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Disease spreading on fitness-rewired complex networks

Jaewan Yoo, J.S. Lee, B. Kahng *

Department of Physics and Astronomy, Seoul National University, Seoul 151-747, Republic of Korea

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ABSTRACT

As people travel, human contact networks may change topologically from time to time. In this paper, we study the problem of epidemic spreading on this kind of dynamic network, specifically the one in which the rewiring dynamics of edges are carried out to preserve the degree of each node (called fitness rewiring). We also consider the adaptive rewiring of edges, which encourages disconnections from and discourages connections to infected nodes and eventually leads to the isolation of the infected from the susceptible with only a small number of links between them. We find that while the threshold of epidemic spreading remains unchanged and prevalence increases in the fitness rewiring dynamics, meeting of the epidemic threshold is delayed and prevalence is reduced (if adaptive dynamics are included). To understand these different behaviors, we introduce a new measure called the "mean change of effective links" and find that creation and deletion of pathways for pathogen transmission are the dominant factors in fitness and adaptive rewiring dynamics, respectively.

1. Introduction

Epidemics is a dynamic process in which a pathogen is transmitted from an infected host to a susceptible host through contact between the two hosts. Therefore, the spread of infection or disease can be modeled as successive contact processes in a population. Using this fact, one of the simplest models for epidemic spread is the susceptible–infected–susceptible (SIS) model in which each individual can be in either one of the two states, susceptible or infected. In this model, susceptible individuals change into infected individuals through contact with an infected individual at a given rate p; the infected individual becomes susceptible again at a rate r. On the other hand, in the susceptible–infected–recovered (SIR) model, infected individuals change to the (unchangeable) recovered state, which can be thought of as an immunized state, at some given rate r [1–3]. Here, the two parameters p and r are reduced to a single parameter in terms of the relative infectivity, $\lambda \equiv p/r$. Using these models, one can investigate the time evolution of patterns in the infected region and either the average fraction of infected individuals at the steady state (in the SIS model) or the total number of infected individuals during the simulation (in the SIR model).

The patterns of disease spreading and their statistical outcomes in these dynamic contact models are highly affected by the topological properties of the underlying contact network in which hosts and contacts represent nodes and links, respectively. In the early stages of epidemic study, it was assumed that contact networks were either homogeneous or fully mixed, which means that an infective individual is equally likely to spread the disease to others. However, several studies have since revealed that many social networks and technological networks transferring data, including the Internet and the World Wide Web, have heterogeneous contact-number distributions that typically show a power-law distribution $P(k) \sim k^{-\gamma}$, where k is the number of contacts (degrees) of each individual [4,5]. This heterogeneous network pattern dramatically influences the behavior of dynamic processes of disease spread because the epidemic threshold depends on the

* Corresponding author. Tel.: +82 2 880 6586; fax: +82 2 884 3002.
E-mail addresses: bkahng@snu.ac.kr, kahng@phya.snu.ac.kr (B. Kahng).

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second moment $\langle k^2\rangle$ [6–11]. For example, the epidemic threshold in the SIR model, which is given by $\lambda_c^{\mathrm{SIR}} = \langle k\rangle/(\langle k^2\rangle - \langle k\rangle)$, is zero when the degree distribution is scale-free, that is, $P(k) \sim k^{-\gamma}$ where $2 < \gamma < 3$, in the thermodynamic limit, whereas the threshold has a finite value when $\gamma > 3$ or the distribution is homogeneous. In the SIS case, it was recently claimed that the epidemic threshold is zero in the thermodynamic limit, regardless of whether the distribution is scale-free or not [12,13]. According to the research, the epidemic threshold $\lambda_{c,N}$ in a network with a finite system size N approaches zero as fast as the power law for scale-free networks, whereas it approaches zero logarithmically slowly in Erdös–Rényi networks [14].

