}{M_S} = \frac{\sum_k k p_k - 2 \sum_k k p_k \epsilon_k}{\sum_k k p_k - \sum_k k p_k \epsilon_k}; \)
4. \( M_{IC} = 0; \)
5. \( \theta \approx 1. \)

2.3.2. Threshold. We will analyze the threshold which means the critical value. Below a threshold, the rumor will die out. And above it, the rumor spreading occur. The number of nodes which change from the susceptible to the infective in a small time interval is proportional to \( P_S^I \). Therefor, if \( P_S^I(t=0) < 0 \), the rumor will become extinct without reaching a fraction of the population.

Applying the initial conditions above into \( P_S^I \) and considering \( \epsilon_k \ll 1 \) gives

\[
\begin{align*}
P_S^I(t=0) &= \frac{\alpha \theta^\prime(\theta)}{g'(\theta)} \cdot \frac{\sum_k k p_k \epsilon_k \left( \sum_k k p_k - 2 \sum_k k p_k \epsilon_k \right)}{\left( \sum_k k p_k - \sum_k k p_k \epsilon_k \right)^2} - \frac{\sum_k k(k-1) \epsilon_k p_k}{\left( \sum_k k p_k \epsilon_k \right)^2} \\
&\quad \cdot \frac{\sum_k k p_k \epsilon_k}{\sum_k k p_k - \sum_k k p_k \epsilon_k} \left[ \gamma \sum_k k p_k \epsilon_k - \frac{\gamma \theta^\prime(\theta) \sum_k k p_k \epsilon_k}{\sum_k k p_k - \sum_k k p_k \epsilon_k} + (\eta - \gamma) g'(1) \cdot 0 \right]
\end{align*}
\]

(2)
\begin{align*}
&- (\alpha + \beta + \delta) \sum_k kp_k \epsilon_k \\
&\quad \sum_k kp_k - \sum_k kp_k \epsilon_k \left(1 - \frac{\sum_k kp_k \epsilon_k}{\sum_k kp_k - \sum_k kp_k \epsilon_k}\right) \\
&\approx \left[ \frac{\alpha g''(1)}{g'(1)} - (\alpha + \beta + \delta) \right] \frac{\sum_k kp_k \epsilon_k}{\sum_k kp_k}.
\end{align*}

Then $\dot{P}_S(t = 0) > 0$ leads to

$$\frac{\alpha g''(1)}{g'(1)} > \alpha + \beta + \delta,$$

i.e.,

$$\frac{\alpha}{\beta + \delta} > \frac{g'(1)}{g''(1) - g'(1)}.$$

Therefore the threshold is

$$\left(\frac{\alpha}{\beta + \delta}\right)^* = \frac{g'(1)}{g''(1) - g'(1)}. \quad (3)$$

Next, we take different values for $\epsilon_k$ as $\epsilon_k = \epsilon$, $\epsilon_k = \epsilon^k$, and $\epsilon_k = \epsilon^{1/k}$. Eq. (3) shows that the choice of $\epsilon_k$ does not affect the outbreak of the rumor. However,

$$P_{S|\epsilon_k = \epsilon}^I < \dot{P}_S|\epsilon_k = \epsilon < \dot{P}_S|\epsilon_k = \epsilon^{1/k}.$$

So it will affect the speed of rumor expansion.

3. Simulations. In this section, we carry out random simulations and numerical simulations to verify the above analytical results and further investigate the effects of parameters on the rumor spreading model.

For random simulations, the networks considered here can be generated by the Configuration Model (CM) with $N = 10^4$. If no otherwise specified, the numerical simulations of model (1) are performed with $N = 10^4$ and for convenience, let maximum degree $k_{max} = 30$ in the network. The truncated Poisson degree distribution and the truncated power law degree distribution here are

$$p_k = \begin{cases} 1 - \sum_{k=2}^{30} \frac{\lambda^k e^{-\lambda}}{k!}, & k = 1, \\
\frac{\lambda^k e^{-\lambda}}{k!}, & k = 2, 3, 4, \ldots, 30, \end{cases}$$

and

$$p_k = \begin{cases} 1 - \sum_{k=2}^{30} (43/90)k^{(-1.2)}, & k = 1, \\
(43/90)k^{(-1.2)}, & k = 2, 3, 4, \ldots, 30, \end{cases} \quad (4)$$

respectively.

The initial conditions of the numerical simulations of model obtained by Volz [17] in 2008 is $\epsilon = 10^{-4}$. For convenience, we define the final rumor size that is the fraction of nodes that is infective, counterattack or refractory, i.e., $I + C + R$. 

