Epidemic spreading with immunization on bipartite networks

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Abstract

Bipartite networks are composed of two types of nodes and there are no links between nodes of the same type. Thus the study of epidemic spread and control on such networks is important for sexually transmitted diseases (STDs). A typical method to prevent or control an epidemic spread is immunization such as vaccination [2]. However, it is generally impossible to perfectly immunize entire populations of two types, so objects or target sets of immunization and immunization rates must be introduced. Immunization rates are parameters representing various effects of immunity.

We consider the SIR and SIS models with immunization targets and rates on bipartite networks. Based on [10, 11] two (partial) immunization policies are treated: "imperfect immunization to targeted nodes" and "insufficient immunity".

The degree of a node is the number of links emanating from it. Using the probability distributions of degrees of both types, we derive the critical infection rates or the thresholds, above which a disease spreads on bipartite networks. Based on [3, 4], sexual contact networks relevant to STDs follow power-law degree distributions; 

\[ p(k_M) \propto k_M^{-\gamma_M} \text{ and } p(k_F) \propto k_F^{-\gamma_F}, \]

regardless of reported countries (Sweden, U.K., Burkina Faso, etc.) and periods (12 months and the life span). Here indices M and F mean two populations of males and females, respectively, and the exponents \( \gamma_M \) and \( \gamma_F \) are positive constants and typically satisfy

\[ 2 < \gamma_M \leq 3 < \gamma_F. \]

This paper deals with the SIR and SIS models with immunization, but the models involving the target sets and immunization rates are restricted to the SIS models, because the derivation of the thresholds for the SIR models becomes a little lengthy. Finally, based on the thresholds, we apply immunization strategies to STDs on heterosexual contact networks as above. The immunization strategies should be implemented on nodes with high degrees in one population and on those with low degrees in the other.

1. Introduction

Bipartite networks or graphs are composed of two types of nodes (or vertices) and there are no links (or edges) between nodes of the same type. Links are allowed to connect nodes of different types. Thus the study of epidemic spread and control on such networks is important for sexually transmitted diseases (STDs). A typical method to prevent or control an epidemic spread is immunization such as vaccination [2]. However, it is generally impossible to perfectly immunize entire populations of two types, so objects or target sets of immunization and immunization rates must be introduced. Immunization rates are parameters representing various effects of immunity.

We consider the SIR and SIS models with immunization targets and rates on bipartite networks. Based on [10, 11] two (partial) immunization policies are treated: "imperfect immunization to targeted nodes" and "insufficient immunity".

The degree of a node is the number of links emanating from it. Using the probability distributions of degrees of both types, we derive the critical infection rates or the thresholds, above which a disease spreads on a bipartite network and below which it dies out.

Due to [3, 4], sexual contact networks relevant to STDs follow power-law degree distributions;

\[ p(k_M) \propto k_M^{-\gamma_M} \text{ and } p(k_F) \propto k_F^{-\gamma_F}, \]

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2. The SIR model without immunization

We distinguish two types of nodes of a bipartite network by indices 1 and 2. Hence all links connect nodes between types 1 and 2. In the SIR model, nodes of the network are divided into the following three groups regarding infection states of a disease ([2], [6, Chap. 10]): Susceptible (S), Infected (I) and Removed (R). A susceptible node (S-node) of type \( i \) becomes an infected node (I-node) at a rate \( \lambda_i \), with infection rates \( \lambda_i \). The parameters \( \lambda_i \) are the infection rates, for which we will derive the critical values for an epidemic outbreak. The disease can be passed from I-nodes to S-nodes of different types through links on bipartite networks. A recovered node (R-node) has either recovered from the disease or died and so they cannot pass the disease to others. An I-node becomes an R-node at a rate \( \delta_i \), with recovery rates \( \delta_i \). We can assume \( \delta_1 = 1 \), without loss of generality.

