

# Effects of degree-biased transmission rate and nonlinear infectivity on rumor spreading in complex social networks

F. Roshani<sup>1,2,3</sup> and Y. Naimi<sup>4</sup><sup>1</sup>*Department of Physics, Alzahra University, Tehran, 19938-91167, Iran*<sup>2</sup>*The Abdus Salam International Centre for Theoretical Physics (ICTP), Strada Costiera 11, I-34014 Trieste, Italy*<sup>3</sup>*Institute for Research in Fundamental Sciences (IPM) School of Particles and Accelerators, P.O. Box 19395-5531, Tehran- Iran*<sup>4</sup>*Department of Physics, Lamerd Branch, Islamic Azad University, Lamerd, Iran*

(Received 10 June 2011; revised manuscript received 22 October 2011; published 22 March 2012)

We introduce a generalized rumor spreading model and analytically investigate the spreading of rumors on scale-free (SF) networks. In the standard rumor spreading model, each node has an infectivity equal to its degree, and connectivity is uniform across all links. To generalize this model, we introduce an infectivity function that determines the number of simultaneous contacts that a given node (individual) may establish with its connected neighbors and a connectivity strength function (CSF) for the direct link between two connected nodes. These lead to a degree-biased propagation of rumors. For nonlinear functions, this generalization is reflected in the infectivity's exponent  $\alpha$  and the CSF's exponent  $\beta$ . We show that, by adjusting exponents  $\alpha$  and  $\beta$ , the epidemic threshold can be controlled. This feature is absent in the standard rumor spreading model. In addition, we obtain a critical threshold. We show that the critical threshold for our generalized model is greater than that of the standard model on a finite SF network. Theoretically, we show that  $\beta = -1$  leads to a maximum spreading of rumors, and computation results on different networks verify our theoretical prediction. Also, we show that a smaller  $\alpha$  leads to a larger spreading of rumors. Our results are interesting since we obtain these results regardless of the network topology and configuration.

DOI: [10.1103/PhysRevE.85.036109](https://doi.org/10.1103/PhysRevE.85.036109)

PACS number(s): 89.75.Hc, 02.50.Ey, 64.60.aq

## I. INTRODUCTION

During recent years, network modeling was the favorite tool used by researchers for representing many social, biological, and physical systems [1,2]. In general terms, complex networks are connected graphs with, at most, a single edge between nodes where nodes stand for individuals and an edge denotes the interaction between individuals [3,4]. The connectivity pattern in these networks encodes information about the structure of the system [5]. An important and much studied characteristic of these networks is their degree distribution  $p(k)$ , defined as the probability that a randomly selected node is connected to  $k$  other nodes. It was found that many networks of interest in various disciplines, such as the Internet [6] and the World Wide Web [7] (in communication technology), sexual contact networks [8], friendship networks [9], scientific collaboration networks [10] (social sciences), metabolic networks [11], and food webs [12] (biological systems) are very heterogeneous with scale-free (SF) degree distribution  $p(k) \sim k^{-2-\gamma}$  (power-law distribution) where  $\gamma$  is a characteristic degree exponent, usually in the range of  $0 < \gamma \leq 1$ . The study of epidemics in heterogeneous networks, therefore, is of practical importance for the control of the spread of viruses, diseases, and biological epidemics.

The modeling of infectious diseases has been used to study the mechanisms by which diseases spread, to predict the future course of an outbreak, and to evaluate strategies to control an epidemic [13]. Among the numerous possible models, the most investigated classical models are the susceptible-infected-susceptible (SIS) model [14–17] and the susceptible-infected-removed (SIR) model [18–20], which can describe the spreading of diseases in social networks or that of computer viruses and trash mail in communication networks. One of the remarkable results for SIS and SIR models in

an infinite-size SF network is that the infection becomes an epidemic regardless of its spreading rate (i.e., the critical threshold of transmission rate is zero).

Rumors, as old basic elements of human interaction [21], spreading rapidly, are difficult to control, are invisible, yet nearly are impossible to ignore, and can have damaging and perhaps even deadly consequences.

Despite its obvious negative connotations, a rumor has the capacity to satisfy certain fundamental personal and social needs and, in this sense, can be beneficial to those who participate in its transmission [22–24]. Rumors help people make sense of what is going on around them. Also, rumor mongering is a way of trying to explain what is happening and why—be it a crime in the neighborhood, a political crisis, or a change in a company's management.

A rumor can be interpreted as an *infection of the mind*. The original model of rumor spreading was introduced by Daley and Kendall (DK) [25] and Daley and Gani [26]. An important variant model of DK is the Maki-Thompson (MK) model [27]. In the past, these models were used extensively to study rumor spreading [28–30]. In the DK model, a closed and homogeneously mixed population is subdivided into three groups: the ignorant, those who have not heard the rumor yet; the spreader, those who have heard the rumor and are willing to transmit it; the stifter, those who have heard the rumor but have lost interest in the rumor and do not transmit it. The rumor is propagated through the population by pairwise contacts between spreaders and others in the society. Any spreader involved in a pairwise meeting attempts to *infect* the other individual with the rumor. If this other individual is an ignorant, it becomes a spreader; otherwise, the spreader meets another spreader or stifter, so they understand that the rumor is known and do not propagate the rumor anymore, therefore, turning into stiflers. In the MK model, the rumor is spread by

directed contacts of the spreaders with others. Furthermore, when a spreader contacts another spreader, only the initiating spreader becomes a stifier.

In the above-mentioned models of rumor spreading, the authors have assumed that the rumors spread across homogeneous networks (with degree distribution peaked around the average value), on the other hand, these calculations were made in the limit of highly simplified models of the topology [28,29]. While in the real world, the topology of such large social networks shows highly complex connectivity patterns in which the degree distribution is skewed and may present heavy tails or, more generally, large fluctuations around the average value [30–32]. Recently, in Ref. [33], the authors introduced a model of rumor spreading on complex networks, which, in comparison with previous models, provides a more realistic description of this process. Their model unifies the MK model of rumor spreading with SIR models of epidemics and has both of these models as limiting cases. They have used approximate analytical and exact numerical solutions of a mean-field equation to examine both the steady-state and the time-dependent behaviors of the model on several models of social networks (homogeneous networks, Erdős-Rényi (ER) random graphs and uncorrelated SF networks). They found that their model shows a different critical behavior on networks with bounded degree fluctuations, such as random graphs and that this behavior is absent in SF networks with unbounded fluctuations in node degree distribution.