Figure 2. Simulation with $\alpha = 0.7$, $\beta = 0.1$, $\delta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$. The blue dotted lines correspond to 100 random simulations for an SICR rumor model in a network with the Poisson degree distribution. The red solid lines show the numerical simulation based on the model (1). The green solid lines show the numerical simulation based on the mean field system in Zan [23]. (a) the trajectories of the densities of the infective. (b) the trajectories of the densities of the final size.

3.1. **The accuracy of model(1).** In this section, we will do some simulations to confirm the accuracy of model (1).

In Figure 2, the blue dotted lines show the results of 100 simulations for an SICR rumor model in a network with the Poisson degree distribution with $\bar{k} = 10$. The red heavy solid lines and green heavy solid lines represent numerical simulation trajectories based on the system (1) and the mean-field systems in Zan [23], respectively. Fig. 2(a) exhibits the trajectories of the densities of the infective and Fig. 2(b) exhibits the trajectories of the densities of the final size (densities of infective, refractory and counter). Obviously, the red lines traverse through the blue lines. This implies that the model of equation (1) is reasonable.

Figure 3. Degree distribution of generated Power-law networks

In order to further verify the reliability of the model (1), we perform random simulations and numerical simulations of model (1) in a refined Power-law network.
The network considered here can be generated by assigning a random number of lines to a set of $N$ nodes according to the distribution $p_k$ defined in (4). Then by the Configuration Model (CM) process, we can get a new network which is shown to have power law characteristics in Figure 3. Figure 3 shows the degree distribution of this new network in double logarithmic coordinates system.

In Figure 4, the blue dotted lines show the results of 100 simulations for an SICR rumor model in a network with the refined power-law degree distribution defined in (4). The red heavy solid lines and green heavy solid lines represents numerical simulation trajectories based on the system (1) and the mean-field systems in Zan [23], respectively. Fig. 4(a) exhibits the trajectories of the densities of the infective and Fig. 4(b) exhibits the trajectories of the densities of the final size (densities of infective, refractory and counter). And obviously, the red lines basically traverse through the blue lines. This implies that the model of equation (1) is reasonable.

Figure 5 compares the spread of SICR rumors in the network with the Poisson degree distribution and the network with the refined power-law degree distribution. The results show that the final size of rumors in the network with the power-law degree distribution is smaller than that with the Poisson degree distribution. Furthermore, the density of spreaders reaches its maximum earlier, which is smaller than that with the Poisson degree distribution. Although heterogeneity accelerates the spread of rumors, it reduces the scale of spread.

3.2. The speed of rumor spreading. Here we investigate how fast rumor spreads by numerical simulations based on the model (1). The speed at which the rumor spreads, which is denoted as $v$, is how many proportions of nodes is told the rumor per unit of time. Let $v_k$ represent how many proportions of nodes with degrees of $k$ hear the rumor per unit time. Then

$$v = \sum_k (\alpha + \beta + \delta)kp_k\theta^k P_S^I, \quad v_k = (\alpha + \beta + \delta)kp_k\theta^k P_S^I.$$
Figure 5. Numerical simulations of the model (1) with $\alpha = 0.7$, $\beta = 0.1$, $\delta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$ in the networks with different degree distribution but with the same averaged degree. The red dotted lines and the blue solid lines correspond to numerical simulations trajectories based on the model (1) in a network with the refined power-law degree distribution and the Poisson degree distribution, respectively. (a) the trajectories of the densities of the infective. (b) the trajectories of the densities of the final size.

Their evolution over time in the Poisson network and the network with the power-law distribution are shown in Fig.6 (a). It shows that, in the network with the power-law distribution, the rumor spreads faster and the speed reaches its maximum earlier. Since the rumor is spread by contact, is it easier for nodes with the larger degree to know rumors because they have more contacts than the nodes with the smaller degree? Figure 7 gives us a confusion that more nodes with the smaller degrees are told the rumor per unit time. Why did this happen? We think this may be related to the degree distribution of the network which is shown in Fig.6(b). Although a node with the larger degree has more contacts than a node with the smaller degree, the non-uniformity of degree distribution makes the nodes with smaller degree emit more edges in the whole network. Due to the non-uniformity of degree distribution, we define the relative propagation velocity as follows

$$\hat{v}_k = \frac{v_k}{p_k} = (\alpha + \beta + \delta)k\theta^k P_{S}.$$  

Figure 8 gives the evolution of the relative propagation velocity over time in the Poisson network and the network with the power-law distribution, respectively, which further confirms that the nodes with the larger degree, under the conditions of equal opportunity, are more likely to be told the rumor.

