Efficient target strategies for contagion in scale-free networks

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Organizations or individuals often have an incentive to target a certain number of agents to launch a contagion process effectively and efficiently, for example, sampling consumers in the diffusion of new products. We present an effective strategy for contagion in scale-free networks. The proposed strategy, *hub strategy*, calls for targeting mostly the highly connected nodes. The biased level implemented in this strategy characterizes its ability to identify hub nodes. We demonstrate that hub strategy can improve the contagion effects evidently. We find that biased level increases first with heterogeneity level of contagion network but decreases with that after a certain value, and decreases with initial adopter rate all the time. Moreover, degree correlations in contagion networks may reduce biased level.

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The spread of innovations (or decisions, new ideas, influences, diseases, opinions, etc.) through social networks can be viewed as dynamic processes in networks [1–6], in which nodes represent agents (individuals or organizations) and edges represent interactions among agents. One important aim of these models is to show how observable collective dynamics might arise from simple behavior of individual agents and their interactions [2,5]. These studies deepen our understanding of social and economic systems, and may be helpful to develop strategy and design policy to control and intervene these systems effectively. Interaction networks are often modeled as regular lattices except for Ref. [6]. But real social networks often exhibit small world effect and power-law degree distributions [7,8], examples including sexual contact network [9], corporate board network [10], etc. The topological structure of interaction networks is an essential factor to emerge out of collective dynamics. For instance, systems are very robust to randomly deleting nodes, even though the proportion is high, but fragile to intentionally removing the highest degree nodes in scale-free networks [11]. Another example is infectious diseases spreading more easily and quickly in scale-free networks than in regular lattices [12]. Therefore, exploiting the connectivity feature of interaction networks to optimize system behaviors is attracting the interests of researchers in many fields. Targeted vaccination strategy has been developed to prevent virus propagation [13,14]. Cascade-based networks dynamics, examples like cascade failure in power transmission grids, is very vulnerable to load and distribute heterogeneity and network heterogeneity. Considering the importance of network topology, researchers have developed strategies incorporating the network structure factor to optimize the robustness of systems to both random and targeted attacks [15,16].

Following the philosophy of the above literatures, this paper has proposed and investigated a strategy to promote decision spread in scale-free social networks. Considering a social system composed of many agents, each of them must decide between two alternative actions, and their decisions depend explicitly on the actions of other members of the population [17]. Decision makers have many reasons to pay attention to each other [18]: lacking of either related information or the ability to process available information; one agent's payoff being an explicit function of the actions of others in collective actions. Regardless of the details, individual decision makers have an incentive to pay attention to the decisions of others. Once certain number of agents has taken one action, their decisions would be observed by other agents and would influence them to take the same action. More and more agents would take that action because of their neighbors already having done that. In economic terms, a contagion process has happened to the system [17]. In many social and economic issues, the more agents adopt the expected action, the more effective and efficient the contagion process is. For example, the more consumers adopt the new product, the more incomes and profits firms can get from the new product diffusion.

Our research interests have mostly been focused on how to pick out a given number of agents as initial adopters in order to launch a contagion process effectively and efficiently. This issue is practical in many applications, such as how to increase new product market share by sending free products to a certain number of consumers [19], in order to persuade them to make the innovations spread widely, etc. [20]. Considering there exist some nodes with a prominent number of neighbors and these nodes may have more influences than others in scale-free contagion networks, we present an approach of targeting agents with most connections (hub nodes) as initial adopters, and this approach is called hub strategy. It is difficult to identify the hub nodes accurately because of incomplete information and uncertainty in social networks. In order to overcome this shortcoming, our hub strategy only requires targeting the higher degree nodes with higher probability, which is different from similar ideas of targeted immunization regime in Ref. [13]. Hub strategy is also different from the traditionally random approach or targeting agents based on their personality characteristics [19].