We know that, in the rumor spreading models studied so far, the transmission rate of a rumor is a constant, but in the real world, it should be different among individuals depending on their intimacy. Thus, in order to make the transmission rate more realistic, we introduce a connectivity strength function (CSF) between connected nodes. For epidemic spreading, for two connected nodes, a larger CSF represents a higher probability of communication between the two nodes.

Also, in the classical epidemic models, each spreader can establish contacts with all his (her) neighbors within one time step, that is to say, each node’s infectivity (a rumor as an infection of the mind) equals its degree. But in the real case, an individual cannot make contact with all acquaintances (connected neighbors) simultaneously. In the case of SIS and SIR models, recently in Ref. [34], the authors have dropped this assumption and have assumed that the infectivity is identical (a constant  $A$ ) for all nodes regardless of their different degrees. Also, in Ref. [35], the authors have proposed a piecewise linear infectivity, which means: If the degree  $k$  of a node is relatively small, its infectivity is  $\alpha k$ ; if  $k$  is large, i.e., surpasses a constant  $A/\alpha$ , then its infectivity is  $A$ . In terms of these assumptions, the heterogeneous infectivity of nodes with different degrees cannot be considered as adequately as possible in SF networks because it means that there may be some nodes with different degrees but with the same infectivity and there is a large number of such nodes independent from the selected constant  $A$  or from the fact that the size of the underlying networks is infinite. In order to tackle this problem, in Ref. [36], the authors introduced the nonlinear infectivity function that controls the number of contacts that a node generates within one time step. We follow this approach and use the nonlinear function for the infectivity of nodes that spread the rumor to their neighbors.

The rest of this paper is organized as follows: In Sec. II, we introduce the standard model of rumor spreading and shortly review epidemic dynamics of this model. In Sec. III, we introduce the generalized rumor spreading model and analytically study the dynamics of this model on infinite SF networks in detail. In Sec. IV, we compare the epidemic behavior of the standard and generalized models on finite SF networks. Finally, our conclusions are presented in the last section.

## II. STANDARD RUMOR SPREADING MODEL

The rumor model is defined as follows. Each of the individuals (the nodes in the network) can be in three different categories with respect to the rumor. In this way, individuals are classified as  $I$ , the ignorant (those who are ignorant of the rumor),  $S$ , the spreader (those who have heard the rumor and actively spread it), and  $R$ , the stifier (those who know the rumor but have ceased to spread it). According to Maki and Thompson [27], the spreading process evolves by directed contact of the spreaders with the rest of the population. However, these contacts only can occur along the links of an undirected social interaction network  $G = (V, E)$ , where  $V$  and  $E$  denote the nodes and the edges of the network, respectively. The model that we call the standard model has been studied in Ref. [33]. By following Ref. [33], the possible events that can occur between the spreaders and the rest of the population are

- (1)  $SI \rightarrow SS$  whenever a spreader meets an ignorant, the ignorant becomes a spreader at a rate  $\lambda$ .
- (2)  $SS \rightarrow RS$  when a spreader contacts another spreader, the initiating spreader becomes a stifier at a rate  $\sigma$ .
- (3)  $S \rightarrow R$  there also is a rate  $\delta$  for a spreader to cease spreading a rumor spontaneously (i.e., without any contact).

### A. Dynamics of the standard model

Let  $I_k(t)$ ,  $S_k(t)$ , and  $R_k(t)$  denote the densities of the ignorant, spreader, and stifier nodes (individuals) with connectivity (degree)  $k$  at time  $t$ , respectively. These quantities satisfy the normalization condition  $I_k(t) + S_k(t) + R_k(t) = 1$  for all  $k$  classes. We shortly review some classical results from Ref. [33] where Nekovee *et al.* described a formulation of this model on networks in terms of interacting Markov chains and used this framework to derive, from first principles, mean-field equations for the dynamics of rumor spreading on complex networks with arbitrary degree correlations as follows:

$$\frac{dI_k(t)}{dt} = -k\lambda I_k(t) \sum_l S_l(t)P(l|k), \quad (1)$$

$$\begin{aligned} \frac{dS_k(t)}{dt} &= k\lambda I_k(t) \sum_l S_l(t)P(l|k) - k\sigma S_k(t) \\ &\quad \times \sum_l [S_l(t) + R_l(t)]P(l|k) - \delta S_k(t), \end{aligned} \quad (2)$$

$$\frac{dR_k(t)}{dt} = k\sigma S_k(t) \sum_l [S_l(t) + R_l(t)]P(l|k) + \delta S_k(t), \quad (3)$$

where the conditional probability  $P(l|k)$  means that a randomly chosen link emanating from a node of degree  $k$  is

connected to a node of degree  $l$ . Moreover, we suppose that the degrees of nodes in the whole network are uncorrelated, i.e.,  $P(l|k) = lp(l)/\langle k \rangle$  where  $p(k)$  is the degree distribution and  $\langle k \rangle$  is the average degree. In this case, Nekovee *et al.* showed that, for leading order in  $\sigma$ , the critical threshold is independent of the stifling mechanism, i.e.,  $\frac{\lambda}{\delta} \geq \frac{\langle k \rangle}{\langle k^2 \rangle}$ , so in particular, for  $\delta = 1$ , the critical threshold is given by  $\lambda_c = \langle k \rangle / \langle k^2 \rangle$ , and it is the same as for the SIR model [18,20]. This result implies the absence of the epidemic threshold in a wide range of SF networks ( $\langle k^2 \rangle \rightarrow \infty$ ,  $\lambda_c \rightarrow 0$ ). This is a bad message for epidemic control since an epidemic occurs in many real networks with any nonzero value of transmission rate  $\lambda$ .

### III. GENERALIZED RUMOR SPREADING MODEL

In order to make transmission rates fit realistic cases more closely, we take the effect of connectivity strength between individuals into account. For example, in social networks, it can re-represent the intimacy, confidence, kinship, etc., between individuals. So, unlike previous studies (in which each individual can spread the rumor with a constant transmission rate  $\lambda$ ), in this paper, we mainly focus on the rumor spreading model in which the transmission rate between two connected nodes is a function of their degrees. Based on this assumption, we define  $g(k,l)$  as a CSF for link  $(k,l)$ . Let  $G_k$  denote the total strength of connectivity for a node with degree  $k$ , which can be obtained by summing the CSFs of the links that are connected to it, i.e.,  $G_k = k \sum_l P(l|k)g(k,l)$ .