3.3. The effects of parameters on the rumor spreading. In this section, we investigate the effects of parameters on the rumor spreading model.

Figure. 9 displays the densities of the infective (a) and the final size (b) under the different parameter of persuading rate $\eta$. Let $\alpha = 0.7$, $\beta = 0.1$, $\delta = 0.2$, $\gamma = 0.2$, and the black solid lines, the green star lines, the red dotted lines and the blue circle lines represent numerical simulation trajectories based on the model (1) with the different value of $\eta = 0.9$, $\eta = 0.7$, $\eta = 0.5$ and $\eta = 0.3$. We can see that the densities of the infective and the final size both decrease as $\eta$ increases. It
The speed of rumor spreading, i.e. $v$

**Poisson networks**

**Power−law networks**

Figure 6. (a) Time evolutions of the speeds of the rumor spreading $v$ in the complex network with poisson degree distribution and Power-law distribution, respectively. (b) Degree distributions

Figure 7. Time evolutions of the speeds of the rumor spreading in the nodes with degree $k = 5, 10, 15$, i.e. $v_k$. (a) In network with Poisson distribution. (b) In network with Power Law distribution.

Figure 8. Time evolutions of the relative propagation velocity in the nodes with degree $k = 5, 10, 15$, i.e. $\hat{v}_k$. (a) In network with Poisson distribution. (b) In network with Power Law distribution.
means that counters play an important role in blocking the spreading of rumor. The damage of rumor spreading will be larger if there is no counter.

**Figure 9.** numerical simulations with $\alpha = 0.7$, $\beta = 0.1$, $\delta = 0.2$, $\gamma = 0.2$. (a) the trajectories of the densities of the infective over time under different persuading rate $\eta$. (b) the trajectories of the densities of the final size over time under different persuading rate $\eta$.

**Figure 10.** Numerical simulations with $\beta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$. (a) the trajectories of the densities of the susceptible over time under different spreading rate $\alpha$ and refuting rate $\delta$. (b) the trajectories of the densities of the infective over time under different spreading rate $\alpha$ and refuting rate $\delta$. (c) the trajectories of the densities of the final size over time under different spreading rate $\alpha$ and refuting rate $\delta$.

Figure 10 shows the densities of the susceptible (a), infective (b) and the final size (c) under the different parameters of spreading rate $\alpha$ and refuting rate $\delta$. When $\alpha + \beta + \delta = 1$, keep $\beta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$ unchanged and assign pairs of $(\alpha, \delta)$ different values. The red dotted lines, the green star lines and the blue solid lines represent numerical simulation trajectories based on the model (1) in the following three situations respectively: $\alpha = 0.5$, $\delta = 0.3$; $\alpha = 0.6$, $\delta = 0.2$; $\alpha = 0.7$, $\delta = 0.1$. Obviously, the densities of the susceptible decrease as $\alpha$ increases, and increase as $\delta$ increases. The densities of the infective and the final size increase as $\alpha$ increases, and decrease as $\delta$ increases. That is to say, if the susceptible nodes are persuaded into counterattack nodes with higher probability rather than into the
infected nodes, the final size of rumors will decrease. In other words, when people hear rumors, they can distinguish the right from the wrong and prevent the rumors instead of spreading them. In that way, the harm of rumors will be reduced.

Although there are many similarities between rumor and disease transmission, their recovery mechanisms are quite different. The recovery mechanism of disease transmission only depends on the infected person himself, not on the network structure; and in the recovery mechanism of rumor transmission, the infected person needs to meet another infected person, refractory person or counterattack person to recover. Therefore, the recovery of infected persons in rumor spreading mechanism is more dependent on the structure of the network. In order to compare the difference between the two recovery mechanisms, we do the numerical simulations of SIR model under the two mechanisms. Results show that the densities of infective nodes which recovery depends on the structure of the network will far blow which depends on themselves. And so do the densities of the final size.

4. **Discussion.** In this study, we establish an SICR model by the methods of PGF and pair-approximation and obtain the following conclusions.