In order to investigate the hub strategy, we model the contagion process as a dynamic process that takes place in a scale-free network. We first construct a scale-free contagion network with N agents, in which each agent is connected to k neighbors with probability $P(k)$. For scale-free network, we

have degree distributions $P(k) \sim k^{-\tau}$, τ is a constant between 2 and 3. Next, we ignore the specific mechanisms involved in the contagion process and consider the decision to be a function of the relative number of other agents who are observed to choose one alternative over the other. Considering the threshold nature in decision process, the decision rules can be described as follows: each agent has two choices of state 0 and state 1, and observes the current states (either 0 or 1) of its k neighbors, and adopts state 1 if at least a threshold fraction ϕ of its k neighbors are in state 1, otherwise it adopts state 0. The decision threshold ϕ of each agent is drawn from a beta distribution of density functions $f(\phi|a,a)=\phi^{1-a}(1-\phi)^{1-a}I_{(0,1)}(\phi)/B(a,a), a \geqslant 1$ and distribution function $F(\phi,a)=\int_0^{\phi} f(x|a,a)dx$, where $B(a,a)$ is the beta function and a is the control parameter. It is evident that $a=1$ represents uniform distributions and $a>1$ can be viewed as a reasonable approximation of normal distributions truncated at $\phi=0$ and $\phi=1$. $F(0,a)=0$ and $F(1,a)=1$ mean all thresholds falling into the interval (0,1). Furthermore, different from the cascade model described in Ref. [21], we relax the assumption of once an agent switched to state 1 it would hold that all the time by permitting the agent to switch back from state 1 to state 0 with the probability of a small constant λ.

Initially, the population is all off (state 0). In order to launch the contagion process at time $t=0$, we pick out N_0 nodes as initial adopters and let them switch from state 0 to state 1. Therefore, the initial adopter rate is $R_0=N_0/N$. The probability of a node with degree k picked out as initial adopter $\rho_k(0)$ depends on its degree, i.e., $\rho_k(0) \sim k^{\alpha}/\Sigma k^{\alpha}P(k), \alpha \in [0,\infty)$. Considering the value of initial adopter rate R_0, we have

$$\rho_k(0) = \frac{k^{\alpha}}{\Sigma_k k^{\alpha} P(k)} R_0, \quad 0 \leqslant \alpha < \infty, \tag{1}$$

where α characterizes the policy's ability to identify hub nodes. In this framework $\alpha=0$ corresponds to the random targeting regime, which means each node has the same probability being targeted; $\alpha>0$ corresponds to a hub targeting regime which means that those nodes with more connections have higher probability of being targeted. After a small fraction R_0 of nodes switches on (state 1), the population then evolves at successive time steps with all nodes updating their states in random, asynchronous order according to the threshold decision rule being stated above.

Our model is very similar with Watts's cascade model [21], but differs from that in some important aspects. First, unlike that model, where the cascade process is perturbed by small fractional nodes changing their states and therefore the initial adopter rate is approximated to zero, the contagion process is intentionally launched, and the initial adopters occupy a certain rate in order to assure that the contagion rate is large enough. Contagion rate is defined as the rate of nodes whose state is 1 in the stationary state. Second, Watts's model assumes agents would remain in state 1 for the duration of the dynamics once they were switched on, but our model permits agents to switch back to state 0 if they are already in state 1. This point makes our model more practical in modeling the contagion process. Finally, unlike the paper exploring the conditions of triggering global cascade, our paper aims at launching a contagion process effectively and efficiently by implementing hub strategy.

Let $\rho_k(t)$ be the relative density of nodes being in state 1 with given connectivity k and $\theta(t)$ the probability of any given edge point to a node being in state 1 at time t. Considering a node being in state 0 with degree k and having exactly a neighbors being in state 1, $F(a/k)$, the probability of that node switching from state 0 to state 1 influenced by its sneighbors' decisions, satisfies with $F(a/k)=P(\phi \leqslant a/k)$. The probability of a node with degree k having exactly a neighbors being in state 1, conforming to binomial distributions, is $\binom{k}{a}(\theta(t))^a(1-\theta(t))^{(k-a)}$. Therefore, $H_k(\theta(t))$, the probability of a node with degree k switching from state 0 to state 1 satisfies the following expression:

$$H_k(\theta(t)) = \sum_{a=0}^{k} F(a/k)\binom{k}{a}(\theta(t))^a(1-\theta(t))^{(k-a)}. \tag{2}$$

