Mechanisms of recoverable prevalence and extinction of viruses on linearly growing scale-free networks

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We investigate mechanisms of the typically observed recoverable prevalence in epidemic spreading. Assuming the time-independent connectivity correlations, we analyze the dynamics of spreading on linearly growing scale-free (SF) networks, and derive the extinction condition related to the rates of network growth, infection, and immunization of viruses. The behavior is consistent with the previous results for SF networks by a mean-field approximation without connectivity correlations. In particular, it is suggested that the growing must be stopped to prevent the spreading of infection. This insight helps to understand the spreading phenomena on communication or social networks.

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In the world-wide communication and transportation, we are able to work on rapid consideration or decision for scientific, technological, social, and business trends, while we are also frightened by threat to dangerous viruses for computer systems and our body itself. The needs become larger for understanding the mechanism of epidemic spreading and for finding effective strategies of the prevention against huge damage in economy or life by computer viruses via e-mails [10] [21], human immunodeficiency virus (HIV) [12], severe acute respiratory syndrome (SARS) [13], and so on. For the purposes, the study of infection disease on network models is useful. In particular, we should remark that many real networks, whose node and link are corresponding to individual (computer or person) and the (communicational, pathological, or sexual) contact between them, have scale-free (SF) structures based on natural rules of network growth with new nodes and preferential attachment for linking as rich-get-richer phenomenon [2] [4], in which the degree distribution exhibits a power-law as $P(k) \sim k^{-\gamma}$, $2 < \gamma < 3$, for the probability of $k$ connections in address-books of e-mails [8] [9] or sexual partners [12].

It is very possible that there exist a common mechanism for epidemic spreading in realistic SF networks, and that the extremely heterogeneity consisted of many normal nodes with less connectivities and a few hubs with much connectivities is crucial for the superspreading [13] and the persistence of viruses in long-time [21]. Indeed, the important properties of SF networks are the robust and vulnerable connectivities against random and targeted attacks, respectively [1]. The property is applicable for the targeted immunization for hubs [7] [19] to prevent the spreading of infection. Thus, the universal and robust structure in growing complex networks attracts physicists, and surprising results have been recently obtained.

In striking contrast with the usual models for epidemic spreading [3], it has been shown [17] that a susceptible-infected-susceptible (SIS) model on heterogeneous SF networks has no epidemic threshold at a large network size nearly infinity; infection can be proliferated, whatever small infection rate they have. This result is closely related to the bond percolation problem. The epidemic threshold in a susceptible-infected-recovered (SIR) model coincides with the threshold for the bond percolation problem on networks [15]. On the other hand, in a closed system of the conventional susceptible-hidden-infected-recovered (SHIR) or SIR model, the number of infection is initially increased and saturated, finally converged to zero as the extinction. The pattern may be different in an open system, in fact, oscillations have been described by a deterministic Kermack-McKendrik model [20]. However a constant population (equal rates of the birth and the death) or territorial competition has been mainly discussed in the model, the growth of computer network or world-wide human contacts is obviously more rapid, and the communications of mailing or the transportation are not competitive. Even in a new report for modeling the SARS epidemic [13], the mechanism of widely rapid spreading is not clear enough, though further treatments for the heterogeneity in transmission through superspreaders has been pointed out and the effectiveness of quarantine has been discussed. The strategic quarantine in both the model and experiences in Hong Kong is potentially related to prevent the typically observed recoverable prevalence [10] [21] in an open system as growing with new individuals. Fig. 1 shows an example of typically observed pattern in the prevalence of computer viruses.

Thus, we consider a growing system on heterogeneous SF networks for e-mails or human contacts, and investigate the mechanisms of recoverable prevalence and extinction of infection. In the previous paper [9], we have derived the conditions of extinction for the spreading in deterministic SIR models on SF networks by a mean-field approximation without the connectivity correlations. Although the connectivity correlations are not found in all growing network models and real systems [18], they are at least quantitatively significant for the spreading on heterogeneous SF networks [5] [6] [11]. In this paper, we extend the previous results to correlated cases between
the degree of connections, and discuss the mechanisms of recoverable prevalence and extinction on linearly growing SF networks.