Here, for each node with degree  $k$ , we keep the total rumor transmission rate and the total stifling process rate constant, which are given by  $\lambda k$  and  $\sigma k$ , respectively. The rumor transmission rate from the  $k$ -degree node to the  $l$ -degree node, are determined by the ratio of  $g(k,l)$  to  $G_k$ , therefore,  $\lambda_{kl}$  can be written as follows:

$$\lambda_{kl} = \lambda k \frac{g(k,l)}{G_k}, \quad (4)$$

from which it is clear that a higher  $g(k,l)/G_k$  leads to a greater probability of the transmission of the rumor through the edge. Similarly, the stifling process rate is  $\sigma_{kl} = \sigma k \frac{g(k,l)}{G_k}$ . In this paper, for simplicity, we focus on uncorrelated networks where the conditional probability satisfies  $P(l|k) = lp(l)/\langle k \rangle$ , and we assume  $g(k,l)$  is a symmetric multiplicative function of the degrees at the edge's end points, namely,  $g(k,l) = \eta(k)\eta(l)$ . Later, we show that this assumption leads to introducing the biased spreading of the rumors. So, one can obtain  $G_k = \frac{k\eta(k)\langle k\eta(k) \rangle}{\langle k \rangle}$ , and  $\lambda_{kl}$  and  $\sigma_{kl}$  are reduced to

$$\lambda_{kl} = \lambda \frac{\langle k \rangle \eta(l)}{\langle k \eta(k) \rangle}, \quad \sigma_{kl} = \sigma \frac{\langle k \rangle \eta(l)}{\langle k \eta(k) \rangle}. \quad (5)$$

Another inappropriate assumption in the details of the standard rumor spreading model is that each spreader can establish contacts with all his (or her) neighbors within one time step, that is to say, each node's infectivity equals its degree. But in the real case, an individual cannot contact all his (or her) friends simultaneously. So, we drop this assumption, and we introduce the infectivity function  $\varphi(k)$  to take control of the number of contacts that a spreading node generates within one time step. To rewrite Eqs. (1)–(3) for the generalized model, we

should replace the  $\lambda$ ,  $\sigma$ , and  $P(l|k)$  by  $\lambda_{kl}$ ,  $\sigma_{kl}$ , and  $\frac{\varphi(l)P(l|k)}{l}$ , respectively. So, we have

$$\frac{dI_k(t)}{dt} = -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} I_k(t) \sum_l S_l(t) p(l) \varphi(l), \quad (6)$$

$$\begin{aligned} \frac{dS_k(t)}{dt} &= \frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} I_k(t) \sum_l S_l(t) p(l) \varphi(l) - \frac{\sigma k \eta(k)}{\langle k \eta(k) \rangle} S_k(t) \\ &\quad \times \sum_l [S_l(t) + R_l(t)] p(l) \varphi(l) - \delta S_k(t), \end{aligned} \quad (7)$$

$$\frac{dR_k(t)}{dt} = \frac{\sigma k \eta(k)}{\langle k \eta(k) \rangle} S_k(t) \sum_l [S_l(t) + R_l(t)] p(l) \varphi(l) + \delta S_k(t). \quad (8)$$

Equation (6) can be integrated exactly to yield

$$I_k(t) = I_k(0) \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t) \right], \quad (9)$$

where  $I_k(0)$  is the initial density of ignorant nodes with connectivity  $k$  and we have used the auxiliary function,

$$\phi(t) = \sum_k p(k) \varphi(k) \int_0^t S_k(t') dt' \equiv \int_0^t \langle \varphi(k) S_k(t') \rangle dt'. \quad (10)$$

In order to get a closed relation for the final size of rumor  $R$ , it is more useful to focus on the time evolution of  $\phi(t)$ . Assuming a homogeneous initial distribution of ignorant nodes, i.e.,  $I_k(0) = I_0$  (without loss of generality, we can set  $I_0 \approx 1$ ), we can obtain a differential expression for  $\phi(t)$  by multiplying Eq. (7) with  $p(k)\varphi(k)$  and summing over all  $k$ 's. After some elementary manipulations, one finds

$$\begin{aligned} \frac{d\phi}{dt} &= \langle \varphi(k) \rangle - \left\langle \varphi(k) \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t) \right] \right\rangle - \delta \phi - \frac{\sigma}{\langle k \eta(k) \rangle} \\ &\quad \times \int_0^t \left\{ \langle \varphi(k) \rangle - \left\langle \varphi(k) \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t') \right] \right\rangle \right\} \\ &\quad \times \langle k \eta(k) \varphi(k) S_k(t') \rangle dt', \end{aligned} \quad (11)$$

On infinite time scales, i.e., at the end of the epidemic, we have  $S_k(\infty) = 0$  and, consequently,  $\lim_{t \rightarrow \infty} d\phi(t)/dt = 0$ , so Eq. (11) becomes

$$\begin{aligned} 0 &= \langle \varphi(k) \rangle - \left\langle \varphi(k) \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi_\infty \right] \right\rangle - \delta \phi_\infty \\ &\quad - \frac{\sigma}{\langle k \eta(k) \rangle} \int_0^\infty \left\{ \langle \varphi(k) \rangle - \left\langle \varphi(k) \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t') \right] \right\rangle \right\} \\ &\quad \times \langle k \eta(k) \varphi(k) S_k(t') \rangle dt', \end{aligned} \quad (12)$$

For  $\sigma = 0$ , one can solve Eq. (12) explicitly to find a closed relation for  $\phi_\infty$ . For  $\sigma \neq 0$ , we solve Eq. (12) for leading order in  $\sigma$ . For this purpose, it is sufficient to obtain  $S_k(t)$  for zeroth order in  $\sigma$ .  $S_k(t)$  of Eq. (7), for zeroth order in  $\sigma$ , is a first order linear differential equation that has the form  $\frac{dy}{dt} + p(t)y = q(t)$ , and it can easily be solved to obtain

$$\begin{aligned} S_k(t) &= 1 - \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t) \right] - \delta \int_0^\infty e^{\delta(t-t')} \\ &\quad \times \left\{ 1 - \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi(t') \right] \right\} dt' + O(\sigma). \end{aligned} \quad (13)$$

Close to the critical threshold, both  $\phi(t)$  and  $\phi_\infty$  are small by writing  $\phi(t) = \phi_\infty f(t)$  where  $f(t)$  is a finite function. Thus, if we only keep the leading order of  $\phi_\infty$ , we obtain

$$S_k(t) \simeq -\delta \frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi_\infty I + O(\phi_\infty^2) + O(\sigma), \quad (14)$$

where  $I$  is a finite and positive integral that has the form  $I = \int_0^t e^{\delta(t-t')} f(t') dt'$ . Setting this in Eq. (12) and expanding the exponential to the relevant order in  $\phi_\infty$ , we obtain

$$0 = \left[ \lambda \frac{\langle k \eta(k) \varphi(k) \rangle}{\langle k \eta(k) \rangle} - \delta \right] \phi_\infty - \frac{\lambda^2 \langle k^2 \eta^2(k) \varphi(k) \rangle}{\langle k \eta(k) \rangle^2} \phi_\infty^2 + O(\sigma^2) + O(\phi_\infty^3). \quad (15)$$