According to the mean field method, we can write the contagion dynamic equation as

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t)\lambda + (1-\rho_k(t))H_k(\theta(t)),$$

$$\rho_k(t)|_{t=0} = \rho_k(0) = \frac{k^{\alpha}}{\sum_k k^{\alpha} P(k)} R_0, \tag{3}$$

where $\theta(t)=(1/\langle k \rangle)\Sigma_k kP(k)\rho_k(t)$. Equation (3) does not consider degree correlations in contagion networks, there must exist some limitations. However, we have relaxed the homogeneity assumption on the node's connectivity usually implemented in regular networks. Moreover, we have also investigated correlated scale-free networks by numerical simulations.

Contagion dynamic equations (3) are infinite-dimensions nonlinear systems. The attractor structure in these systems is very interesting and still unclear in the mathematical community. It is difficult to get analytical results. Therefore, we further study hub strategy by extensively numerical simulations. Let contagion rate ρ be the average density of the adopted nodes in the stationary state in which the ratio of adopted nodes varies less than a given value (in most simulations, this value is 0.001). By numerical simulations, we find ρ is relevant to α. This behavior might arise from sparsely heterogeneous network and finite simulation time steps (we end the simulation if the variation is less than 0.001), but we still do not assure that. Furthermore, for $\lambda > 0$, the only genuine attractor of the dynamics is the "ground state" where all nodes are inactive. With nonzero probability $\lambda^{\rho N}$ all active nodes are deactivated at the same time. So after an INFINITE amount of time, all activity must die out. However, it turns out that there are metastable states with an extremely long lifetime in the presence of the random deactivation process. Which of these metastable states

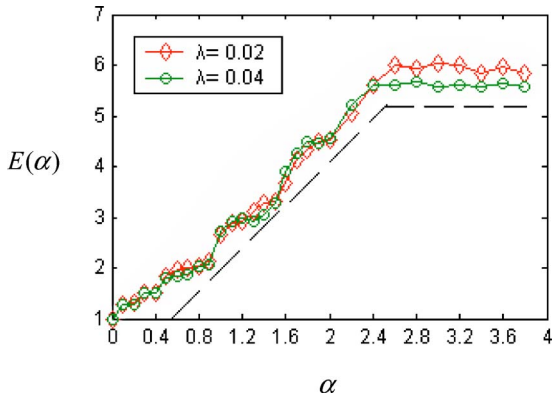

FIG. 1. (Color online) Improvement effects $E(\alpha)$ as a function of α with network size $N=1000$ and initial adopter rate $R_0=0.015$. The specific value of α corresponding to the highest improvement effects is called the biased level, and it is about 2.5. Numerical simulations have been averaged over 100 random scale-free networks with power law index $\tau=2$–3.

is reached is highly dependent on the "contagion strategy," i.e., on the parameter α. We report numerical simulation results in the following.

Improvement effects of hub strategy: Hub strategy requires targeting agents with the most connections, and α characterizes its ability to identify hub nodes. Let $\rho(\alpha)$ be the contagion rate for the given α, we define $E(\alpha)=\rho(\alpha)/\rho(0)$ as improvement effects that characterize how much the contagion effects can be improved by implementing hub strategy, where $\rho(\alpha)$ corresponds to the contagion rate for the hub targeting regime and $\rho(0)$ corresponds to the contagion rate of the random targeting regime. As shown in Fig. 1, improvement effects $E(\alpha)$ increase proportionally with α first but keep little variation when α is larger than a certain value (this specific value is called biased level). This is reasonable because increasing α can target more hub nodes when α is small. When most hub nodes are already targeted, there is no improvement derived from increasing α continually. Because biased level is the value of α which corresponds to the highest contagion rate ρ [or improvement effects $E(\alpha)$], it is necessary to implement hub strategy with this value in reality applications. In Fig. 1, this value is about 2.5. It should be pointed out that the value of biased level would be adjusted by other factors discussed in the following paragraphs.