Although such results are theoretically interesting, further studies were needed to allow application to actual disease spreading phenomena. As people travel and meet others (personally or without any intention) in common places, social contact networks, in which the epidemic spreading problem has yet to be studied, change topologically from time to time. Therefore, recent research has focused on the epidemic spreading problem in this kind of dynamically changing network [15–19]. For example, Gross et al. [20] introduced an adaptive model in which susceptible nodes are allowed to disconnect edges linked to infected nodes; next, the disconnected edges are randomly rewired to other susceptible nodes [21,22]. Because this adaptive rewiring mechanism separates the whole network into susceptible and infected clusters, called the "isolation effect", the spread of disease toward susceptible nodes is suppressed. As a result, the epidemic threshold becomes larger than that of a static network. However, in this model, the original degree distribution is not preserved during the epidemic process and the degree distribution eventually approaches a Poisson-like distribution, regardless of the initial topology. This alteration of the network topology can lead to a change in the second moment $\langle k^2\rangle$, which can produce variation of the epidemic threshold. As these two effects, that is, isolation and change of the second moment due to alteration of degree distribution, were mixed together in the previous adaptive model, the isolation effect was not examined independently. On the other hand, in swapping models [23,24] the degree distribution is preserved. However, in swapping models, edge swapping takes place randomly, that is, without using an adaptive rewiring rule.

In this paper, we introduce edge rewiring dynamics that are based on individual fitness, preserve the degree of each node, and take into account individual adaptive behavior. We also examine the isolation effect independently. The obtained network preserves the degree distribution stochastically. We study the SIR and SIS models of epidemic spreading on these networks and find the following results: when edge rewiring is carried out based only on individual fitness and not adaptive dynamics, the epidemic threshold does not change; however, disease spreading prevails in both the SIR and SIS models on networks whose degree distributions follow a power law. When adaptive rewiring is also taken into account, on the other hand, reaching of the epidemic threshold is delayed and the prevalence, which is the fraction of people who have ever been infected, is reduced. However, this threshold delay is smaller than that obtained in the previous adaptive model. Based on this result, we suggest that the isolation of infected people is an effective strategy for preventing the spread of infectious disease.

2. Fitness rewiring method and modified adaptive model

Here, we introduce a rule of rewiring edges that is based on both individual fitness to preserve the number of contacts for each individual and adaptive behavior to avoid transmission of the pathogen from an infected neighboring person. Each node is assigned its own fitness, $p_i = i^{-\mu}/\sum_{j=1}^{N} j^{-\mu}$, where i is a node index and μ is a control parameter. A link between two nodes (i, j) is connected with a probability $f_{ij} = 1 - \mathrm{e}^{-2pNp_ip_j}$. Then, the constructed network is scale-free with the degree exponent $\gamma = 1 + 1/\mu$ [25]; its existing probability is given by

$$P_G(A) = \frac{1}{Z}\prod_{i<j}(1-f_{ij})^{1-A_{ij}} f_{ij}^{A_{ij}}, \tag{1}$$

where A_{ij} is an adjacency matrix element of the network. When the network is changed from A to A' by rewiring, the transition probability is denoted by $W(A \to A')$. The detailed balance is written as

$$W(A \to A')P_G(A) = W(A' \to A)P_G(A'). \tag{2}$$

If the above detailed balance is satisfied, f_{ij} is preserved. Because $\langle k_i\rangle = \sum_j f_{ij}$, the preservation of f_{ij} guarantees the preservation of $\langle k_i\rangle$. We can choose any appropriate form for the transition probability once the detailed balance condition is satisfied. For convenience, we use a transition probability of the following form (it is analogous to the conventional Metropolis algorithm used in the Monte Carlo simulation),

$$W(A \to A') = \min\left[\frac{P_G(A')}{P_G(A)}, 1\right] = \min\left[\frac{f_{mn}(1-f_{ij})}{(1-f_{mn})f_{ij}}, 1\right]. \tag{3}$$

Now, we consider the modified adaptive model in which the fitness rewiring method is included. The rewiring process of the modified adaptive model was performed in the following way. When we take into account the system's adaptive behavior, an existing link between two nodes (i, j) is selected randomly among the pairs containing an infected node and then the link between them is removed from the system. A link is also added between a disconnected pair of nodes (m, n) that do not contain an infected node, under the condition that f_{mn} satisfies condition (3), where $f_{ij} = 1 - \mathrm{e}^{-2pNp_ip_j}$, $f_{mn} = 1 - \mathrm{e}^{-2pNp_mp_n}$, $p_i = i^{-\mu}/\sum_{j=1}^{N} j^{-\mu}$ and p is a mean degree of a whole network. The rewiring process is schematically depicted in Fig. 1.