Let \( p(k_1) \) and \( p(k_2) \) be the probability distributions of nodes with degree \( k_1 \) in population 1 and of nodes with degree \( k_2 \) in population 2, respectively. The averages or moments are defined by

\[ \langle k_i \rangle = \sum_{k_i} k_i p(k_i), \quad \langle k_i^2 \rangle = \sum_{k_i} k_i^2 p(k_i), \]

for type \( i \).

Within population of each type \( i \), the densities of S-, I-, R-nodes with degree \( k_i \) at time \( t \) are denoted by variables \( S_{k_i}(t), I_{k_i}(t), R_{k_i}(t) \), respectively. Since there are no rewiring of links, we have

\[ S_{k_i}(t) + I_{k_i}(t) + R_{k_i}(t) = 1. \]

Following the dynamical mean-field approach ([5, 8, 9]), we see that the spreading process in population 1 on a bipartite network can be described by the system
of differential equations:
\[
\begin{align*}
\frac{dS_k}{dt} &= -\lambda_1 k_1 S_k(t) \theta_2(t), \quad (1) \\
\frac{d\rho_k}{dt} &= \lambda_1 k_1 S_k(t) \theta_2(t) - \delta_1 \rho_k(t), \quad (2) \\
\frac{dR_k}{dt} &= \delta_1 \rho_k(t). \quad (3)
\end{align*}
\]

The term \(\lambda_1 k_1 S_k(t) \theta_2(t)\) in (1) and (2) indicates the fraction of newly infected nodes through \(k_1\) links, while \(\theta_2(t)\) is the probability of contact with I-nodes of population 2 from which the disease spreads. Hence \(\theta_2(t)\) can be written as

\[
\theta_2(t) = \frac{\sum k_2 p(k_2) \rho_k(t)}{\sum k_2 p(k_2)} = \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) \rho_k(t).
\]

By the bipartite nature, analogous equations in population 2 to Eqs. (1)–(4) are valid under the interchange with 1 and 2. We indicate those equations by Eqs. (1’)–(4’), respectively. The initial conditions of \(S_k\) are set by

\[
S_k(0) = 1 \quad (i = 1, 2),
\]

meaning that almost all nodes are S-nodes at first.

To begin with we solve Eqs. (1) and (1’) under the initial conditions (5);

\[
S_k(t) = e^{-\lambda_1 k_1 \phi_2(t)}, \quad S_k(t) = e^{-\lambda_2 k_2 \phi_1(t)},
\]

where

\[
\phi_2(t) = \int_0^t \theta_2(\tau)d\tau, \quad \phi_1(t) = \int_0^t \theta_1(\tau)d\tau.
\]

Using (3’), (4) and \(R_k(0) = 0\) for all \(k_2\), the auxiliary function \(\phi_2(t)\) has an expression:

\[
\phi_2(t) = \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) \rho_k(\tau)d\tau = \frac{1}{\delta_2 \langle k_2 \rangle} \sum k_2 p(k_2) R_k(\tau).
\]

We derive the differential equation for \(\phi_2(t)\). Using (6), it follows that

\[
\frac{d\phi_2(t)}{dt} = \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) \rho_k(t)
\]

\[
= \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2)(1 - S_k(t) - R_k(t))
\]

\[
= 1 - \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) S_k(t) - \delta_2 \phi_2(t)
\]

\[
= 1 - \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) e^{-\lambda_2 k_2 \phi_1(t)} - \delta_2 \phi_2(t).
\]

We are concerned with a steady state of the epidemic outbreak, in which one has the limits

\[
\Phi_i = \lim_{t \to \infty} \phi_i(t) \quad (i = 1, 2),
\]

together with the conditions

\[
\lim_{t \to \infty} \frac{d\phi_i(t)}{dt} = 0 \quad (i = 1, 2).
\]

Using these relations in Eq. (7) and then interchanging 1 and 2, we get the coupled equations for \(\Phi_i\) as follows:

\[
\delta_2 \Phi_2 = 1 - \frac{1}{\langle k_2 \rangle} \sum k_2 p(k_2) e^{-\lambda_2 k_2 \Phi_1}, \quad (8)
\]

\[
\delta_1 \Phi_1 = 1 - \frac{1}{\langle k_1 \rangle} \sum k_1 p(k_1) e^{-\lambda_1 k_1 \Phi_2}. \quad (9)
\]