1. Simulations confirm that the model obtained in this paper can better model the rumor spreading in the complex networks.
2. The choice of the initial rumor spreader will not affect whether the rumor breaks out or not, but only affects the speed of the rumor propagation.
3. Rumors spread faster in Scale-free networks than in Poisson networks. However, the rumor size in the latter is higher than that in the former.
4. Under the conditions of equal opportunity, it is true that the nodes with larger degrees are more likely to be told the rumor.
5. Different from the disease recovery mechanism, the rumor recovery mechanism is dependent on the network. This makes its recovery rate not a constant for the rumor propagator. In the early stage of rumor spreading, the neighbors of the rumor infected nodes are susceptible, so their recovery time is prolonged; however, in the middle and later stage of rumor spreading, the number of the susceptible neighbors of the rumor infected nodes is decreasing, so that the rumor infected nodes recover faster and faster. Therefore, this recovery mechanism will reduce the scale of transmission.
Table 1. Key variables and parameters

| Symbol | Description |
|--------|-------------|
| $\alpha$ | Spreading rate. The constant rate at which a susceptible node becomes an infective node when it contacts an infective node |
| $\beta$ | Ignoring rate. The constant rate at which a susceptible node becomes a refractory node when it contacts an infective node |
| $\delta$ | Refuting rate. The constant rate at which a susceptible node becomes a counterattack node when it contacts an infective node |
| $\gamma$ | Stifling rate. The constant rate at which an infective node becomes a refractory node when it contacts another infective or refractory node |
| $\eta$ | Persuading rate. The constant rate at which an infective node becomes a refractory node when it contacts a counterattack node |
| $p_k$ | The probability that a node will have degree $k$ |
| $g(x)$ | The probability generating function for the degree distribution $\{p_k\}$ |
| $P^I_S$ | The probability that an arc with an ego in set $X$ has an alter in $Y$ |
| $A_X$ | Set of arcs $(ego,alter)$ such that node ego is in set $X$ |
| $M_X$ | Fraction of arcs in set $A_X$ |
| $A_{X,Y}$ | Set of arcs $(ego,alter)$ s.t $ego \in X$ and $alter \in Y$ |
| $M_{X,Y}$ | Fraction of arcs in set $A_{X,Y}$ |

Since the flow of people, for an SICR rumor propagation process, is one-way, the rumor in a closed population disappears eventually. However, if the population is not closed, it is possible for the rumor to be persistent. This is a question worthy of our follow-up study.

In addition, some special network structures, such as clustering and small-world network, are not considered in this paper. It is another question we need to follow up on.

Appendix: The dynamics of SICR model in random networks. In this section, we develop an SICR rumor spreading model involving the variables $\theta, P^I_S, P_C^S, P^R_S, M_{IC}, I_k$. $N$ is the total number of nodes. $A$ is the set of all arcs. For ease of reference, the notations used in this paper are summarized in Table 1.

Firstly, we introduce the variable $P^I_S$. Consider a susceptible ego at time $t$ with a degree $k$. Then this ego will correspond a set of $k$ arcs, and we can mark them as $\{(ego,alter_1), (ego,alter_2), \cdots, (ego,alter_k)\}$. For each $(ego, alter_i)$, we will assume the probability that $alter_i$ is infective node is $P^I_S = M_{SI}/M_S$. In a time $dt$, there is an expected number $(\alpha + \beta + \delta)kP^I_S dt$ that the infective alter transmits the rumor to ego. Consequently, the hazard for a susceptible node knowing the rumor at time $t$ is

$$\lambda_k(t) = - (\alpha + \beta + \delta)kP^I_S(t).$$

Now, let $S_k(t)$ represent the probability of nodes with degree $k$ that remain susceptible at time $t$. Obviously, the dynamics of $S_k(t)$ is

$$S_k(t) = -(\alpha + \beta + \delta)kP^I_S(t)S_k(t). \quad (5)$$

From Eq. (5), we have

$$S_k(t) = exp[- \int_0^t (\alpha + \beta + \delta)kP^I_S(\tau)d\tau] = \{exp[- \int_0^t (\alpha + \beta + \delta)P^I_S(\tau)d\tau]\}^k. \quad (6)$$
Then, we use the symbol $\theta$ to express a node with degree 1 remain susceptible at time $t$. Therefore, from Eq. (6), we have

$$\theta = \exp[-\int_0^t (\alpha + \beta + \delta) P^I_S(\tau) d\tau].$$

(7)

The dynamics of $\theta$ is easily placed as

$$\dot{\theta} = -(\alpha + \beta + \delta) P^I_S \theta.$$ 

(8)

Thus, the fraction of nodes which remain susceptible at time $t$ is

$$S = p_0 + p_1 \theta + p_2 \theta^2 + p_3 \theta^3 + \cdots = g(\theta).$$

(9)

And the dynamics of $S$ is

$$\dot{S} = -(\alpha + \beta + \delta) P^I_S \theta g'(\theta).$$

(10)

In order to get the dynamics of $\theta$, we need to know the variable $P^I_S$. The derivation of $P^I_S$ follows as

$$\dot{P}^I_S = \frac{d}{dt} \frac{M_{S1}}{M_S} = \frac{\dot{M}_{S1} M_S - M_{S1} \dot{M}_S}{M_S^2}. $$