Heterogeneous SIR model.—On heterogeneous SF networks, we consider a deterministic SIR model \([14]\) with macroscopic equations for the number of states. The state transition is from susceptible, infected, to recovered or immune, whose numbers for each connectivity \(k\) are denoted by \(S_k(t) > 0\), \(I_k(t) > 0\), and \(R_k(t) > 0\), respectively. Microscopically, susceptible nodes stochastically become the infected by contacts with infected nodes, which are recovered by the detection or immunization. Assuming that infection sources exist in an initial small network and that both network growth and the spread of viruses are simultaneously progressed, we introduce a linear kernel \([11]\) as \(N_k(t) \sim a_k \times t\), \(N_k(t) = S_k(t) + I_k(t) + R_k(t)\), the growth rate \(a_k \overset{\text{def}}{=} Ak^{-\nu}\) for \(A > 0\), \(\nu > 2\). Note that the total \(N(t) = \sum_k N_k(t) \sim (\sum_k a_k \times t)\) means a linear growth of network size. Since the maximum degree increases as progressing the time and approaches to infinity, it has a nearly constant growth rate \(\sum_{k=m} a_k \sim \int_{m}^{\infty} Ak^{-\nu} dk = \frac{A m^{1-\nu}}{\nu - 1}\) for large \(t\). As shown in \([11]\), the introduction of linear kernel is not contradic-
able to the preferential (linear) attachment \([2][4]\).

We first consider a simple case with only the detection of viruses. The time evolutions of \(S_k\) and \(I_k\) for each class of the connectivity \(k\) are given by

\[
\frac{dS_k(t)}{dt} = -bkS_k(t)\Theta_k(t) + a_k,
\]

\[
\frac{dI_k(t)}{dt} = -\delta_0 I_k(t) + bkS_k(t)\Theta_k(t),
\]

where \(b\) and \(\delta_0\) denote the infection and detection rates between 0 and 1, the shadow variable \(R_k(t)\) is implicitly defined by \(\frac{dR_k(t)}{dt} = \delta_0 I_k(t)\). The factor \(\Theta_k(t) \overset{\text{def}}{=} \sum_{l \neq k} \frac{n_l}{n_k} I_l(t)\) represents the expectation of infection by contacts from degree classes \(\{l\}\) to the degree class \(k\).

We assume the correlation between degrees \(n_{kl} \neq n_{lk}\), \(n_k \overset{\text{def}}{=} \sum_l n_{kl}\), and \(N_k(t) \sim n_{kl} \times t\) which is defined by the number of nodes with degree \(k\) attached to a node with degree \(l\) at the time \(t\). We can easily check \(\frac{dR_k(t)}{dt} = a_k\) and the solution \(N_k(t) = N_k(0) + a_k \times t \sim a_k \times t\) because of the initial small size (almost all \(N_k(0) = 0\) except for a few small \(k\)). The network is keeping the power-law degree distribution \(P(k) \sim k^{-\nu}\) in growing. Although the growing in our model is not exactly same in Krapivsky-Renier’s GN model \([11]\), the growth rate \(a_k\) is close to their \(n_k = \frac{k}{(k+1)(k+2)} \sim 4k^{-3}\) in large \(t\).

We consider a section of \(I_l = I_l^*\): const. for all \(l \neq k\). Fig. 2 (a) shows the nullclines of

\[
\frac{dS_k}{dt} = 0 : S_k = \frac{a_k}{kb\Theta_k} = \frac{a_k n_k}{kb(n_{kk} I_k + \sum_{l \neq k} n_{kl} I_l^*)},
\]

\[
\frac{dI_k}{dt} = 0 : S_k = \frac{\delta_0 I_k}{bk\Theta_k} = \frac{n_k \delta_0 I_k}{kb(n_{kk} I_k + \sum_{l \neq k} n_{kl} I_l^*)},
\]

and the vector filed for Eqs. (1)(2). There exist an equilibrium point: \(I_k^* = \frac{a_k}{\delta_0 + \delta_r} = \frac{A_k}{\delta_0 kr}, S_k^* = \frac{a_k}{bk\Theta_k} \sim \frac{A_k^2}{bk^{\nu-1} \sum_{l \neq k} n_{kl}^*}\). On the state space, the viruses are not extinct by only the detection in even \(I_l^* = 0\) \((l \neq k)\), unless the growing is stopped as \(a_k = 0\).