Note that \(\Phi_2 = 0\) if \(\Phi_1 = 0\) and vice versa, so the epidemic outbreak occurs when \(\Phi_1 > 0\). The right hand sides of Eqs. (8) and (9) are monotone increasing and concave functions of \(\Phi_1\) and \(\Phi_2\), respectively. Therefore, the function

\[
\frac{1}{\delta_1} \{1 - \frac{1}{\langle k_1 \rangle} \sum k_1 p(k_1) e^{-\lambda_1 k_1 \Phi_2}\}
\]

which is derived from (9), is also a monotone increasing and concave function of \(\Phi_1\) via Eq. (8), and its value at \(\Phi_1 = 1\) is less than 1 from the assumption \(\delta_1 = 1\). Thus the condition for the outbreak is given by

\[
\frac{d}{d\Phi_1} \frac{1}{\delta_1} \{1 - \frac{1}{\langle k_1 \rangle} \sum k_1 p(k_1) e^{-\lambda_1 k_1 \Phi_2}\}|_{\Phi_2=0} \geq 1.
\]

Remarkably the relation

\[
\frac{d\Phi_2(0)}{d\Phi_1} = \frac{\lambda_2 \langle k_2^2 \rangle}{\delta_2 \langle k_2 \rangle},
\]

it follows that the critical infection rates of \(\lambda_1\) and \(\lambda_2\), or thresholds, satisfy

\[
\lambda_1 \lambda_2 = \delta_2 \delta_2 \frac{\langle k_1 \rangle \langle k_2 \rangle}{\langle k_1^2 \rangle \langle k_2^2 \rangle}.
\]

This condition coincides with Eq. (5) in [3], which was obtained for the SIS model of STDs without immunization. Also in [7] a similar expression was derived using the generating function methodology.

Since the SIS model does not contain R-nodes, it is easier to deal with the epidemic spreading with immunization within the framework introduced in Section 1.

3. The SIS models with immunization
In this section we consider the spreading and control of a disease based on SIS models with target sets and immunization rates. In these models R-nodes are absent and nodes that are recovered from the disease instantly become S-nodes again.

A typical method to prevent or control an epidemic spreading is immunization such as vaccination [2]. Since it is very difficult or impossible to perfectly immunize an entire population, the object or target of immunization must be prescribed. Within population \(i\) \((i = 1, 2)\) we prescribe the object or target set, \(T_i\), for immunization.

We assume that the target sets are characterized in terms of degrees \(k_i\). The notation \(k_i \in T_i\) means that the population of all nodes with degree \(k_i\) is an object of immunization. In case of \(T_1 = \{k_1|k_1 \geq K_1\}\), for example, the population of all nodes with degrees exceeding a size \(K_1\) is collectively an object of immunization. The notation \(T_i\) indicates the complement of \(T_i\), meaning that \(T_i\) is not an object of immunization. The summation restricted to all \(k_i\) in \(T_i\) will be denoted by \(\sum_{T_i}\), and similarly for \(\bar{T}_i\) by \(\sum_{\bar{T}_i}\). Furthermore, the averages of \(k_i\) and \(k_i^2\) over \(T_i\) are denoted by \((k_i)_{T_i}\) and \((k_i^2)_{T_i}\):

\[
(k_i)_{T_i} = \sum_{k_i \in T_i} k_i p(k_i) = \sum_{T_i} k_i p(k_i),
\]

\[
(k_i^2)_{T_i} = \sum_{k_i \in T_i} k_i^2 p(k_i) = \sum_{T_i} k_i^2 p(k_i).
\]

Similarly the averages \((k_i)_{\bar{T}_i}\) and \((k_i^2)_{\bar{T}_i}\) over \(\bar{T}_i\) are also defined.