(11)

But the derivation of $P^I_S$ depends on $M_S$, $\dot{M}_S$, $M_{S1}$ and $\dot{M}_{S1}$. $M_S$ is

$$M_S = \sum_k k S_k p_k \sum_k k p_k = \frac{\theta g'(\theta)}{g'(1)}.$$ 

(12)

We only need to take the derivative of Eq. (12) to get

$$\dot{M}_S = \frac{d}{dt} \frac{\theta g'(\theta)}{g'(1)} = \frac{-(\alpha + \beta + \delta) P^I_S \theta g'(\theta) + \theta g''(\theta)}{g'(1)}. $$

(13)

$M_{S1}$ follows as

$$M_{S1} = M_S \cdot \frac{M_{S1}}{M_S} = M_S \cdot P^I_S = \frac{P^I_S \theta g'(\theta)}{g'(1)}. $$

(14)

For the goal of $\dot{M}_{S1}$, we must arrange the change of arcs $SI$. We can consider it from three aspects.

1) The susceptible nodes become the infective nodes, counter nodes or refractory nodes due to the contacting with the infective nodes. So, $M_{S1}$ decreases at the rates

$$(\alpha + \beta + \delta) M_{S1}. $$

(15)

2) The change of the arcs $SI$ caused by the different states of the neighbors of susceptible nodes’ excess degree. So we will give PGF for the excessive degree distribution of the ego node $S$ in arcs $SI$.

$$g_{SI}(X_S, X_I, X_C, X_R) = \frac{g'[\theta(P^S_S X_S + P^I_S X_I + P^C_S X_C + P^R_S X_R)]}{g'(\theta)}.$$ 

(16)

The detailed derivation of Eq. (15) is similar to [7]. The mean of the distribution can be obtained because of the property of PGF. So using Eq. (15), we have the following equations.

$$\delta_{SI}(I) = \left[\frac{d}{dx_I} g_{SI}(X_S, X_I, X_C, X_R)\right]|_{X_S=X_I=X_C=X_R=1} = \frac{\theta P^I_S g''(\theta)}{g'(\theta)} $$

(17)
\[
\delta_{SI}(C) = \left[ \frac{d}{dX_C} g_{SI}(X_S, X_I, X_C, X_R) \right]_{X_S = X_I = X_C = X_R = 1} = \frac{\theta P^C g''(\theta)}{g'(\theta)}
\]

(18)

Thus, \( M_{SI} \) increases at the rates \( (\alpha - \dot{S})\delta_{SI}(S)/(\alpha + \beta + \delta) - (\dot{S})\delta_{SI}(I)/\gamma' \).

(3) \( M_{SI} \) decrease as the infective nodes become refractory nodes. We can describe it by the following formula,

\[
M_{SI}(\gamma \delta_{IS}(I) + \gamma \delta_{IS}(R) + \eta \delta_{IS}(C)) = \frac{A_{SI}(\gamma \delta_{IS}(I) + \gamma \delta_{IS}(R) + \eta \delta_{IS}(C))}{A} = \frac{\sum_k \gamma \{[SI_k I] + [SI_k R] + \eta [SI_k C]\}}{A}
\]

\( = \frac{\gamma \sum_k [SI_k] \gamma \sum_k [SI_k S] + (\eta - \gamma) \sum_k [SI_k C]}{A} \) (19)

Since

\[
P_I^S = \frac{M_{IS}}{M_I} = \frac{g'(1)M_{IS}}{\sum_k kI_k p_k}, \quad (20)
\]

\[
P_I^C = \frac{M_{IC}}{M_I} = \frac{g'(1)M_{IC}}{\sum_k kI_k p_k}, \quad (21)
\]

and

\[
M_{IS} = M_{SI},
\]

we have

\[
P_I^S = \frac{P_I^C \sum_k kS_k p_k}{\sum_k kI_k p_k} = \frac{P_I^C g'(\theta)}{\sum_k kI_k p_k}.
\]

(23)

From Eq. (23) and

\[
P_I^C = \frac{M_{IC}}{M_I} = \frac{g'(1)M_{IC}}{\sum_k kI_k p_k}, \quad (24)
\]

we can expand three tuples with degrees as follows,

\[
\sum_k [SI_k] = \sum_k k(k - 1)I_k p_k N P_I^S = \frac{P_I^C g'(\theta) \sum_k k(k - 1)I_k p_k N}{\sum_k kI_k p_k}.
\]