Network heterogeneity level and hub strategy: Contagion network structure represents the influence pattern among agents. A node's degree represents not only how many nodes it can influence but also which nodes can influence it. For scale-free networks with degree of distributions $P(k)\approx\Phi_k^{-\tau}$ (Φ is a normalized factor), parameter τ characterizes the heterogeneity level of the contagion network [21]. Figure 2(a) shows the relationship between contagion rate ρ and network heterogeneity level τ. Given the value of biased level α, ρ increases with τ but decreases with that while τ is larger than a certain value. Therefore, there must exist an optimal value of τ and α so that the combination could maximize contagion rate ρ. Figure 2(b) shows the relationship between network heterogeneity level τ and corresponding biased level α. Intuitively, it is necessary to increase α so as to target hub

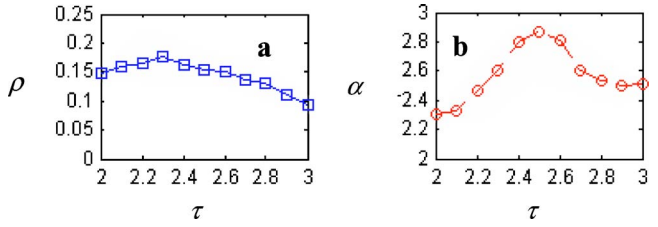

FIG. 2. (Color online) (a) Contagion rate ρ as a function of network heterogeneity level τ with $\alpha=2.5$. Because of ρ increasing first and decreasing later with τ for given α, there must exist certain relationships between heterogeneity level τ and biased level α. (b) The relationship between biased level α and corresponding heterogeneity level τ with $N=1000$ and $R_0=0.015$. Scale-free networks with $\tau=2$–3 are realized with the extended BA model [22].

nodes more accurately when τ is small, and small α can also assure targeting most hub nodes for high heterogeneous networks. Results shown in Fig. 2(b) have demonstrated this intuition. Our research results suggest that we should adjust the biased level α according to heterogeneity level τ of the contagion network at hand so as to obtain the best contagion effects.

Initial adopter rate and hub strategy; In order to launch a contagion process, we target a certain rate of agents (namely initial adopter rate) as initial adopters to influence the decisions of the remaining agents. Our results show improvement effects $E(\alpha)$ decrease with initial adopter rate R_0 simply due to saturation effects. By investigating the relationship between initial adopter rate and hub strategy, we find that the biased level α of hub strategy also decreases proportionally with initial adopter rate R_0 (as Fig. 3 shows). Considering that R_0 is about 0.01 in many economic problems [19], the appropriate biased level is 1.5–2.5.

Degree correlations and hub strategy: Considering that many social networks are assortatively mixed [23], we have further investigated the uncorrelated and correlated scale-free networks by comparing the biased level implemented in hub strategy. Random scale-free networks with power law index in the range of 2 to 3 are realized with an extended Barabási and Albert model (BA) [22]. In this model, the end points of the new edges are chosen according to a mixture of probabil-

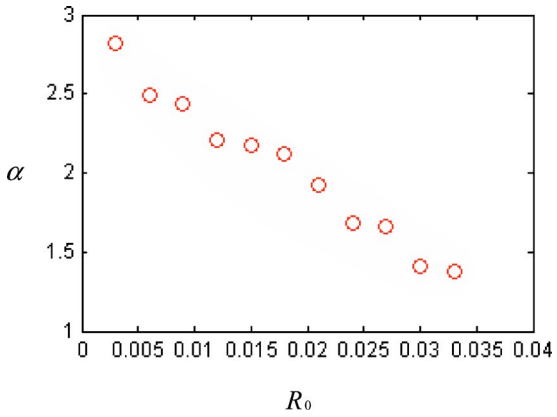

FIG. 3. (Color online) The relationship between initial adopter rate R_0 and corresponding biased level α.