Two SIS models below involve immunization targets \(T_i\) and rates \(\alpha_i\) \((0 \leq \alpha_i \leq 1)\). The rates \(\alpha_i\) are assumed to be constants, although they can be dependent on \(k_i\). The condition \(\alpha_i = 1\) implies the perfect immunization for \(T_i\), while \(\alpha_i = 0\) means no immunization. The variables \(S_{k_i}(t), r_{k_i}(t)\) indicate the densities of S-, I-nodes with degree \(k_i\) at time \(t\), as before.

**A. Imperfect immunization to targeted nodes**

This is the case where all of targeted nodes in \(T_i\) may not be immunized, because some are overlooked or hidden. The immunization rates \(\alpha_i\) \((0 \leq \alpha_i \leq 1)\) represent the effectiveness of immunization such as vaccination coverage.

Since immunized nodes are no longer S-nodes, we have

\[
S_{k_i}(t) = \begin{cases} 
1 - \alpha_i - r_{k_i}(t), & \text{for } k_i \in T_i, \\
1 - r_{k_i}(t), & \text{for } k_i \in T_i.
\end{cases}
\]

Therefore, Eqs. (1)–(3) in Section 2 are replaced by the differential equations for population 1

\[
\frac{d\rho_{k_i}(t)}{dt} = \begin{cases} 
\lambda_1 k_1 (1 - \alpha_1 - \rho_{k_i}(t)) \theta_2(t) - \delta_1 \rho_{k_i}(t), & \text{if } k_i \in T_1, \\
\lambda_1 k_1 (1 - \rho_{k_i}(t)) \theta_2(t) - \delta_1 \rho_{k_i}(t), & \text{if } k_i \in \bar{T}_1,
\end{cases}
\]

where \(\theta_2(t)\) is the same probability as (4). Similar equations for \(\rho_{k_2}(t)\) follow from Eqs. (1')–(3') and \(\theta_1(t)\) of (4').

At the steady state, as in Section 2, we will have the conditions

\[
\lim_{t \to \infty} \frac{d\rho_{k_i}(t)}{dt} = 0, \quad \lim_{t \to \infty} \frac{d\rho_{k_2}(t)}{dt} = 0
\]

for all \(k_1\) and \(k_2\), and the limits

\[
\Theta_1 = \lim_{t \to \infty} \theta_1(t), \quad \Theta_2 = \lim_{t \to \infty} \theta_2(t).
\]

So we get from (11),

\[
\lim_{t \to \infty} \rho_{k_i}(t) = \left\{ \begin{array}{ll}
(1 - \alpha_1)\lambda_1 k_1 \Theta_2 / (\delta_1 + \alpha_1 k_1 \Theta_2), & \text{if } k_i \in T_1, \\
\lambda_1 k_1 \Theta_2 / (\delta_1 + \alpha_1 k_1 \Theta_2), & \text{if } k_i \in \bar{T}_1.
\end{array} \right.
\]

By interchanging 1 and 2, we also have

\[
\Theta_1 = \frac{1}{(k_1)} \left( \sum_{T_1} (1 - \alpha_1) \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2) \right) + \sum_{\bar{T}_1} \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2).
\]

(12)

(13)

Substituting these into (4') in the limit, we have the equation for \(\Theta_1\) as follows:

\[
\Theta_1 = \frac{1}{(k_1)} \left( \sum_{T_1} (1 - \alpha_1) \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2) \right) + \sum_{\bar{T}_1} \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2).
\]

(12)

(13)

If the coupled Eqs. (12), (13) have solutions \(\Theta_1 > 0\) and \(\Theta_2 > 0\), then an endemic outbreak occurs. The right hand sides of Eqs. (12) and (13) are monotone increasing and concave functions of \(\Theta_2\) and \(\Theta_1\), respectively. Hence the right hand side of (12) becomes a monotone increasing and concave function of \(\Theta_1\) via Eq. (13), and furthermore its value at \(\Theta_1 = 1\) is less than 1. Therefore, we see that the thresholds of \(\lambda_1\) and \(\lambda_2\) satisfy

\[
\frac{d}{d\Theta_1} \frac{1}{(k_1)} \left( \sum_{T_1} (1 - \alpha_1) \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2) + \sum_{\bar{T}_1} \lambda_1 k_1^2 p(k_1) \Theta_2 / (\delta_1 + \lambda_1 k_1 \Theta_2) \right) \big|_{\Theta_1 = 0} = 1.
\]

