Heterogeneous Protection in Regular and Complete Bi-partite Networks

J. Omic, R. E. Kooij, and P. Van Mieghem

Delft University of Technology,
Faculty of Electrical Engineering, Mathematics and Computer Science,
P.O Box 5031, 2600 GA Delft, The Netherlands.
Email: {P.VanMieghem, J.S.Omic,R.E.Kooij}@ewi.tudelft.nl.
Dr. ir. Kooij is also with TNO Information Communication Technology,
P.O Box 5050, 2600 GB Delft.

Abstract. We examine the influence of heterogeneous curing rates for a SIS model, used for malware spreading on the Internet, information dissemination in unreliable networks, and propagation of failures in networks. The topology structures considered are the regular graph which represents the homogenous network structures and the complete bi-partite graph which represents the hierarchical network structures. We find the threshold in a regular graph with m different curing rates.
Further, we consider a complete bi-partite graph with 2 curing rates and find the threshold for any distribution of curing rates among nodes. In addition, we consider the optimization problem and show that the minimum sum of the curing rates that satisfies the threshold equation is equal to the number of links in the graph. The optimization problem is simplified by assuming fixed curing rates δ_1, δ_2 and optimization of the distribution of curing rates among different sets of nodes.

Key words: Virus spread, epidemic threshold, heterogeneous networks

1 Introduction

The Susceptible-Infected-Susceptible (SIS) infection model, which arose in mathematical biology, is often used to model the spread of computer viruses [7], [5], [14], epidemic algorithms for information dissemination in unreliable distributed systems like P2P and ad-hoc networks [9], [10], and propagation of faults and failures in networks like BGP [8].

The SIS model assumes that a node in the network is in one of two states: *infected* and therefore infectious, or *healthy* and therefore susceptible to infection. The SIS model usually assumes instantaneous state transitions. Thus, as soon as a node becomes infected, it becomes infectious and likewise, as soon as a node is cured it is susceptible to re-infection. There are many models that consider more aspects like incubation periods, variable infection rate, a curing process that takes a certain amount of time and so on [4], [7], [13]. In epidemiological theory, many authors refer to an epidemic threshold τ_c, see for instance [4], [1],

[7] and [14]. If it is assumed that the infection rate along each link is β while the curing rate for each node is δ then the effective spreading rate of the virus can be defined as $\tau = \beta/\delta$. The epidemic threshold can be defined as follows: for effective spreading rates below τ_c the virus contamination in the network dies out - the mean epidemic lifetime is of order $\log n$, while for effective spreading rates above τ_c the virus is prevalent, i.e. a persisting fraction of nodes remains infected with the mean epidemic lifetime [5] of the order e^{n^α}. In the case of persistence we will refer to the prevailing state as a metastable state or steady state. It was shown in [12] and [5] that $\tau_c = 1/\rho(A)$ where $\rho(A)$ denotes the spectral radius of the adjacency matrix A of the graph. Recently, the epidemic threshold formula has also been verified by using the N-intertwined model [16], which consists of a pair of interacting continuous Markov chains.

It is the aim of this paper to derive results for the epidemic threshold in the case of heterogeneous curing rates for regular and complete bi-partite graphs. A regular graph is an approximation of the random graph for large N and it represents a significant set of networks used in telecommunications. Further, the complete bi-partite graph represents a hierarchal type of topology, also frequently used in telecommunications. Notice that (core) telecommunication networks often can be modeled as a complete bi-partite topology. For instance, the so-called double-star topology (i.e. $K_{M;N}$ with $M = 2$) is quite commonly used because it offers a high level of robustness against link failures. For example, the Amsterdam Internet Exchange,[1] one of the largest public Internet exchanges in the world, uses this topology to connect its four locations in Amsterdam to two high-throughput Ethernet switches. Sensor networks are also often designed as complete bi-partite graphs.