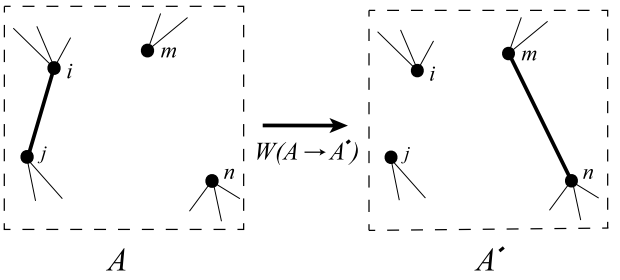

Fig. 1. Schematic diagram of fitness rewiring method. An existing link connecting nodes i and j, and a pair of nodes m and n where no link exists between them, are randomly selected when the adjacency matrix of a network is A. With the transition probability $W(A \to A')$ (see Eq. (3)), remove the existing link connecting nodes i and j and add a new link between nodes m and n, where A' is the adjacency matrix of the rewired network. When adaptive dynamics are added to the fitness rewiring method, there are some constraints for selecting nodes: at least one of the nodes i and j must be infected and no infected node is allowed to be selected as node m or n.

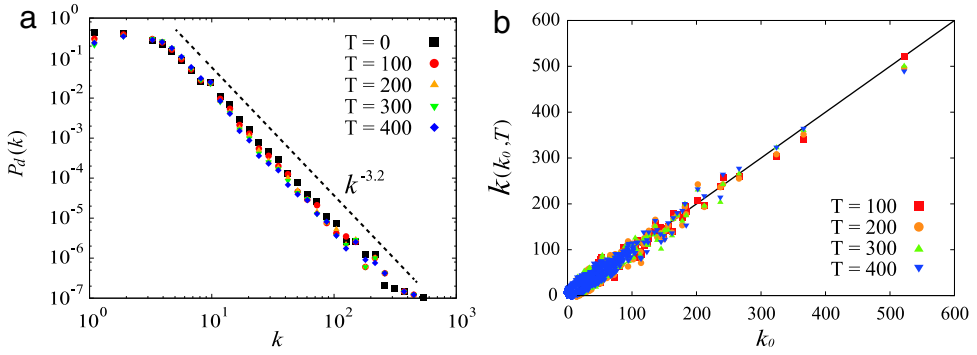

Fig. 2. (a) Degree distributions of original and rewired networks at various Monte Carlo times T. The figure shows the degree distribution is preserved under adaptive model with fitness rewiring method. (b) The relation between $k(k_0, T)$ and k_0 for various Monte Carlo times. Here, k_0 is the initial degree of a node in the original ($T = 0$) network and $k(k_0, T)$ is the degree of a node with initial degree k_0 at time T.

As the link updates satisfy the detailed balance condition, the Markov process conserves the given probability (1) and the resulting network maintains the same degree distribution stochastically. Moreover, the degree of each node in the rewired network fluctuates around its initial mean degree $2pNp_i$.

In simulations, one Monte Carlo step includes the rewiring of each link and a state update for every node. Each susceptible node can be infected when it is next to an infected node. Each infected node changes to a susceptible node (SIS) or a recovered node (SIR) at a given rate. We control the relative rate between epidemic spreading dynamics and edge rewiring process by introducing a parameter ω. In the simulation, for the deletion of an existing link and addition of a new link, we use the modified transition probability ωW instead of W which is defined by Eq. (3). Here, $\omega = 0$ corresponds to a fixed network because the transition probability is always zero. The simulations run until all infected people have recovered, in the SIR model, or the number of infected people reaches a steady state, in the SIS model. All simulations are carried out in a scale-free network with a degree exponent $\gamma = 3.2$, composed of system size $N = 10^5$, and with average degree $\langle k \rangle = 5.0$. All data are obtained after taking the average over 10^4 (10^2) realizations of the SIR (or SIS) model.