The nonzero solution of the equation above is given by

$$\phi_\infty = \frac{\lambda \frac{\langle k \eta(k) \varphi(k) \rangle}{\langle k \eta(k) \rangle} - \delta}{\lambda^2 \frac{\langle k^2 \eta^2(k) \varphi(k) \rangle}{\langle k \eta(k) \rangle^2} (1/2 + \sigma \delta \frac{\langle k \eta(k) \varphi(k) \rangle}{\langle k \eta(k) \rangle} I)}. \quad (16)$$

In order to have a positive value for  $\phi_\infty$ , the condition

$$\frac{\lambda}{\delta} \geq \frac{\langle k \eta(k) \rangle}{\langle k \eta(k) \varphi(k) \rangle} \quad (17)$$

must be fulfilled. Thus, for leading order in  $\sigma$ , the critical threshold is independent of the stifling mechanism, and for  $\delta = 1$ , the critical threshold is given by

$$\lambda_c = \frac{\langle k \eta(k) \rangle}{\langle k \eta(k) \varphi(k) \rangle}. \quad (18)$$

If  $\lambda$  is below  $\lambda_c$ , the rumor dies out, whereas, if  $\lambda$  is above  $\lambda_c$ , the rumor spreads on the network. For different rumor spreading models in the real world, different  $\varphi(k)$  and  $\eta(k)$  should be adopted. But in this paper, we only take the nonlinear (power-law) function form into account in the next section.

Finally,  $R$  is given by

$$R = \sum_k p(k) \left\{ 1 - \exp \left[ -\frac{\lambda k \eta(k)}{\langle k \eta(k) \rangle} \phi_\infty \right] \right\}. \quad (19)$$

The solution to the above equation depends on the form of  $p(k)$ .

#### A. The epidemic threshold for the generalized model of a rumor with nonlinear infectivity and nonlinear CSF on SF networks

In this model, we assume that  $\varphi(k) = k^\alpha$  where  $0 < \alpha \leq 1$ ; it means that each spreader can establish contacts with  $k^\alpha$  neighbors within one time step. The exponent  $\alpha$  will control the infectivity among nodes with different degrees. Since  $0 < \alpha \leq 1$ , it can be balanced to make the contacts fall into a more realistic range. Also, the node infectivity will grow nonlinearly by increasing degree  $k$ .

Furthermore, we suppose that  $\eta(k) = ak^\beta$ , where  $a$  is a positive quantity and  $\beta$  is a real exponent. So, according to Eq. (5), the spreading rate is  $\lambda_{kl} = \lambda \frac{(k)l^\beta}{(k^{1+\beta})}$ . The exponent  $\beta$  allows the tuning of the dependence of the transmission process on the node's degree. When  $\beta \neq 0$ , in the random transmission of the rumor, we introduce a bias toward high-degree ( $\beta > 0$ ) or low-degree (when  $\beta < 0$ ) neighbors. Also, when  $\beta = 0$ , the standard (unbiased) spreading process is recovered. By setting

the above-mentioned  $\varphi(k)$  and  $\eta(k)$  in Eq. (18), we get  $\lambda_c$ , the epidemic threshold, of degree-biased transmission of the rumor on the network,

$$\lambda_c = \frac{\langle k^{\beta+1} \rangle}{\langle k^{\alpha+\beta+1} \rangle}. \quad (20)$$

Now, we consider the epidemic threshold in the case of general SF networks in which the degree distribution is  $p(k) = ck^{-2-\gamma}$ ,  $0 < \gamma \leq 1$ , where  $c$  is the normalization constant. For this purpose, we obtain  $\langle k^{\beta+1} \rangle = c(k_c^{\beta-\gamma} - m^{\beta-\gamma})/(\beta - \gamma)$  and  $\langle k^{\alpha+\beta+1} \rangle = c(k_c^{\alpha+\beta-\gamma} - m^{\alpha+\beta-\gamma})/(\alpha + \beta - \gamma)$ , where  $k_c(m)$  denotes the largest (smallest) degree in the underlying network. By substituting these into Eq. (20), one can rewrite the epidemic threshold as follows:

$$\lambda_c = \frac{\alpha + \beta - \gamma}{\beta - \gamma} \frac{k_c^{\beta-\gamma} - m^{\beta-\gamma}}{k_c^{\alpha+\beta-\gamma} - m^{\alpha+\beta-\gamma}}. \quad (21)$$

From Eq. (21), one can see that, if the largest degree present in the network tends to infinity ( $k_c \rightarrow \infty$ , or equally,  $N \rightarrow \infty$  since  $k_c \propto N^{1/(\gamma+1)}$  [37]), the epidemic threshold  $\lambda_c$  tends toward zero if  $\gamma < \alpha + \beta$ ; on the other hand, if  $\gamma > \alpha + \beta$ , the epidemic threshold  $\lambda_c$  takes a finite value, given by

$$\lambda_c = m^{(-\alpha)} \frac{\alpha + \beta - \gamma}{\beta - \gamma}. \quad (22)$$

Thus, the critical border is  $\gamma = \alpha + \beta$ . One can adjust the infectivity's exponent  $\alpha$  and the CSF's exponent  $\beta$  to obtain a nonzero threshold for a given network (i.e., a fixed value of  $\gamma$ ).

Let us concentrate on the quantity  $\beta$ . For  $\beta > 0$ , if one chooses exponents  $\alpha$  and  $\beta$  such that  $0 < \alpha \leq 1$ ,  $\beta > 0$ , and  $\gamma > \alpha + \beta$ , one obtains the model of rumor spreading in which  $\lambda_c$  is a finite value above which a rumor spreads. As mentioned before, when  $\beta > 0$ , we have the model that exhibits the biased spreading of the rumor from low-degree nodes to high-degree nodes. We call this model a *down-up epidemic model*. On the other hand, for  $\beta < 0$  together with an allowable  $\alpha$  that satisfies the constraint  $\gamma > \alpha + \beta$  (for  $\beta \leq -\alpha$  the constraint  $\gamma > \alpha + \beta$  always is satisfied since  $0 < \gamma \leq 1$ ), the model has a finite critical threshold for the spreading rate above which a rumor can propagate in the system. In this case, we have the model in which a rumor is biased to spread from high-degree nodes toward low-degree nodes. We call this model an *up-down epidemic model*. We believe that these models are more remarkable than the models studied previously [29,30,33].