The rest of the paper is organized as follows. In Section 2, we present the classical model by Kephart and White which describes the homogenous spread of a virus on regular graphs and the SIS model for the complete bi-partite graph also analyzed in [19]. In Section 2.1, we derive and analyze the spread of viruses in regular graphs in case of m curing rates. In Section 2.2, we discuss a specific case of regular graphs with 2 curing rates. In Section 2.3, we consider the spread of viruses on the complete bi-partite graphs with two curing rates. In the following section, we give solution for the optimization problem on complete bi-partite graph in the heterogenous case. We summarize our results in Section 4.

2 Virus spread on regular and bi-partite graphs

In order to explain our model of spread for computer viruses with heterogeneous curing rates, it is useful to first discuss the spread of viruses with homogeneous curing rate.

The homogenous model for regular graph is based on a classical result by Kephart and White [7] for SIS models. We consider a connected graph with

[1] see www.ams-ix.net

N nodes, where every node has degree k. We denote the number of infected nodes in the population at time t by $X(t)$. The probability that a randomly chosen node is infected is $v(t) \equiv X(t)/N$. Now, the rate at which the infection probability changes is due to two processes: susceptible nodes becoming infected and infected nodes being cured. The change in probability $v(t)$ due to the curing of infected nodes is $\delta v(t)$. The rate at which the infection probability $v(t)$ grows is proportional to the probability of a node being susceptible, i.e. $1 - v(t)$. For every susceptible node, the rate of infection is the product of the infection rate per node (β), the degree of the node (k) and the probability that on a given link the susceptible node connects to an infected node ($v(t)$). Therefore, we obtain the following differential equation describing the time evolution of $v(t)$:

$$\frac{dv(t)}{dt} = \beta k v(t)(1 - v(t)) - \delta v(t). \tag{1}$$

The solution to Eq. (1) is

$$v(t) = \frac{v_0(1-\rho)}{v_0 + (1 - \rho - v_0)e^{-(\beta k - \delta)t}}, \tag{2}$$

where v_0 is the initial probability of infected nodes. The steady state solution is

$$v_\infty = \frac{\beta k - \delta}{\beta k} \tag{3}$$

An epidemic steady state only exist for $v_\infty > 0$, therefore, the epidemic threshold equals to $\tau_c = \frac{1}{k}$. For k-regular graphs, the spectral radius of the adjacency matrix [2] is equal to k, therefore $\tau_c = \frac{1}{k}$ is in line with the result in [12].

Further, we will consider the complete bi-partite graphs with one curing rate δ. The SIS model for the complete bi-partite graph is presented in [19]. A complete bi-partite graph $K_{M,N}$ consists of two disjoint sets S_1 and S_2 containing respectively M and N nodes, such that all nodes in S_1 are connected to all nodes in S_2, while within each set no connections occur. Figure 1 gives an example of a complete bi-partite graph with 6 nodes. Since there are two sets of nodes

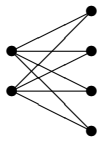

Fig. 1. Complete bi-partite graph $K_{2,4}$

with different degrees, equation (1) does not hold. A node from the set S_1 is connected to N nodes from the set S_2. The probability that a randomly chosen

node is infected in set S_1 is $v_1(t) \equiv X_1(t)/M$. The rate at which probability $v_1(t)$ grows is proportional to the probability that a node in the set S_1 is susceptible multiplied by the degree of the node N and the probability that a node connects to an infected node from set S_2, which is $v_2(t) \equiv X_2(t)/N$. For the set S_1 and S_2, we can write differential equations

$$\frac{dv_1(t)}{dt} = \beta N v_2(t)(1 - v_1(t)) - \delta v_1(t),$$
$$\frac{dv_2(t)}{dt} = \beta M v_1(t)(1 - v_2(t)) - \delta v_2(t)$$

For $\frac{dv_1(t)}{dt} = 0$ and $\frac{dv_2(t)}{dt} = 0$, we have the steady state solution

$$v_{1\infty} = \frac{\beta^2 MN - \delta^2}{N\beta(\delta + \beta M)},\ v_{2\infty} = \frac{\beta^2 MN - \delta^2}{M\beta(\delta + \beta N)}$$

Now, the epidemic threshold equals $\tau_c = \frac{\beta}{\delta}\Big|_{v_\infty = 0} = \frac{1}{\sqrt{MN}}$, which is the reciprocal of the spectral radius of the adjacency matrix for the complete bi-partite graph [2].