(25)

\[
\sum_k [SI_k S] = \sum_k k(k - 1)I_k p_k N (P_I^S)^2 = \frac{(P_I^C g'(\theta))^2 \sum_k k(k - 1)I_k p_k N}{(\sum_k kI_k p_k)^2}
\]

(26)

and

\[
\sum_k [SI_k C] = \sum_k k(k - 1)I_k p_k N P_I^S P_I^C = \frac{g'(1)M_{IC} P_I^C g'(\theta) \sum_k k(k - 1)I_k p_k N}{(\sum_k kI_k p_k)^2}.
\]

(27)

From Eqs. (25) (26) and (27), Eq. (19) is

\[
P_I^C g'(\theta) \sum_k k(k - 1)I_k p_k \{\gamma \sum_k kI_k p_k - P_I^C g'(\theta)\} + (\eta - \gamma) g'(1)M_{IC}
\]

\[
= \frac{g'(1)(\sum_k kI_k p_k)^2}{g'(1)\sum_k kI_k p_k}.
\]

(28)
So, we have
\[
\hat{M}_{SI} = \frac{\alpha P_S^f P_S^g \theta^2 g''(\theta) - (\alpha + \beta + \delta) P_S^f \theta g'(\theta) + \theta P_S^f g''(\theta)}{g'(1)}
\]
\[
= \frac{P_S^f \theta g'(\theta) \sum_k k(k - 1) I_k p_k \{ \gamma \left( \sum_k k I_k p_k - P_S^f \theta g'(\theta) \right) + (\eta - \gamma) g'(1) M_{IC} \}}{g'(1) \left( \sum_k k I_k p_k \right)^2}.
\]

(29)

Now, it is easy to gain \( \hat{P}_S^f \) from Eqs. (11)-(14) and (29).
\[
\hat{P}_S^f = - \frac{P_S^f \sum_k k(k - 1) I_k p_k \{ \gamma \left( \sum_k k I_k p_k - P_S^f \theta g'(\theta) \right) + (\eta - \gamma) g'(1) M_{IC} \}}{g'(1) \left( \sum_k k I_k p_k \right)^2}
\]
\[
+ \frac{\alpha P_S^f P_S^g \theta^2 g''(\theta)}{g'(\theta)} - (\alpha + \beta + \delta) P_S^f (1 - P_S^f)
\]

(30)

Next we can consider \( \hat{M}_{IC} \).
\[
\hat{M}_{IC} = \delta M_{SI} + \frac{\alpha (-\tilde{S}) \delta_{SI}(C)}{(\alpha + \beta + \delta) g'(1)} + \frac{\delta (-\tilde{S}) \delta_{SI}(I)}{(\alpha + \beta + \delta) g'(1)} - \eta M_{IC} - \eta M_{IC} \delta_{IC}(C)
\]
\[
- \gamma [M_{IC} \delta_{IC}(I) + M_{IC} \delta_{IC}(R)]
\]

(31)

Similarity to the analysis of Eqs. (25) (26) and (27), we can get
\[
\eta M_{IC} \delta_{IC}(C) = \frac{\eta \sum_k [C I_k C]}{\mathcal{A}} = \frac{\eta g'(1) (M_{IC})^2 \sum_k k(k - 1) I_k p_k}{(\sum_k k I_k p_k)^2}
\]

(32)

and
\[
\gamma [M_{IC} \delta_{IC}(I) + M_{IC} \delta_{IC}(R)] = \frac{\gamma \sum_k ([I I_k C] + [R I_k C])}{\mathcal{A}}
\]
\[
= \frac{\gamma \sum_k k(k - 1) I_k p_k P_F^C (P_F^l + P_R^l)}{g'(1)}
\]
\[
= \frac{\gamma \sum_k k(k - 1) I_k p_k P_F^C (1 - P_F^s - P_F^r)}{g'(1)}
\]
\[
= \frac{\gamma \sum_k k(k - 1) I_k p_k M_{IC}}{\sum_k k I_k p_k} (1 - \frac{P_S^f \theta g'(\theta) + g'(1) M_{IC}}{\sum_k k I_k p_k})
\]

(33)

So, \( M_{IC} \) becomes
\[
\hat{M}_{IC} = \frac{\delta P_S^f \theta g'(\theta) + \theta^2 P_S^g \theta g''(\theta) (\alpha P_S^C + \delta P_S^f)}{g'(1)} - \frac{\eta g'(1) (M_{IC})^2 \sum_k k(k - 1) I_k p_k}{(\sum_k k I_k p_k)^2}
\]

(34)
− \eta M_{IC} \sum_{k} k(k - 1)I_k p_k M_{IC} \sum_{k} k I_k p_k (1 - \frac{P_S \theta g'(\theta) + g'(1) M_{IC}}{M_{IC}}).