#### IV. THE EPIDEMIC THRESHOLD FOR THE GENERALIZED RUMOR SPREADING MODEL ON FINITE SF NETWORKS

In the real world, an epidemic always occurs on a finite network, although the size of the network may be very large. In Ref. [38], the authors studied the epidemic threshold  $\lambda_c(k_c)$  for the SIS model on bounded SF networks with the soft and hard cutoff  $k_c$  when  $\varphi(k) = k$  and  $\eta(k) = 1$ . The term hard cutoff denotes a network that does not possess any nodes with connectivity  $k$  larger than  $k_c$ , and the maximum connectivity  $k_c$  of any node is related to network age, measured as the number of nodes  $N$ ,

$$k_c = mN^{1/(1+\gamma)}, \quad (23)$$

where  $m$  is the minimum connectivity of the network. In this case, the normalized connectivity distribution has the form [38]

$$p(k) = \frac{(1 + \gamma)m^{1+\gamma}}{1 - (k_c/m)^{-1-\gamma}} k^{-2-\gamma} \theta(k_c - k), \quad (24)$$

where  $\theta(x)$  is the heaviside step function. Now, we apply the same argument for the epidemic threshold of the standard and generalized model of rumor spreading on a finite network. First, the standard model: For this model, we have  $\varphi(k) = k$  ( $\alpha = 1$ ) and  $\eta(k) = 1$  ( $a = 1, \beta = 0$ ), so the epidemic threshold  $\lambda'_c(k_c)$  is given by

$$\lambda'_c(k_c) = \frac{\langle k \rangle}{\langle k^2 \rangle} = \frac{\int_m^{k_c} k^{-1-\gamma} dk}{\int_m^{k_c} k^{-\gamma} dk} \simeq \frac{1 - \gamma}{\gamma m} (k_c/m)^{(\gamma-1)}. \quad (25)$$

From Eqs. (23) and (25), we obtain

$$\lambda'_c(N) \simeq \frac{1 - \gamma}{\gamma m} (N)^{(\gamma-1)/(\gamma+1)}. \quad (26)$$

If  $\gamma = 1$ , we find

$$\lambda'_c(N) \simeq 2[m \ln(N)]^{-1}. \quad (27)$$

Equations (26) and (27) show that the effective epidemic threshold approaches zero as the size of the network increases.

Second, the generalized model: We consider the epidemic threshold  $\lambda_c^*(k_c)$  for  $\varphi(k) = k^\alpha$ ,  $0 < \alpha < 1$ , and  $\eta(k) = ak^\beta$  where  $a > 0$  and  $\beta$  is a real number. After a similar calculation, one obtains

$$\begin{aligned} \lambda_c^*(k_c) &= \frac{\int_m^{k_c} k^{\beta-1-\gamma} dk}{\int_m^{k_c} k^{\alpha+\beta-1-\gamma} dk} \\ &= m^{(-\alpha)} \frac{\alpha + \beta - \gamma}{\beta - \gamma} \frac{[(k_c/m)^{\beta-\gamma} - 1]}{[(k_c/m)^{\alpha+\beta-\gamma} - 1]}. \end{aligned} \quad (28)$$

Note that, as the size of the network increases, the ratio  $(k_c/m)$  becomes sufficiently large [see Eq. (23)] such that, when  $\alpha + \beta < \gamma$ , we have  $(k_c/m)^{\beta-\gamma} = (k_c/m)^{\alpha+\beta-\gamma} = 0$  and  $\lambda_c^*$  has a positive value, otherwise,  $\lambda_c^*$  is going to approach zero. Let us focus on the up-down epidemic model ( $\beta < 0$ ). Thus, the above equality can be rewritten as follows:

$$\lambda_c^*(k_c) = \begin{cases} m^{(-\alpha)} \frac{\alpha+\beta-\gamma}{\gamma-\beta} (k_c/m)^{\gamma-\alpha-\beta}, & \alpha + \beta > \gamma \\ m^{(-\alpha)} \frac{\gamma-\alpha-\beta}{\gamma-\beta}, & \alpha + \beta < \gamma. \\ m^{(-\alpha)} \frac{1}{\alpha \ln(k_c/m)}, & \alpha + \beta = \gamma \end{cases} \quad (29)$$

Combining Eqs. (23) and (29), we have

$$\lambda_c^*(N) = \begin{cases} m^{(-\alpha)} \frac{\alpha+\beta-\gamma}{\gamma-\beta} (N)^{(\gamma-\alpha-\beta)/(\gamma+1)}, & \alpha + \beta > \gamma \\ m^{(-\alpha)} \frac{\gamma-\alpha-\beta}{\gamma-\beta}, & \alpha + \beta < \gamma. \\ m^{(-\alpha)} \frac{\gamma+1}{\alpha \ln(N)}, & \alpha + \beta = \gamma \end{cases} \quad (30)$$

It is obvious, from the equations above, that the positivity of the critical value  $\lambda_c^*(N)$  is unrelated to size  $N$  of the network when  $\alpha + \beta < \gamma$  [second term in Eq. (30)], and it is the same as the critical threshold  $\lambda_c$  for the infinite SF network [see Eq. (22)].

To compare models under condition  $\alpha + \beta > \gamma$ , we take the ratio of Eq. (26) and the first term in Eq. (30),

$$\frac{\lambda'_c(N)}{\lambda_c^*(N)} = \frac{(1 - \gamma)(\gamma - \beta)}{\gamma m^{(1-\alpha)} (\alpha + \beta - \gamma) N^{(1-\alpha-\beta)/(\gamma+1)}}. \quad (31)$$

It is straightforward that  $\frac{\lambda'_c(N)}{\lambda_c^*(N)} < 1$  when the size of network  $N > N_0$ , where  $N_0$  is a positive integer, means that the epidemic threshold  $\lambda_c^*(N)$  is greater than  $\lambda'_c(N)$  on finite SF networks with the same size  $N > N_0$ , so an epidemic rumor has more difficulty in propagation for the case  $\{\varphi(k) = k^\alpha, \eta(k) = ak^\beta, \beta < 0, \alpha + \beta > \gamma\}$  than for the case  $\{\varphi(k) = 1, \eta(k) = 1\}$  on finite SF networks with the same size.