2.1 Virus spread on regular graphs with m curing rates

In this section, we derive the threshold for the spread of viruses on regular graphs with m curing rates.

Assume that $n_1, n_2, .., n_m$ denotes the fraction of nodes with curing rate $\delta_1, \delta_2, .., \delta_m$ ($\sum_{i=1}^{m} n_i = 1$). It is important to note that one of the assumptions is complete symmetry of the problem. For every node i, a fraction n_1 of neighbors has the curing rate δ_1, a fraction n_2 has curing rate δ_2 and so on.

Denote the number of infected nodes of type i in the population at time t by $X_i(t)$. The probability that a randomly chosen node of type i is infected is $v_i(t) \equiv \frac{X_i(t)}{Nn_i}$. Now, the rate at which the probability of infection for nodes of type i changes is due to two processes: susceptible nodes becoming infected and infected nodes being cured. The curing rate for an infection probability v_i is $\delta_i v_i$. The rate at which the probability v_i grows is proportional to the probability of a node of type i being susceptible, i.e. $1 - v_i$. For every susceptible node the rate of infection is the product of the infection rate per node (β) and the probability that on a given link the susceptible node connects to an infected node ($\sum_{j=1}^{m}(n_j k)v_j$).

Therefore, we obtain the following differential equation describing the time evolution of $v_i(t)$:

$$\frac{dv_i}{dt} = \beta k(\sum_{j=1}^{m} n_j v_j)(1 - v_i) - \delta_i v_i,\ i = 1, .., m \tag{4}$$

Note that for $\delta_1 = \delta_2 = .. = \delta_m$, the system of equations (4) reduces to Eq. (1) with $v = \sum_{j=1}^{m} n_j v_j$.

For the general case with different curing rates, it is impossible to obtain an explicit solution for the system of equations (4). The standard approach for this type of system of nonlinear differential equations, is to study the qualitative behavior in the phase space.

Theorem 1. *Consider connected regular graphs where each node has exactly k neighbors. Assume that the infection rate along each link is β while the curing rate for each node is δ_i for a fraction n_i of the nodes, with $i = 1, .., m \leqslant k$ and $\sum_{i=1}^{m} n_i = 1$. Complete symmetry is assumed, where each node sees the same fraction of different curing rates. If we define the effective spreading rate as $\tau = \frac{\beta}{\delta^*}$, where δ^* is defined as the weighted harmonic mean of $\delta_1, ..., \delta_m$, i.e. $\delta^* = \frac{1}{\sum_{i=1}^{m} \frac{n_i}{\delta_i}}$, then the epidemic threshold satisfies $\tau_c = \frac{1}{k}$.*

Proof. We denote the fraction of infected nodes of type i ($1 \leqslant i \leqslant m$) at time t as $v_i(t)$. This leads to a system of m differential equations (4).

We will use a Lyapunov function [3] to show that, under the condition $\beta \sum_{t=1}^{m} \frac{n_t}{\delta_t} - \frac{1}{k} \leq 0$, the origin is a global attractor for $\{v_1 \geq 0,\ v_2 \geq 0, .., v_m \geq 0\}$, hence, that the virus dies out. Let $V(v_1, v_2, .., v_m) = \prod_{j=1}^{m} \delta_j \sum_{s=1}^{m} \frac{v_s}{\delta_s}$. Then, we have

$$\begin{aligned} \frac{dV}{dt} &= -\left(\sum_{s=1}^{m} v_s\right)\left(\beta k V - \beta k \prod_{j=1}^{m} \delta_j \sum_{t=1}^{m} \frac{n_t}{\delta_t} + \prod_{j=1}^{m} \delta_j\right) \\ &= -\left(\sum_{s=1}^{m} v_s\right)\left(\beta k V - k \prod_{j=1}^{m} \delta_j \left(\beta \sum_{t=1}^{m} \frac{n_t}{\delta_t} - \frac{1}{k}\right)\right). \end{aligned}$$

The claim follows directly by applying Lyapunov's stability theorem. Next we consider the case $\beta \sum_{t=1}^{m} \frac{n_t}{\delta_t} - \frac{1}{k} > 0$. We first note that any trajectory of the system (4) can never leave the box $B = \{(v_1, ..., v_m) | 0 \leq v_1 \leq 1, ..., 0 \leq v_m \leq 1\}$. This follows from $\frac{dv_1}{dt}|_{v_1=0} = \beta k(\sum_{j=1}^{m} n_j v_j) \geq 0$, and similar inequalities at the borders of the box B.