We notice that the method to gain \dot{P}_C^S is very similar to that for \dot{P}_I^S. So

\dot{P}_C^S = \frac{d}{dt} M_{SC} = \frac{M_{SC}}{M_S} - \frac{M_{SC} \dot{M}_S}{M_S^2} \tag{35}

and

M_{SC} = M_S \cdot \frac{M_{SC}}{M_S} = M_S \cdot P_C^S = \frac{P_S \theta g'(\theta)}{g'(1)} \tag{36}

can be obtained. The PGF and excessive degree of arcs SC can be obtained in the same way as the above. We have

\begin{equation}
g_{SC}(X_S, X_I, X_C, X_R) = \frac{g'[\theta(P_S^S X_S + P_I^I X_I + P_C^C X_C + P_R^R X_R)]}{g'(\theta)} \tag{37}
\end{equation}

and

\begin{equation}
\delta_{SC}(I) = \left[ \frac{d}{dX_I} g_{SC}(X_S, X_I, X_C, X_R) \right]_{X_S = X_I = X_C = X_R = 1} = \frac{\theta P_S^I g''(\theta)}{g'(\theta)}. \tag{38}
\end{equation}

Then we can obtain

\begin{equation}
M_{SC} = \frac{\delta(-\dot{S}) \delta_{SI}(S)}{(\alpha + \beta + \delta) g'(1)} - (\alpha + \beta + \delta) M_{SC} \delta_{SC}(I) = \frac{\theta^2 P_S^I g''(\theta) \delta P_S^I - (\alpha + \beta + \delta) P_S^C}{g'(1)}. \tag{39}
\end{equation}

Now applying Eqs. (12) (13) (36) and (39) to Eq. (35), we have

\begin{equation}
\dot{P}_C^S = \frac{\delta P_S^I P_S^S \theta g''(\theta)}{g'(\theta)} + (\alpha + \beta + \delta) P_S^C P_S^I. \tag{40}
\end{equation}

We can get \dot{P}_S^S by the same way. Firstly, we have

\begin{equation}
\dot{P}_S^S = \frac{d}{dt} M_{SS} = \frac{\dot{M}_{SS} M_S - M_{SS} \dot{M}_S}{M_S^2} \tag{41}
\end{equation}

and

\begin{equation}
M_{SS} = M_S \cdot \frac{M_{SS}}{M_S} = M_S \cdot P_S^S = \frac{P_S^S \theta g'(\theta)}{g'(1)} \tag{42}
\end{equation}

Moreover we can get

\begin{equation}
\dot{M}_{SS} = \frac{-2(-\dot{S}) \delta_{SI}(S)}{g'(1)}. \tag{43}
\end{equation}

Applying Eqs. (12) (13) (42) and (43) to Eq. (41), we have

\begin{equation}
\dot{P}_S^S = \frac{(\alpha + \beta + \delta) P_S^I P_S^S [g'(\theta) - \theta g''(\theta) + g'(\theta)]}{g'(\theta)}. \tag{44}
\end{equation}
Finally, the dynamics of the infective nodes is
\[
\dot{I}_k = k(\alpha S_k P^I_k - \gamma I_k P^I_k - \gamma I_k P^R_k - \eta I_k P^C_k)
\]
\[
= k\alpha \theta \theta_k P^I_k - \frac{k\gamma I_k \sum_k I_k \theta_k - P^I_k \theta g'(\theta)}{\sum_k I_k \theta_k} + k(\eta - \gamma) I_k g'(1) M IC
\]
(45)

Acknowledgments. This work is supported by the National Natural Science Foundations of China under Grant (Nos. 11571210, 11331009, 11501339, 11101251, 11001157, 11471197, 11701348) and the Youth Science Foundation of Shanxi Province (No. 2010021001-1). The authors wish to thank the anonymous referees for their helpful feedback and suggestions.