We first check if the degree distribution is preserved through the modified adaptive rewiring process. Fig. 2(a) shows the degree distributions of the original network and the rewired networks at various Monte Carlo steps. We can confirm that all the degree distributions of the rewired networks are preserved. Next, to check whether the fitness is also preserved, we investigate the relationship between the degrees of each node in the rewired network and those in the original network in Fig. 2(b). Indeed, the degrees in the rewired networks fluctuate around the corresponding degrees in the original network, indicating that the fitness of each node is preserved throughout the rewiring process.

3. Prevalence in the rewiring networks

In this section, we investigate the way in which the infectivity threshold (λ_c) changes as the network is rewired under preservation of individual fitness. Here, the term "prevalence" refers to the fraction of nodes that has ever been infected throughout the epidemic dynamics, in the SIR model, and are infected in the steady state, in the SIS model. Fig. 3(a) and (b) shows the numerical results for prevalence in the static and rewired networks (without using the adaptive rule) in the SIR model and the SIS model, respectively, in which the infectivity (λ) and the rewiring rate (ω) are controlled. Note that $r = 0.05$ in these simulations. As shown in the figure, it appears that the epidemic threshold does not change for either the SIR or the SIS models in this case. However, the prevalence increases as the rewiring rate ω increases in both models, which

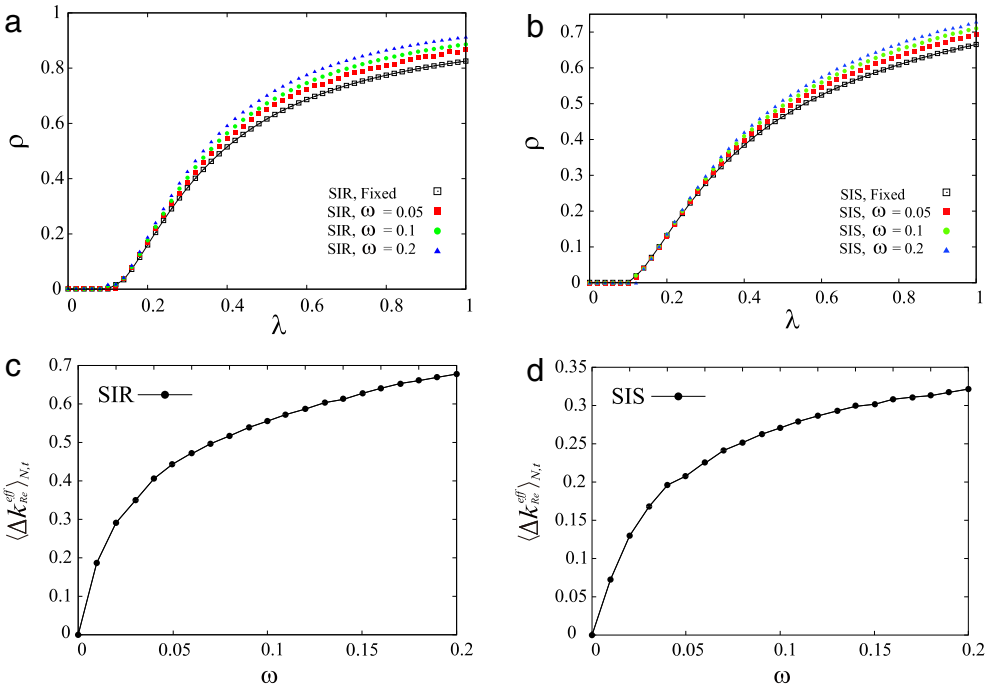

Fig. 3. (Color online) Prevalence (ρ) as a function of infectivity ($\lambda \equiv p/r$) is plotted (a) for the SIR model, and (b) for the SIR model for various rewiring rates (ω) on a dynamic network with fitness rewiring. Results for the static network (denoted by Fixed) correspond to the case in which the rewiring rate $\omega = 0$. Plot of mean change of effective links by rewiring ($\langle \Delta k_{Re}^{eff} \rangle_{N,t}$) versus rewiring rate ($\omega$) (c) for the SIR model, and (d) for the SIS model. Simulations are run on networks of system size $N = 10^5$, with $\gamma = 3.2$, and with $\langle k \rangle = 5$.