From the construction of the above Lyapunov function V, we can see that for $\beta \sum_{t=1}^{m} \frac{n_t}{\delta_t} - \frac{1}{k} > 0$, and for $(v_1, ..., v_m) \in B$ and sufficiently close to the origin, $\frac{dV}{dt} > 0$. This implies that the origin has an unstable manifold in B. Therefore, since any trajectory of system (4) can never leave the box B, system (4) has an attractor as the ω-limit set and, hence, the virus does survive. This finishes the proof of the theorem. □

2.2 Virus spread on regular graphs with two curing rates

The two dimensional case ($m = 2$) of virus spread on a regular graph can be analyzed in more details. Applying Theorem 1, the spreading process has a threshold at $\tau = \frac{\beta}{\delta^*} = \frac{1}{k}$, where $\delta^* = \frac{\delta_1\delta_2}{n_1\delta_2+n_2\delta_1}$.

The phase portrait of two examples are depicted in Figure 2. The attractor for the case where virus survives is given by $(v_1, v_2) = (0.22, 0.17)$.

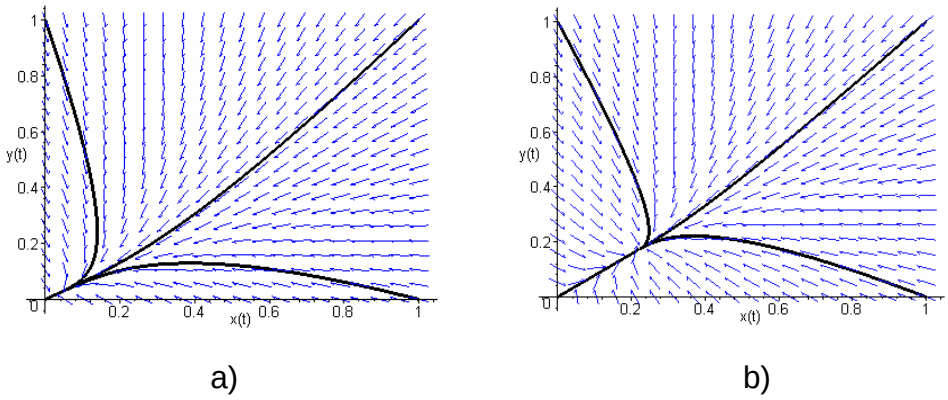

Fig. 2. Phase portrait for a regular graph with the two curing rates where a) virus dies out $\beta = 0.2$, $\delta_1 = 0.8$, $\delta_2 = 1.2$, $k = 4$, $n_1 = n_2 = 0.5$. b) virus survives $\beta = 0.4$, $\delta_1 = 0.8$, $\delta_2 = 1.2$, $k = 4$, $n_1 = n_2 = 0.5$.

For system (4) where $m = 2$, it can be proven that the attractor is an equilibrium point of a nodal type, situated on a straight line L. It can also be shown that the system does not contain other equilibrium points in A or closed orbits. Therefore, in the case $m = 2$, this equilibrium point is a global attractor of system (4) in A.