Effect of immunization.—Next, we study the effect of random and hub immunization. With the randomly immune rate \(0 < \delta_r < 1\), the time evolutions are given by

\[
\frac{dS_k}{dt} = -bkS_k(t)\Theta_k(t) + a_k - \delta_r S_k(t),
\]

\[
\frac{dI_k}{dt} = -\delta_0 I_k(t) + bkS_k(t)\Theta_k(t) - \delta_r I_k(t),
\]

where the shadow variable \(R_k(t)\) is also defined by \(\frac{dR_k(t)}{dt} = \delta_0 I_k(t) + \delta_r(S_k(t) + I_k(t))\).

On the section by assuming \(I_l^* > 0\) for \(l \neq k\), the nullclines are

\[
\frac{dS_k}{dt} = 0 : S_k = \frac{a_k}{\delta_r + bk\Theta_k},
\]

\[
\frac{dI_k}{dt} = 0 : S_k = \frac{(\delta_0 + \delta_r) I_k}{bk\Theta_k},
\]

for Eqs. (3)(4). At the intersection of nullclines, from

\[
I_k^* = \frac{a_k - \delta_r I_l^*}{\delta_0 + \delta_r} = \frac{a_k}{\delta_0 + \delta_r} \left(1 - \frac{\delta_r}{\delta_r + bk\Theta_k}\right),
\]

the self-consistent solution is given by

\[
\Theta_k^* = \sum_{l \neq k} \frac{n_k}{n_k} I_l^* = \frac{1}{\sum_{l \neq k} n_{kl}(1 - \frac{\delta_r}{\delta_r + bk\Theta_k})} + a_k \frac{1}{(1 - \frac{\delta_r}{\delta_r + bk\Theta_k})}.
\]

When the right hand side of (5) is denoted by \(f(\Theta_k)\), for \(\exists \Theta_k > 0, \frac{df(\Theta_k)}{d\Theta_k}\bigg|_{\Theta_k=0} > 1\) is necessary. The condition is

\[
\frac{df(\Theta_k)}{d\Theta_k}\bigg|_{\Theta_k=0} = k_{max} b \times \frac{n_{kk}}{n_{kk}\delta_r(\delta_r + \delta_0)} > 1.
\]
This means the growth and infection rates should be larger than the immune and detection rates according to the connectivity correlation of degree \( k \).

On the other hand, we assume \( I_t^* = 0 \) for \( l \neq k \) to discuss the extinction. The necessary condition of extinction is given by that the point \((0, \frac{dS}{dt})\) on the hyperbolic nullcline of \( \frac{dS}{dt} = 0 \) is below the line \( S_k = \hat{S}_k \triangleq \frac{(\delta_0 + \delta_r) n_k}{k b n_{kk}} \): const. of \( \frac{dI}{dt} = 0 \). From the condition

\[
\frac{a_k}{\delta_r} < \frac{(\delta_0 + \delta_r) n_k}{k b n_{kk}},
\]

we obtain

\[
\delta_r > -\delta_0 + \sqrt{\delta_0^2 + 4b a_k b \times n_{kk}/n_k}
\]

\[
\approx -\delta_0 + \sqrt{2 \delta_0^2 + 4kb \times n_{kk}},
\]

for \( n_k \approx a_k \) asymptotically. From \( \delta_r < 1 \), we further derive

\[
A < \min_{k \geq m} \left\{ \frac{1 + 2\delta_0}{b n_{kk}} \left[ \frac{1 + 2\tau}{b n_{kk}} \right]^{(k-1)(k+2)/(2k+1)} \right\},
\]

as a limitation of growth rate or a weak correlation

\[
n_{kk} < \frac{1 + 2\delta_0}{4kb},
\]

for the extinction. In this extinction case, there exists a stable equilibrium point, otherwise a saddle and a stable equilibrium point as shown in Figs. 2 (c)(d). Note that \( \delta_r > 0 \) is important for the extinction. When no growing \( a_k = 0 \) (as a closed system) is applied to the above results, the condition (6) for damping oscillation is not satisfied, while the extinction condition (8) is satisfied. In other words, it is suggested that a growing network causes the recoverable prevalence of infection.