is the opposite effect from the one seen in previous rewiring models accompanying alteration of a network topology [26]. We can consider the increase of prevalence in the fitness rewiring model as being due to the creation of new paths for pathogens to pass through created by the rewiring process. To verify this argument, we introduce a new measure called the *mean change of effective link by rewiring* $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$. Here, the effective link refers to the link in which one end is infected and the other end is susceptible, so that infection can occur at the next time step. At the beginning of the simulation, Δk_{Re}^{eff} is set to zero. During the rewiring process, if the effective link is deleted (added), we subtract (add) 1 from (to) Δk_{Re}^{eff}; otherwise, nothing occurs. Once the simulation ends, we divide Δk_{Re}^{eff} by the number of nodes N and the total elapsed time t of the simulation, which results in the mean change of the effective link by rewiring $\langle \Delta k_{Re}^{eff} \rangle_{N,t} \equiv \Delta k_{Re}^{eff}/Nt$. Fig. 3(c) and (d) shows plots of $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$ versus the rewiring rate for both the SIR and SIS models. As shown in the figure, the effective link increases as the rewiring rate increases, which means that new pathways for pathogen transmission are created by fitness rewiring dynamics.

Next, we add adaptive dynamics to the fitness rewiring rule and obtain prevalence versus infectivity for the SIR and SIS models; the results are presented in Fig. 4(a) and (b), respectively. In this figure, our data are denoted by "Mod.", which refers to the modified adaptive model and data for the previous adaptive model are denoted by "Adp." As shown in Fig. 4(b), it is found that reaching the epidemic threshold is delayed as the rewiring rate increases in the SIS model. In the SIR model, it is not immediately obvious that reaching the threshold is delayed in Fig. 4(a), which shows the simulation result in which the exponent of the degree distribution of the network is 3.2. To show the delay in the SIR model more clearly, we also present a plot of prevalence versus infectivity when the exponent of the degree distribution of a network is set to another value, 4.0, as shown in the inset of Fig. 4(a). This behavior is reasonable in the sense that the link between infected and susceptible nodes is disconnected in the rewiring process. However, as shown in the figure, if we compare the delay of reaching the threshold here to that of the previous adaptive models (in which the degree distribution is not preserved), we can see that the delay in our case is relatively small. Moreover, the prevalence is also reduced compared to that of the static case. In the SIS model, an explosive epidemic transition takes place, which appears more distinctly as the rewiring rate increases. To understand prevalence reduction and threshold delay in this case, we also examine the change of the effective link in the SIR and SIS models; as shown in Fig. 4(c) and (d), $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$ decreases as the rewiring rate increases, which is the opposite effect to the one operating in the pure fitness rewiring model. This shows that the reduction of the effective link due to the isolation from the adaptive rewiring dynamics is much stronger than the increase in the effective link due to fitness rewiring dynamics. Therefore, isolation is an effective strategy for reducing the creation of pathways that allow pathogen transmission, and thus, for suppressing epidemic outbreak.