Lemma 1. *The set of differential equations given by (4) for $m = 2$, has a straight line solution of the form $v_2 = \lambda v_1$.*

Proof. We have that

$$\left(\frac{dv_2}{dt} = \lambda\frac{dv_1}{dt}\right)_{v_2=\lambda v_1} = -v_1(\beta k n_1\lambda^2+(\beta k(n_1-n_2)-\delta_1+\delta_2)\lambda-\beta k n_2) \equiv -v_1 f(\lambda)$$

$f(\lambda)$ has got exactly one negative root and one positive root. The positive root λ_1 satisfies

$$\lambda_1 = \frac{\beta k(n_2 - n_1) + \delta_1 - \delta_2 + \sqrt{\Delta}}{2\beta k n_1},$$

where $\Delta = \beta^2 k^2 + 2\beta k(n_1 - n_2)(\delta_2 - \delta_1) + (\delta_1 - \delta_2)^2$. Therefore the straight line $L : v_2 = \lambda_1 v_1$ is a solution of system (4) for $m = 2$, which for $0 \leq v_1 \leq 1$ is situated in A.

By application of the Poincaré-Bendixson theorem [3] on A, the ω-limit set for the system (4) for $m = 2$, can be either an equilibrium point or an isolated periodic orbit. From the fact that there is a line solution through the equilibrium point, it follows that the ω-limit set is the equilibrium point. □

2.3 Virus spread on complete bi-partite graphs with two curing rates

We will now derive a model for virus spread on the complete bi-partite graph $K_{M,N}$ with two different spreading rates. The result is general and it can be reduced to the case with all nodes in one set (S_1) having one curing rate δ_1 and in the other (S_2) δ_2.

Let us assume that a fraction p, with $p \in [0, 1]$, of nodes belonging to S_1 and a fraction q, with $q \in [0, 1]$, of nodes belonging to set S_2 have a curing rate δ_1, the rest have a curing rate δ_2. The total fraction of nodes with the curing rate δ_1 is $s = \frac{Mp+Nq}{M+N}$.

Denote the number of infected nodes of type 1 in the population of nodes from set S_1 at time t by $X_{i1}(t)$. The probability that a randomly chosen node of type 1 from set S_1 is infected is $v_{i1}(t) \equiv \frac{Xi_1(t)}{Mp}$. Similarly, let v_{i2} denote the infection probability for nodes of type 2 from set S_1, (v_{j1} denotes type 1, set S_2; and v_{j2} denotes type 2, set S_2). Now, the rate at which the probability of infection for nodes of type 1, set S_1 changes is due to two processes: susceptible nodes becoming infected and infected nodes being cured. The curing rate for an infection probability v_{i1} for nodes of type 1, set S_1 is $\delta_1 v_{i1}$. The rate at which the probability v_{i1} grows is proportional to the probability of a node of type 1, set S_1 being susceptible, i.e. $1 - v_{i1}$. For every susceptible node the rate of infection is the product of the infection rate per node (β), the degree of the node (N) and the probability that on a given link the susceptible node connects to an infected node ($qv_{j1} + (1 - q)v_{j2}$).

Similarly, we obtain the differential equations for the other probabilities (v_{i2}, v_{j1}, v_{j2}):

$$\begin{cases} \frac{dv_{i1}}{dt} = \beta N(qv_{j1} + (1-q)v_{j2})(1 - v_{i1}) - \delta_1 v_{i1}, \\ \frac{dv_{i2}}{dt} = \beta N(qv_{j1} + (1-q)v_{j2})(1 - v_{i2}) - \delta_2 v_{i2}, \\ \frac{dv_{j1}}{dt} = \beta M(pv_{i1} + (1-p)v_{i2})(1 - v_{j1}) - \delta_1 v_{j1}, \\ \frac{dv_{j2}}{dt} = \beta N(pv_{i1} + (1-p)v_{i2})(1 - v_{j2}) - \delta_2 v_{j2}, \end{cases} \tag{5}$$

The same set of equations can be obtained by the N-intertwined model [16].