Our method to study the generalized SIR [18,19], SIRS [39], and similar epidemic processes.

## V. MAXIMUM SPREADING

Regardless of the network topology and configuration, for any form of  $p(k)$ , Eq. (19) can be simplified by expanding the exponential for the first order in  $\phi_\infty$ , and one obtains

$$R \simeq \lambda \phi_\infty. \quad (32)$$

Throughout the rest of the paper, we set  $\lambda = 1$  without loss of generality and vary the value of other parameters. Since  $R$  will then be equal to the function  $\phi_\infty$ , in the following, we will concentrate on this function and will present some of its analytical properties. Later on, we will check our analytical results by simulating the rumor model on different networks.

One important question is that, under which conditions will exponents  $\alpha$  and  $\beta$  lead to maximum spreading of a rumor? This maximum simply is given by the final density of stiflers and is called reliability of the rumor process. For long enough times, Eq. (32) determines the percent of people that have heard the rumor. So the value of  $\beta$  that maximizes  $\phi_\infty$  results in maximum spreading of a rumor. We denote  $\Xi(k) = k\xi(k) = k^{\beta+1}$ , so Eq. (16) reduces to

$$\phi_\infty = \frac{\langle \Xi(k) \rangle^2 \langle \Xi(k) \varphi(k) \rangle - \delta \langle \Xi(k) \rangle^3}{\langle \Xi^2(k) \varphi(k) \rangle \left[ \frac{1}{2} \langle \Xi(k) \rangle + \sigma \delta I \langle \Xi(k) \varphi(k) \rangle \right]}. \quad (33)$$

We need to find the derivative  $\partial \phi_\infty / \partial \beta$  for finding the maximum. By the chain rule, we have

$$\frac{\partial \phi_\infty}{\partial \beta} = \frac{\partial \phi_\infty}{\partial \Xi} \frac{\partial \Xi}{\partial \beta}. \quad (34)$$

The function  $\phi_\infty$  can be maximized if  $\partial \phi_\infty / \partial \Xi = 0$ . It can be shown that a possible solution is  $\partial_k \Xi = 0$  (namely,  $\Xi$  should not depend on variable  $k$ ). More accurately, when  $\partial_k \Xi = 0$ , Eq. (21) reduces to

$$\phi_\infty = \frac{\langle \varphi(k) \rangle - \delta}{\langle \varphi(k) \rangle \left[ \frac{1}{2} + \sigma \delta I \langle \varphi(k) \rangle \right]}. \quad (35)$$

It is obvious that the function above does not depend on the function  $\Xi$ , so the functional equation  $\partial \phi_\infty / \partial \Xi = 0$  always is satisfied. For the case where  $\Xi(k) = k^{\beta+1}$ , the condition  $\partial_k \Xi = 0$  leads to  $\beta = -1$ . So, the maximum amount of rumor spreading occurs when  $\beta = -1$ . This result indicates that nodes with smaller degrees play a significant role in rumor spreading. More precisely, it says a spreader node has a number

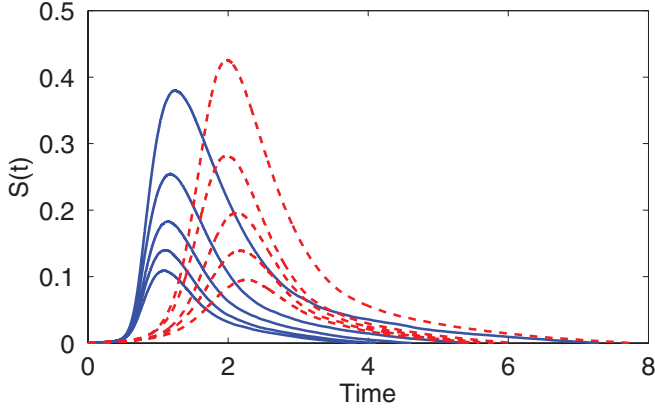


FIG. 1. (Color online) Time evolution of the density of spreaders for different values of  $\sigma$  for both the standard rumor model (solid-blue curves) and the generalized rumor model with  $\beta = -1$  (dashed-red curves). Standard SF network of size  $N = 2000$  with  $\langle k \rangle = 8$ . The values of  $\sigma$  from below go from 1.0 to 0.2 at fixed increments of 0.2. The other parameters are  $\{\alpha = 1, \lambda = 1, \delta = 0.5\}$ .

of connected neighbors with different degrees, and for a given time step, a neighboring node with the smaller degree will be chosen with higher probability ( $\lambda_{kl} = \lambda \frac{k^l \beta}{k^{l+\beta}}$ ), so as to maximize the final stifter density.

In a similar way, we investigate the variation in  $\phi_\infty$  with respect to  $\alpha$  ( $0 < \alpha \leq 1$ ) for a fixed value of  $\beta$ . We take the functional derivative  $\partial\phi_\infty/\partial\alpha$ . It can easily be shown that, in the range of  $0 < \alpha \leq 1$ ,  $\phi_\infty$  has a negative slope,  $\partial\phi_\infty/\partial\alpha < 0$ , so, in this range, the function  $\phi_\infty$  is a decreasing function. In other words, by decreasing  $\alpha$ , the final population that has heard the rumor increases.

VI. SIMULATION RESULTS AND DISCUSSION

We consider a standard SF network. The network has been generated according to  $p(k) \sim k^{-3}$ , the number of nodes is  $N = 2000$ , and the average degree is  $\langle k \rangle = 8$ . In the following and throughout the paper, all calculations reported are performed by starting the rumor from a randomly chosen

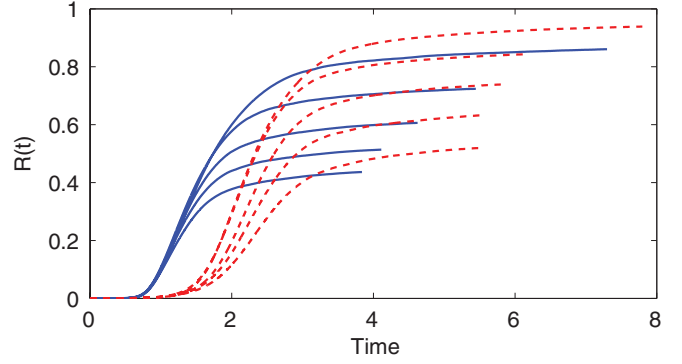


FIG. 2. (Color online) Time evolution of the density of stifiers for different values of  $\sigma$ . The dashed-red (solid-blue) curves are the stifier density for the generalized model (standard model). At the end of the epidemic in the generalized model, society reaches a higher level of rumor spreading than in the standard model.

initial spreader. One of the most important practical aspects of any rumor-mongering process is whether or not it reaches a large number of individuals who have heard the rumor.