Noting

\[
\frac{d\Theta_2(0)}{d\Theta_1} = \frac{\lambda_2}{\delta_2 (k_2)} ((1 - \alpha_2) (k_2^2)_{T_2} + (k_2^2)_{\bar{T}_2}),
\]

3
and using the identities
\[ \langle k_i^1 \rangle_{T_1} = \langle k_i^2 \rangle - \langle k_i^1 \rangle_{T_1}, \quad \langle k_i^2 \rangle_{T_2} = \langle k_i^2 \rangle - \langle k_i^2 \rangle_{T_2}, \]
we see that at the thresholds of \( \lambda_1 \) and \( \lambda_2 \), the relation
\[ \lambda_1 \lambda_2 = \frac{\delta_1 \delta_2 (\langle k_i^1 \rangle_{T_1})}{(\langle k_i^2 \rangle - \alpha_1 (\langle k_i^1 \rangle_{T_1}) (\langle k_i^2 \rangle - \alpha_2 (\langle k_i^2 \rangle_{T_2})))} \tag{14} \]
holds. In the case of no immunization (\( \alpha_1 = \alpha_2 = 0 \)) it coincides with (10).

**B. Insufficient immunity**

The parameters \( \alpha_i \) of this model represent the levels of immunity. The higher they are, the less infected S-nodes are. Thus we have
\[ S_{k_i}(t) = \begin{cases} (1 - \alpha_i)(1 - \rho_{k_i}(t)), & \text{for } k_i \in T_i, \\ 1 - \rho_{k_i}(t), & \text{for } k_i \in T_i. \end{cases} \]
So instead of (11) we obtain
\[ \frac{d\rho_{k_i}(t)}{dt} = \begin{cases} \lambda_1 k_i (1 - \alpha_i)(1 - \rho_{k_i}(t))\theta_2(t) - \delta_1 \rho_{k_i}(t), & \text{if } k_i \in T_i, \\ \lambda_1 k_i (1 - \rho_{k_i}(t))\theta_2(t) - \delta_1 \rho_{k_i}(t), & \text{if } k_i \in T_i. \end{cases} \]

Similar equations for \( \rho_{k_i}(t) \) also hold by interchanging 1 and 2. This model is the same as the one treated in [1] for unipartite networks. Repeating the above calculations in A, it follows that the condition for thresholds takes the same form as (14).

As for the condition of the thresholds for SIR models with immunization policies A and B, we are able to derive it by dividing each population \( i \) into two parts \( T_i \) and \( T_i^c \) as in [10], and using the procedures in Section 2. Then we see that the same condition (14) is also obtained for on bipartite networks. Thus we conclude that the SIR and SIS models with immunization rates all have the same condition of thresholds as expressed in (14).

Furthermore, populations 1 and 2 may adopt different immunization policies. Even if population 1 adopts the policy A, while population 2 adopts the policy B, we get the same condition of thresholds again.

**4. Immunization strategies**

Suppose that the degree distributions of a bipartite network follow power-laws;
\[ p(k_1) \propto k_1^{-\gamma_1} \quad \text{and} \quad p(k_2) \propto k_2^{-\gamma_2} \]
for types 1 and 2, respectively. Moreover, the two exponents satisfy
\[ 2 < \gamma_1 \leq 3 < \gamma_2, \]
as in most sexual contact networks [3]. Other cases such as
\[ 2 < \gamma_1, \gamma_2 < 3, \quad 3 < \gamma_1, \gamma_2, \]
can be discussed in similar ways.

By (14) the geometric mean of the two critical thresholds
\[ \lambda_c = \sqrt{\frac{\delta_1 \delta_2 (\langle k_i^1 \rangle_{T_1})}{(\langle k_i^2 \rangle - \alpha_1 (\langle k_i^1 \rangle_{T_1}) (\langle k_i^2 \rangle - \alpha_2 (\langle k_i^2 \rangle_{T_2})))}} \]
provides an overall threshold of the epidemic spreading. In order to control the spreading of STDs, we try to raise the value of \( \lambda_c \) by employing two immunization strategies. It is desirable to implement both strategies simultaneously.