By replacing \( a_k = A k^{-\nu} \) with the Krapivsky-Render’s \( n_k \) [11] and using their \( n_{kk} = \frac{k}{k+1} \frac{(k-1)(k+2)}{k(k+1)(k+2)} \), \( k \geq 2 \), we can check that \( n_{kk} < \frac{1 + 2\delta_0}{4kb} \) is satisfied for any \( 0 < b, \delta_0 < 1 \). The reason why viruses tend to be extinct in their GN model by even random immunization is that the infected parts are disconnected by recovered nodes on the tree structure, while our model generally has other bypass links than the links of tree in the difference of the degree distributions \( a_k t \) and their \( n_k t \) for small \( k \), therefore infection can be spreading.

Moreover, for the hub immunization [7], \( \delta_r \) is replaced by \( 0 < \delta_h k^\tau < k^\tau, \tau > 0 \). Then, the necessary condition of extinction in (8) is relaxed to larger

\[
A < \min_{k \geq m} \left\{ \frac{m^\tau + 2\delta_0}{b n_{kk}} \left[ \frac{m^\tau + 2\delta_0}{b n_{kk}} \right]^{(k-1)(k+2)/(2k+1)} \right\},
\]

\[
n_{kk} < \frac{m^\tau (m^\tau + 2\delta_0)}{4kb}.
\]

However this is under the assumption of \( I_t^* = 0 \). If the general case for a section \( I_t^* \neq 0 \) is similar to Fig. 2 (a), then viruses in the class of degree \( k \) are not extinct unless the growing is stopped as \( a_k = 0 \), otherwise they approaches to the extinction as shown from Fig. 2 (b).
to (d). In fact, at the point \((\ldots, I_k, S_k, \ldots, I_l, S_l, \ldots) = (\ldots, 0, \frac{S}{N}, \ldots, 0, \frac{S}{N}, \ldots)\), the eigenvalues of Jacobian

\[
\begin{pmatrix}
\cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\
\cdots & -\delta_r & -c_{kk} & 0 & -c_{kl} & \cdots \\
0 & d_{kk} & \cdots & 0 & c_{kl} & \cdots \\
\cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\
0 & -c_{lk} & \cdots & -\delta_r & -c_{ll} & \cdots \\
0 & c_{lk} & \cdots & 0 & d_{ll} & \cdots \\
\cdots & \cdots & \cdots & \cdots & \cdots & \cdots 
\end{pmatrix}
\]

are \(\lambda = -\delta_r < 0\) and \(\lambda \sim d_{kk} < 0\) from the condition (7), where \(d_{kk} \equiv -\left(\delta_0 + \delta_r\right) + \frac{\kappa_{kk} N_{kk}}{N}\) and \(c_{kl} \equiv \frac{\kappa_{kl} N_{kl}}{N}\). If \(c_{kl}\) and \(c_{lk}\) with respect to the cross correlations are negligibly weak. Another extinction route is given by \(b \to 0\), in which case the condition (8) is satisfied for any \(A\) and \(N_{kk}\). It is consistent with the result of no epidemic threshold [17].

These dynamics are qualitatively same to the previous results by a mean-field approximation without the connectivity correlations [9]. In summary, we have investigate mechanisms of recoverable prevalence and extinction of viruses in SIR models with the connectivity correlations on linearly growing SF networks, and derived the necessary condition of extinction related to the growth, infection, detection, and immune rates. The results suggest that we must stop the growing of network or close it by quarantine to prevent the spreading of infection. A more general case in linearly growing SF networks on no assumption of correlation \(N_{kl} \sim n_{kl} \times t\) is further study.

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