Figures 1 and 2 show the time evolution of the density of spreaders and stifiers, respectively, for different values of the stifling process rate  $\sigma$  when the forgetting process rate is  $\delta = 0.5$ . Figure 1 shows that, as expected, the number of individuals who spread the rumor increases as the stifling process rate  $\sigma$  decreases. Also, in the generalized model, a larger population spreads the rumor than the standard model. Interestingly, Fig. 2 shows that the final densities of the population who have heard the rumor (or stifiers) in the generalized model (red-dashed curves) are larger than that of the standard model (blue-solid curves).

To verify the maximization issue, in the active phase, we compute, for a number of different network topologies and configurations, the stifier density  $R$  vs  $\beta$  at the end of the epidemic. The results are summarized in Figs. 3(a)–3(c). Despite different network topologies [SF networks, small-world (SW) networks, and ER random graphs] and wide variations in network parameters, such as the size, the average degree, and the degree exponent, we observe that  $R$  is maximized

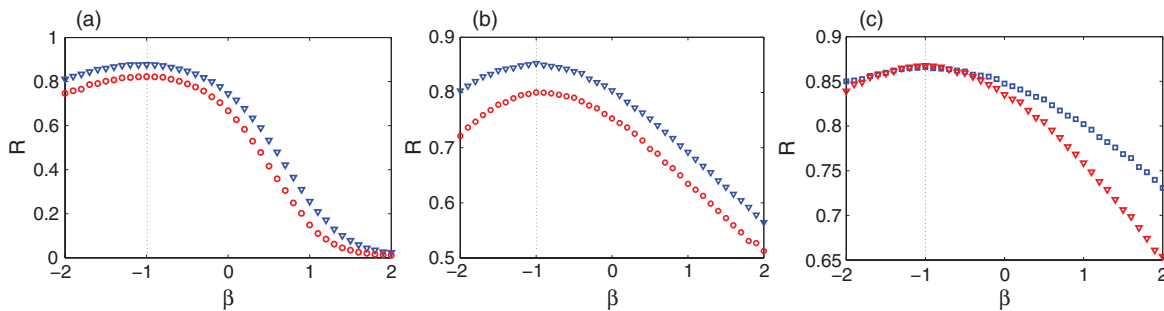


FIG. 3. (Color online) Density of stifiers  $R$  as a function of  $\beta$  at the end of the epidemic. (a) Standard SF networks of size  $N = 2000$  (triangles) with average degree  $\langle k \rangle = 10$  and  $N = 5000$  (circles) with  $\langle k \rangle = 12$ . The other rates for these two networks are  $\{\delta = 0.3, \sigma = 0.4\}$  and  $\{\delta = 0.4, \sigma = 0.6\}$ , respectively. (b) The ER random graphs of size  $N = 2000$  with  $\langle k \rangle = 5$  and  $N = 3000$  with  $\langle k \rangle = 6$ . (c) The SW networks with different degrees of randomness as determined by the rewiring probability  $p = 0.4$  (squares) and  $0.8$  (triangles). In (c), the network size and the average degree are fixed as  $N = 2000$  and  $\langle k \rangle = 6$ . The other reaction rates for all models in (b) and (c) are the same  $\{\delta = 0.3, \sigma = 0.4\}$ . Consistent with theoretical prediction, regardless of the network topology and configuration, the maximum value of stifier density, i.e., the number of individuals who have heard the rumor, evidently occurs for  $\beta = -1$ .

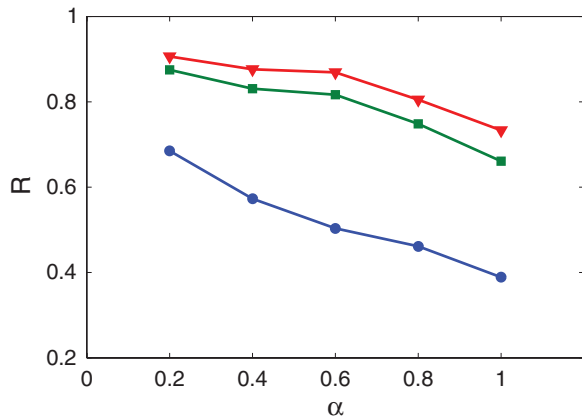


FIG. 4. (Color online) Density of stiflers  $R$  as a function of  $\alpha$  at the end of the epidemic. The SF network with size  $N = 2000$ ,  $\langle k \rangle = 10$ , and  $k_{\max} = 192$  (triangles) and the ER network with size  $N = 2000$ ,  $\langle k \rangle = 5$ , and  $k_{\max} = 14$  (circles). In both networks, the other parameters are  $\{\beta = -1, \delta = 1, \sigma = 0.7\}$ . The squares represent the SF network with size  $N = 2000$ ,  $\langle k \rangle = 10$ , and  $k_{\max} = 152$  by choosing  $\{\beta = -0.5, \delta = 1, \sigma = 0.5\}$ .

for  $\beta = -1$ . In these computations, we have kept  $\alpha$  fixed at  $\alpha = 1$ .

Figure 4 shows the density of stiflers as a function of  $\alpha$  in SF networks and ER random graphs. The curves have a negative slope as we predicted theoretically. By decreasing  $\alpha$ , the final population who have heard the rumor increases. This is a notable result and can be interpreted in the following way. We have three types of interaction in the network: the spreader-ignorant ( $SI \rightarrow SS$ ), the spreader-spreader ( $SS \rightarrow RS$ ), and the spreader-stifler ( $SR \rightarrow RR$ ) interactions. When exponent  $\alpha$  decreases, the ignorant is connected to a fewer number of spreaders simultaneously, and therefore, there is less probability that an ignorant converts into a spreader. So, by decreasing  $\alpha$ , we expect the number of spreaders that are created to decrease. But when one considers an  $SS$  ( $SR$ ) interaction, a decrease in  $\alpha$  would mean that a spreader meets fewer connected spreaders (stiflers) simultaneously, and therefore, the probability that a spreader becomes a stifler is smaller. In this case, we infer that the smaller  $\alpha$  is, the

longer a spreader spreads a rumor. The longer lifetime of the spreaders leads to further spreading of rumors in the network, and therefore, at the end of an epidemic, we have a larger final population who have heard the rumor. In general then, we conclude that a smaller  $\alpha$  leads to a larger density of stiflers.