In order to simplify the system of equations, we will substitute

$$i_1 = pv_{i1},\ i_2 = (1-p)v_{i2},\ j_1 = qv_{j1},\ j_2 = (1-q)v_{j2}$$

and

$$i = i_1 + i_2,\ j = j_1 + j_2$$

Therefore, we obtain the following differential equations for $i_1(t)$, $i_2(t)$, $j_1(t)$, $j_2(t)$:

$$\begin{cases} \frac{di_1}{dt} = p\beta Nj - \beta Nji_1 - \delta_1 i_1, \\ \frac{di_2}{dt} = (1-p)\beta Nj - \beta Nji_2 - \delta_2 i_2, \\ \frac{dj_1}{dt} = q\beta Mi - \beta Mij_1 - \delta_1 j_1, \\ \frac{dj_2}{dt} = (1-q)\beta Ni - \beta Nij_2 - \delta_2 j_2, \end{cases} \tag{6}$$

By solving the system of equations 6 for the steady state ($\frac{di_1}{dt} = \frac{di_2}{dt} = \frac{dj_1}{dt} = \frac{dj_2}{dt} = 0$) we can calculate the threshold:

$$\frac{\beta}{\delta^*} = \tau_c = \frac{1}{\sqrt{MN}} \tag{7}$$

$$\delta^* = \frac{\delta_1\delta_2}{\sqrt{\delta_1^2(1-p)(1-q) + \delta_2^2 pq + \delta_1\delta_2(p(1-q)+q(1-p))}}$$

Theorem 2. *Consider complete bi-partite graphs $K_{M,N}$ consisting of two disjoint sets S_1 and S_2 containing respectively M and N nodes. Assume that the infection rate along each link is β. For the nodes in S_1 a fraction p has curing rate δ_1 and in S_2 a fraction q of the nodes has curing rate δ_1, while the curing rate for a fraction $(1-p)((1-q))$ of the nodes is δ_2. If we define the effective spreading rate as $\tau = \frac{\beta}{\delta^*}$, where δ^* is defined as $\delta^* = \frac{\delta_1\delta_2}{\sqrt{(1-p)(1-q)\delta_1^2 + pq\delta_2^2 + \delta_1\delta_2(p(1-q)+q(1-p))}}$, then the epidemic threshold satisfies $\tau_c = \frac{1}{\sqrt{MN}}$.*

Proof. First, we will show that if $\frac{\beta}{\delta^*} \leqslant \frac{1}{\sqrt{MN}}$, the virus dies out. $(0,0,0,0)$ is an equilibrium point for system (5). We will use a Lyapunov function to show that, under the condition $\frac{\beta}{\delta^*} \leqslant \frac{1}{\sqrt{MN}}$, the origin is a global attractor for $i_1 \geq 0, i_2 \geq 0, j_1 \geq 0, j_2 \geq 0$.

Let $V(i_1, i_2, j_1, j_2) = \delta_1\delta_2^2 i_1 + \delta_1^2\delta_2 i_2 + \beta N(p\delta_2 + (1-p)\delta_1)(\delta_2 j_1 + \delta_1 j_2)$. Then,

$$\begin{aligned} \frac{dV}{dt} = &(\beta^2 MN((1-p)(1-q)\delta_1^2 + pq\delta_2^2 + \\ &+ \delta_1\delta_2((1-p)q + (1-q)p)) - \delta_1^2\delta_2^2)(i_1 + i_2) \\ &- \beta N\delta_2(\beta M(p\delta_2 + (1-p)\delta_1) + \delta_1\delta_2)i_1 j_1 \\ &- \beta N\delta_1(\beta M(p\delta_2 + (1-p)\delta_1) + \delta_2^2)i_1 j_2 \\ &- \beta N\delta_2(\beta M(p\delta_2 + (1-p)\delta_1) + \delta_1^2)i_2 j_1 \\ &- \beta N\delta_1(\beta M(p\delta_2 + (1-p)\delta_1) + \delta_1\delta_2)i_2 j_2. \end{aligned}$$

The extinction of the virus follows directly by applying Lyapunov's stability theorem. Next we will show that if $\frac{\beta}{\delta^*} > \frac{1}{\sqrt{MN}}$, the virus survives. We first note that

any trajectory of the system (5) can never leave the box $B = \{(i_1, i_2, j_1, j_2) | 0 \leq i_1 \leq 1,\ 0 \leq i_2 \leq 1, 0 \leq j_1 \leq 1, 0 \leq j_2 \leq 1\}$. This follows from $\frac{di_1}{dt}|_{i_1=0} = p\beta N(j_1 + j_2) \geq 0$, and similar inequalities at the borders of the box B.