REFERENCES

[1] L. Buzna, K. Peters and D. Helbing, Modelling the dynamics of disaster spreading in networks, Physica A, 363 (2006), 132–140.
[2] D. J. Daley and D. G. Kendall, Stochastic rumours, Ima Journal of Applied Mathematics, 1 (1965), 42–55.
[3] L.-A. Huo, P. Q. Huang and X. Fang, An interplay model for authorities’ actions and rumor spreading in emergency event, Physica A, 390 (2011), 3267–3274.
[4] L. A. Huo, L. Wang, N. X. Song, C. Y. Ma and B. He, Rumor spreading model considering the activity of spreaders in the homogeneous network, Physica A, 468 (2017), 855–865.
[5] R. L. Jie, J. Qiao, G. J. Xu and Y. Y. Meng, A study on the interaction between two rumors in homogeneous complex networks under symmetric conditions, Physica A, 454 (2016), 129–142.
[6] C. Lefèvre and P. Picard, Distribution of the final extent of a rumour process, Journal of Applied Probability, 31 (1994), 244–249.
[7] J. X. Li, J. Wang and Z. Jin, SIR dynamics in random networks with communities, Journal of Mathematical Biology, 77 (2018), 1117–1151.
[8] Q. M. Liu, T. Li and M. C. Su, The analysis of an SEIR rumor propagation model on heterogeneous network, Physica A, 469 (2017), 372–380.
[9] K. Ma, W. H. Li, Q. T. Guo, X. Q. Zheng, Z. M. Zheng, C. Gao and S. T. Tang, Information spreading in complex networks with participation of independent spreaders, Physica A, 492 (2018), 21–27.
[10] H. Matsuda, N. Ogita, A. Sasaki and K. Sato, Statistical mechanics of population: The lattice Lotka-Volterra model, Progress of Theoretical Physics, 88 (1992), 1035–1049.
[11] Y. Moreno, M. Nekovee and A. Pacheco, Dynamics of rumor spreading in complex networks, Physical Review E, 69 (2004), 066130.
[12] M. Nekovee, Y. Moreno, G. Bianconi and M. Marsili, Theory of rumour spreading in complex social networks, Physica A, 374 (2007), 457–470.
[13] M. E. J. Newman, S. Forrest and J. Balthrop, Email networks and the spread of computer viruses, Physical Review E, 66 (2002), 035101.
[14] Z. Qian, S. T. Tang, X. Zhang and Z. M. Zheng, The independent spreaders involved SIR Rumor model in complex networks, Physica A, 429 (2015), 95–102.
[15] F. Roshani and Y. Naimi, Effects of degree-biased transmission rate and nonlinear infectivity on rumor spreading in complex social networks, Physical Review E, 85 (2012), 036109.
[16] Z. Y. Ruan, M. Tang and Z. H. Liu, Epidemic spreading with information-driven vaccination, Physical Review E, 86 (2012), 036117.
[17] E. Volz, SIR dynamics in random networks with heterogeneous connectivity, Journal of Mathematical Biology, 56 (2008), 293–310.
[18] C. Wan, T. Li, Y. Wang and et al., Rumor Spreading of a SICS Model on complex social networks with counter mechanism, Open Access Library Journal, 03(07) (2016), 1–11.
[19] J. J. Wang, L. J. Zhao and R. B. Huang, 2S2R rumor spreading model in homogeneous networks, Physica A, 413 (2014), 153–161.
[20] J. J. Wang, L. J. Zhao and R. B. Huang, SIRaRu rumor spreading model in complex networks, Physica A, 398 (2014), 43–55.
[21] Y. Wang, X. Yang, Y. Han and et al., Rumor spreading model with trust mechanism in complex social networks, *Communications in Theoretical Physics*, 59(4) (2013), 510–516.

[22] L.-L. Xia, G.-P. Jiang, B. Song and Y.-R. Song, Rumor spreading model considering hesitating mechanism in complex social networks, *Physica A*, 437 (2015), 295–303.

[23] Y. L. Zan, J. L. Wu, P. Li and Q. L. Yu, SICR rumor spreading model in complex networks: Counterattack and self-resistance, *Physica A*, 405 (2014), 159–170.

[24] D. H. Zanette, Critical behavior of propagation on small-world networks, *Physical Review E*, 64 (2001), 050901.

[25] D. H. Zanette, Dynamics of rumor propagation on small-world networks, *Physical Review E*, 65 (2002), 041908.

[26] L. J. Zhao, X. Y. Qiu, X. L. Wang and J. J. Wang, Rumor spreading model considering forgetting and remembering mechanisms in inhomogeneous networks, *Physica A*, 392 (2013), 987–994.

[27] L. J. Zhao, J. J. Wang, Y. C. Chen, Q. Wang, J. J. Cheng and H. X. Cui, SIHR rumor spreading model in social networks, *Physica A*, 391 (2012), 2444–2453.

[28] L. Zhu and Y. G. Wang, Rumor spreading model with noise interference in complex social networks, *Physica A*, 469 (2017), 750–760.

Received December 2018; revised July 2019.

E-mail address: lijinxian@sxu.edu.cn
E-mail address: 842603506@qq.com
E-mail address: jinzhu263.net