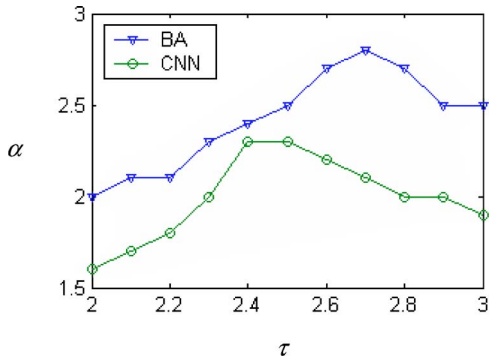

FIG. 4. (Color online) Biased level α corresponding to network heterogeneity level τ for uncorrelated network (BA) and correlated network (CNN) with $N=1000$ and $R_0=0.02$. Simulations are averaged over 100 realizations.

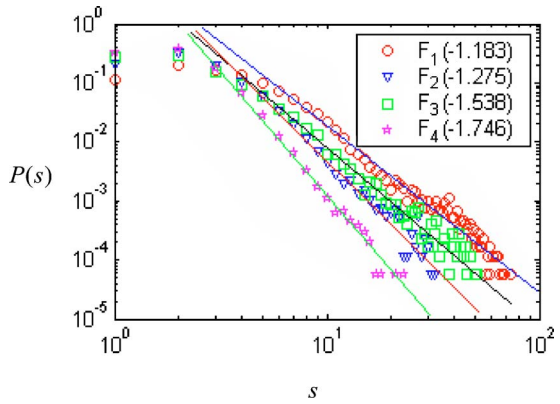

FIG. 5. (Color online) Plot of the probability density of having a cascade of size s in log-log scale for threshold function $F_1=F(\phi,1)$, $F_2=F(\phi,2)$, $F_3=F(\phi,3)$, and $F_4=F(\phi,4)$ with $N=1000$. The standard deviations of decision threshold for each threshold distribution function are 0.2882, 0.2235, 0.1837, and 0.1676, respectively. We fit the curves roughly and find that $P(s)\sim s^{-b}$, where b (labeled in the parentheses) decreases with the standard deviations of the threshold function. The peaks at the end of the each curve are due to finite size effects. Numerical simulations have been averaged over 100 realizations.

ity γ for preferential attachment and $1-\gamma$ for uniform attachment. And τ is a function of γ. Correlated networks with the same power law index are realized with the connecting nearest-neighbor model (CNN) [24]. Networks are generated by iteratively performing the following rules: (1) introduce a new vertex with probability $1-\mu$ and create an edge from the new vertex to a vertex j selected at random; (2) convert one potential edge selected at random with probability μ into an edge. We can modulate μ to get required τ. As Fig. 4 shows, the characteristics of degree correlations in contagion networks reduce the biased level for each τ. This result suggests that the biased level should take a smaller value in correlation networks than in random scale-free networks.

Threshold function and hub strategy: We have not found distinctly any qualitative difference in different threshold functions that conform to the beta distributions. We further investigated the distribution of the contagion cascade. As shown in Fig. 5, cascade distributions can be roughly fitted by power law distribution and the scale index (as labeled in the parentheses) decreases with standard deviation of the agents' decision thresholds. Therefore, the heterogeneity of decision threshold distribution may affect the time scale for the contagion dynamics.

In summary, we have proposed an efficient strategy to launch a contagion process—hub strategy, which exploits the scale-free characteristic of the contagion network. By extensive numerical simulations, we find that the biased level implemented in the hub strategy should be adjusted according to network heterogeneity level and initial adopter rate. Moreover, degree correlations in the contagion network also reduce the biased level. Threshold function impacts on the time scale of the contagion dynamics, but not on the stationarity results. Implications of our results for launching the contagion process are that (1) we should target hub nodes with certain accuracy, (2) implementing the hub strategy must take into account specific contexts, especially contagion network and initial adopter rate, etc. Our research demonstrates that statistical mechanics may be a good approach to study social and economic problems.

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