From the construction of the Lyapunov function, we can see that for $\beta^2 MN((1-p)(1-q)\delta_1^2 + pq\delta_2^2 + \delta_1\delta_2((1-p)q + (1-q)p)) - \delta_1^2\delta_2^2) - \delta_1^2\delta_2^2 > 0$ and for $(i_1, i_2, j_1, j_2) \in B$ and sufficiently close to the origin, $\frac{dV}{dt} > 0$. This implies that the origin has an unstable manifold in B. Therefore, because any trajectory of system (5) can never leave the box B, system (5) has an attractor as the ω-limit set and hence the virus does survive. □

The result from Theorem 2 holds for non-symmetric cases: a node from set S_1 sees different portion of nodes with curing rate δ_1 than a node from set S_2 ($p \neq q$). In the symmetric case ($p = q$), a more general result with m different curing rates can be derived, as in the case of the regular graph, described in Theorem 1.

3 Optimal heterogenous protection of complete bi-partite graphs

We will not consider the simple case of optimization for a regular graph.

For any bi-partite graph, the threshold for the heterogenous case is fixed and equal to $\delta^* = \beta\sqrt{MN}$. The threshold can be reached for different values of δ_1, δ_2, p and q. For example, for $(\delta_1 = \beta M, \delta_2 = \beta N, p = 1, q = 0)$ the threshold is reached with δ_1 applied on nodes from set S_1, while for $(\delta_1 = \beta M, \delta_2 = \beta N, p = 1, q = 0)$ the threshold is also reached and the curing rate δ_1 is now applied on the nodes from the other set. The question is how can we decide which solution is better. One of the options is to minimize the total protection strategy applied on the network, while reaching the threshold. The total protection strategy can be defined as a sum of all protection strategies and we will denote it by D

$$D = \sum_{l=1}^{M+N} \delta_l = Mp\delta_1 + M(1-p)\delta_2 + Nq\delta_1 + N(1-q)\delta_2 \tag{8}$$

For the previous two cases, the total protection strategy is different. In case $(\delta_1 = \beta M, \delta_2 = \beta N, p = 1, q = 0)$, the total protection strategy is $D = \beta(M^2 + N^2)$, and in the other case, $D = 2\beta MN$, which is always smaller than or equal to the first case.

Let us formulate the optimization problem as follows:

Problem 1. Minimize

$$D = Mp\delta_1 + M(1-p)\delta_2 + Nq\delta_1 + N(1-q)\delta_2 \tag{9}$$

subject to the conditions

$$\beta\sqrt{MN} = \frac{\delta_1\delta_2}{\sqrt{(1-p)(1-q)\delta_1^2 + pq\delta_2^2 + \delta_1\delta_2(p(1-q)+q(1-p))}} \tag{10}$$
$$0 \leq p, q \leq 1$$
$$0 < \delta_1, \delta_2$$

The optimization problem is non-linear with non-linear conditions. However, from [17], we know that the minimum of the function D for any graph and any set of curing rates is equal to the number of links L in the network, multiplied by 2β,

$$D_{\min} = 2\beta L.$$

In the case of the complete bi-partite graph, the minimum is $D_{\min} = 2\beta MN$ and it is reached for $(\delta_1 = \beta M, \delta_2 = \beta N, p = 1, q = 0)$ or $(\delta_1 = \beta N, \delta_2 = \beta M, p = 0, q = 1)$. This means that for $N > M$, the larger curing rate proportional to the number of links in set S_1 will be assigned to the nodes from that set. The larger curing rate is assigned to the more connected nodes.

Further, we can have a situation, where curing rates (δ_1, δ_2) are fixed and we will optimize the parameters (p, q). This optimization problem can be formulated as follows.

Problem 2. For two fixed curing rates δ_1, δ_2, minimize function (8), subject to the conditions (10).