## VII. CONCLUSION AND REMARKS

In this paper, we have studied the dynamical behavior of the generalized model of rumor spreading with a degree-biased transmission rate (the result of nonlinear CSF) and nonlinear infectivity. We have shown that one can adjust the infectivity's exponent  $\alpha$  and the CSF's exponent  $\beta$  to control the epidemic threshold that is absent for the standard rumor spreading model in SF networks. In the case of general infinite SF networks, we analytically showed that  $\beta < 0$  and  $\beta > 0$  lead to two different models, i.e., the up-down epidemic model (the biased spreading of the rumor toward low-degree neighbors) and the down-up epidemic model (the biased spreading of the rumor toward high-degree neighbors), respectively, in which critical threshold  $\lambda_c$  takes a positive value. Also, in the case of finite SF networks, we obtained the epidemic threshold  $\lambda'_c$  for the standard model and the epidemic threshold  $\lambda_c^*$  for the generalized model, we concluded that  $\lambda_c^*$  is a positive value when  $\alpha + \beta < \gamma$  and is unrelated to size  $N$  of the network. We showed that  $\lambda_c^*$  is greater than  $\lambda'_c$  (when  $\beta < 0$  and  $\alpha + \beta > \gamma$ ) on a finite SF network with the same size. Finally, we showed that  $\beta = -1$  leads to maximum spreading of the rumor theoretically, and computation results on different networks verify our theoretical prediction. Also, we showed that a smaller  $\alpha$  leads to larger spreading of the rumor. Our results are interesting since we obtain these results regardless of the network topology and configuration.

## ACKNOWLEDGMENTS

F.R. is deeply indebted to Silvio Franz for his continuous help, support, encouragements, Matteo Marsili and Giuseppe Mussardo for useful discussions, Nader Heydari for carefully reading of the manuscript. This work was supported by the research council of the Alzahra University.

- 
- [1] S. N. Dorogovtsev and J. F. Mendes, *Adv. Phys.* **51**, 1079 (2002).
  - [2] R. Albert and A.-L. Barabási, *Rev. Mod. Phys.* **74**, 47 (2002).
  - [3] S. H. Strogatz, *Nature (London)* **410**, 268 (2001).
  - [4] D. J. Watts and S. H. Strogatz, *Nature (London)* **393**, 440 (1998).
  - [5] M. E. J. Newman, *SIAM Rev.* **45**, 167 (2003).
  - [6] R. Pastor-Satorras and A. Vespignani, *Evolution and Structure of Internet: A Statistical Physics Approach* (Cambridge University Press, Cambridge, UK, 2004).
  - [7] B. A. Huberman, *The Laws of the Web* (MIT Press, Cambridge, MA, 2001).
  - [8] F. Liljeros *et al.*, *Nature (London)* **411**, 907 (2001).
  - [9] L. A. N. Amaral, A. Scala, M. Barthélemy, and H. E. Stanley, *Proc. Natl. Acad. Sci. USA* **97**, 11149 (2000).
  - [10] M. E. J. Newman, *Phys. Rev. E* **64**, 016131 (2001).
  - [11] H. Jeong *et al.*, *Nature (London)* **407**, 651 (2000).
  - [12] J. M. Montoya and R. V. Solé, *J. Theor. Biol.* **214**, 405 (2002).
  - [13] D. J. Daley and J. Gani, *Epidemic Modelling: An Introduction* (Cambridge University Press, Cambridge, UK, 2001).
  - [14] R. Pastor-Satorras and A. Vespignani, *Phys. Rev. Lett.* **86**, 3200 (2001).
  - [15] R. Pastor-Satorras and A. Vespignani, *Phys. Rev. E* **63**, 066117 (2001).
  - [16] M. Boguñá and R. Pastor-Satorras, *Phys. Rev. E* **66**, 047104 (2002).
  - [17] M. Boguñá, R. Pastor-Satorras, and A. Vespignani, *Phys. Rev. Lett.* **90**, 028701 (2003).
  - [18] R. M. May and A. L. Lloyd, *Phys. Rev. E* **64**, 066112 (2001).
  - [19] A. L. Lloyd and R. M. May, *Science* **292**, 1316 (2001).

- [20] Y. Moreno, R. Pastor-Satorras, and A. Vespignani, *Eur. Phys. J. B* **26**, 521 (2002).
- [21] S. Galam, *Physica A* **320**, 571 (2003).
- [22] A. J. Kimmel, *J. Behav. Fin.* **5**, 134 (2004).
- [23] M. Kosfeld, *J. Math. Econ.* **41**, 646 (2005).
- [24] D. Stauffer and M. Sahimi, *Eur. Phys. J. B* **57**, 147 (2007).
- [25] D. J. Daley and D. G. Kendal, *J. Inst. Math. Appl.* **1**, 42 (1965).
- [26] D. J. Daley and J. Gani, *Epidemic Modelling* (Cambridge University Press, Cambridge, UK, 2000).
- [27] D. P. Maki, *Mathematical Models and Applications, with Emphasis on Social, Life, and Management Sciences* (Prentice Hall, Englewood Cliffs, NJ, 1973).
- [28] B. Pittel, *J. Appl. Probab.* **27**, 14 (1987).
- [29] A. Sudbury, *J. Appl. Probab.* **22**, 443 (1985).
- [30] C. Lefevre and P. Picard, *J. Appl. Probab.* **31**, 244 (1994).
- [31] M. E. J. Newman, S. Forrest, and J. Balthrop, *Phys. Rev. E* **66**, 035101 (2002).
- [32] G. Csanyi and B. Szendroi, *Phys. Rev. E* **69**, 036131 (2004).
- [33] M. Nekovee, Y. Moreno, G. Bianconi, and M. Marsili, *Physica A* **374**, 457 (2007).
- [34] T. Zhou, J.-G. Liu, W.-J. Bai, G. Chen, and B.-H. Wang, *Phys. Rev. E* **74**, 056109 (2006).
- [35] X. Fu, M. Small, D. M. Walker, and H. Zhang, *Phys. Rev. E* **77**, 036113 (2008).
- [36] H. Zhang and X. Fu, *Nonlinear Anal. Theory, Methods Appl.* **70**, 3273 (2009).
- [37] R. Cohen *et al.*, *Phys. Rev. Lett.* **85**, 4626 (2000).
- [38] R. Pastor-Satorras and A. Vespignani, *Phys. Rev. E* **65**, 035108 (2002).
- [39] J. Z. Liu *et al.*, *J. Stat. Mech.* (2004) P08008.