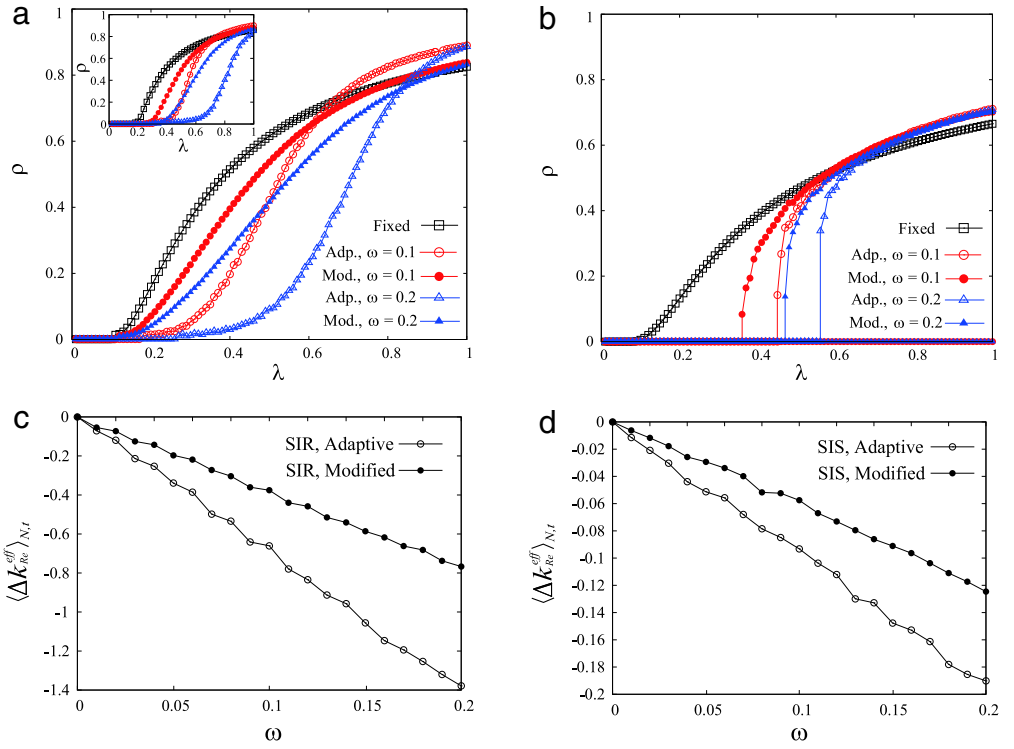

Fig. 4. (Color online) Prevalence (ρ) as a function of infectivity (λ) is plotted (a) for the SIR model, and (b) for the SIR model for various rewiring rates (ω) on the dynamic network with adaptive (denoted by Adp.) and fitness–adaptive rewiring (denoted by Mod.). Results for the static network (denoted by Fixed) correspond to the case in which the rewiring rate $\omega = 0$. Plot of mean change of effective links by rewiring ($\langle \Delta k_{Re}^{eff} \rangle_{N,t}$) versus rewiring rate ($\omega$) (c) for the SIR model, and (d) for the SIS model on the adaptive- and fitness–adaptive-rewired network. Simulations are run on networks of system size $N = 10^5$, with $\gamma = 3.2$, and with $\langle k \rangle = 5$. Inset: Prevalence (ρ) as a function of infectivity (λ) is plotted for the SIR model for various rewiring rates (ω) on the dynamic network with adaptive and fitness–adaptive rewiring when the exponent of degree distribution of the network is 4.0.

4. Conclusion and prospect

In this study, we introduce a new kind of edge rewiring dynamics called "fitness rewiring" that preserves the degree of each node and the degree distribution. On the dynamic network with fitness rewiring, we observe that the prevalence increases when infectivity increases for both the SIR and SIS models; however, their epidemic thresholds do not change. To understand the increase in prevalence, we introduce a new measure called the "mean change of effective link" by rewiring $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$. We find that $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$ increases as rewiring is strengthened, which means that pathways that allow pathogen transmission are created by the fitness rewiring process. We also take into account individual adaptive behavior in the fitness rewiring model to examine the isolation effect independently. In this case, we find that reaching the epidemic threshold is delayed and that the prevalence is reduced for both the SIR and SIS models. These results derive from the fact that $\langle \Delta k_{Re}^{eff} \rangle_{N,t}$ decreases as the rewiring rate increases, which means that the decrease of the effective link is caused by the isolation effect. From these simulation results, we can conclude that the suppression of disease spreading because of isolation of the network is a much stronger effect than boosting epidemic from fitness rewiring, which suggests that isolation is an efficient way of reducing the effective link and preventing the outbreak of an epidemic.

Acknowledgments

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