Under the condition \( 2 < \gamma_1 \leq 3 \) the mean square \( \langle k_i^1 \rangle \) tends to infinity in the limit of infinite population, although real networks have finite sizes. First of all we must set \( \alpha_1 = 1 \) for population 1. For, if \( \alpha_1 < 1 \), then
\[ \langle k_i^1 \rangle - \alpha_1 (\langle k_i^1 \rangle)_{T_1} \geq (1 - \alpha_1) (\langle k_i^1 \rangle) \rightarrow \infty \]
in the limit of infinite population, no matter how \( T_1 \) is chosen. So by setting \( \alpha_1 = 1 \), we have
\[ \langle k_i^1 \rangle - \alpha_1 (\langle k_i^1 \rangle)_{T_1} = (\langle k_i^1 \rangle)_{T_1} < K_1^2 \]
for a target set \( T_1 = \{ k_1 | k_1 \geq K_1 \} \). Therefore, an immunization strategy to population 1 is the perfect immunization to nodes with an upper half of degrees as in [9].

On the contrary, \( \langle k_i^2 \rangle \) is finite by \( \gamma_2 > 3 \). This implies that nodes with high degrees make little contribution to \( \langle k_i^2 \rangle \) and hence they are negligible. Thus one should take \( T_2 = \{ k_2 | k_2 \leq K_2 \} \), for some positive \( K_2 \), as a target set of population 2. Then we have
\[ (\langle k_i^2 \rangle)_{T_2} - \alpha_2 (\langle k_i^2 \rangle)_{T_2} = (\langle k_i^2 \rangle)_{T_2} + (1 - \alpha_2) (\langle k_i^2 \rangle)_{T_2} \rightarrow 0, \]
when \( \alpha_2 \rightarrow 1 \) and \( K_2 \) becomes larger. Hence the immunization strategy to population 2 is concentrated on nodes with a lower half of degrees. Since \( (\langle k_i^2 \rangle)_{T_2} \) is also finite, \( \alpha_2 \) need not be one, and as for \( K_2 \) generally a small size is enough.

**References**

[1] X. Fu, M. Small, D. Walker and H. Zhang, Epidemic dynamics on scale-free networks with piecewise linear infectivity and immunization, Physical Review E 77, 036113, 2008.

[2] J. Giesecke, Modern Infectious Disease Epidemiology, E. Arnold Pub., London, 2002.

[3] J. Gómez-Gardeñes, V. Latora, Y. Moreno and E. Profumo, Spreading of sexually transmitted diseases in heterosexual populations, Proceedings of the National Academy of Sciences 105, 1399–1404, 2008.
[4] F. Liljeros, C. Edling, L. Amaral, H. Stanley and Y. Aberg, The web of human sexual contacts, Nature 411, 907-908, 2001.

[5] Y. Moreno, R. Pastor-Satorras and A. Vespignani, Epidemic outbreaks in complex heterogeneous networks, European Physical Journal B 26, 521-529, 2002.

[6] J. D. Murray, Mathematical Biology, Springer Verlag, New York, 2002.

[7] M. E. J. Newman, Spread of epidemic disease on networks, Physical Review E 66 016128, 2002.

[8] R. Pastor-Satorras and A. Vespignani, Epidemic spreading in scale-free networks, Physical Review Letters 86, 3200–3203, 2001.

[9] R. Pastor-Satorras and A. Vespignani, Immunization of complex networks, Physical Review E 65, 036104, 2002.

[10] S. Tanimoto, Epidemic spreading with immunization rate on complex networks, arXiv:1104.2364, 2011.

[11] Y. Wang, G. Xiao, J. Hua, T. H. Cheng and L. Wang, Imperfect targeted immunization in scale-free networks, Physica A 388, 2535–2546, 2009.