From the threshold condition we can determine one of the variables p or q. We will derive equations for variable q (the case with p is analogue),

$$q = \frac{\delta_1(MN\delta_1(1-p) + MN\delta_2 p - \delta_1\delta_2^2)}{MN(\delta_1^2(1-p) + \delta_1\delta_2(2p-1) + \delta_1^2)} \tag{11}$$

By substituting q in D, the total sum of curing rates becomes a function of parameter p only and optimization is simplified. The function is of the form $D(p) = \frac{P_2(p)}{P_1(p)}$ where $P_1(p)$ is a polynomial of the first order in p and $P_2(p)$ is a polynomial in the second order in p.

Lemma 2. *For any fixed δ_1, δ_2, the optimal solution of minimization problem 2 is on the boundary of the region ($p = 0$ or $p = 1$ or $q = 0$ or $q = 1$).*

Proof. The function $D(p)$ is not defined for $P_1(p) = 0$, which holds for $p = \frac{\delta_1}{\delta_1 - \delta_2}$. The value $\frac{\delta_1}{\delta_1 - \delta_2}$ does not belong to the interval $[0, 1]$. The second derivative of $D(p)$ is strictly negative in the interval $q \in [0, 1]$.

$$\frac{d^2D(p)}{dp^2} = -\frac{2\delta_1^2\delta_2^2(\delta_1 - \delta_2)^2}{(\delta_1(1-q) + \delta_2 q)} < 0, q \in [0, 1]$$

Therefore, $D(p)$ is concave in the interval of interest and minimum is on the boundaries of the interval. □

For given δ_1, δ_2, it is not always possible to reach the threshold. In the case $\delta_1, \delta_2 < \beta\sqrt{MN}$, the threshold cannot be reached and the network is in the state of permanent infection. For example, if $\delta_1, \delta_2 < \beta\sqrt{MN}$ and $\delta_1 > \delta_2$, if we take only the larger curing rate for the whole network, we have $\frac{\beta}{\delta_1} < \beta\frac{1}{\sqrt{MN}}$. In the case $\delta_1, \delta_2 > \beta\sqrt{MN}$, the network is cured, however, the network is above the threshold and a higher curing rate than necessary is applied.

If the threshold can be reached, Lemma 2 shows that either set S_1 or set S_2 is completely protected with only one curing rate. In order to minimize the sum of curing rates we are interested how many times we can apply smaller curing rates. Without loss of generality, let $\delta_1 < \beta\sqrt{MN} < \delta_2$ and $N > M$. Firstly, we will assign δ_2 to all the nodes from larger set with N nodes and δ_1 to the smaller set. If the effective spreading rate obeys $\frac{\beta}{\delta^*} > \frac{1}{\sqrt{MN}}$, than $p = 1$, and q can be calculated from equation (11). In the case $\frac{\beta}{\delta^*} < \frac{1}{\sqrt{MN}}$, the network is cured and below the threshold. Then $q = 0$ and p can be calculated from the condition for the threshold.

4 Conclusion

The epidemic theory is widely applied on many networking problems. The SIS model, on which we have focused here, is applied in malware modeling in the Internet [7], [5], [14], information dissemination in P2P and ad-hoc networks [9], [10] and propagation of faults and failures [8]. The two types of topologies that we considered, namely the regular graph and the complete bi-partite graph, arise as subnet structures in telecommunication networks. We have studied the influence of heterogenous protection in regular and complete bi-partite graphs.

Using Lyapunov's stability theorem, we have shown that for regular graphs, the epidemic threshold satisfies $\frac{\beta}{\delta^*} = \frac{1}{k}$, where δ^* is defined as the weighted harmonic mean of $\delta_1, ..., \delta_m$. This result holds under the assumption of complete symmetry, where each node sees the same fraction of different curing rates. Without this assumption, the problem becomes significantly complex [18].

Further, we have considered the heterogenous case with 2 curing rates for the complete bi-partite graph. The threshold, given by Eq. (7) becomes the geometric mean of curing rates δ_1, δ_2 for $p = 1, q = 0$ and the weighted harmonic mean if $p = q$. For other values of p and q, total curing rate δ^* belongs to the interval $[\delta_1, \delta_2]$.

Many different pairs of curing rate can satisfy the threshold equation, therefore the question which solution is more optimal rises. We consider the optimality of heterogeneous protections for complete bi-partite graph with the respect to sum of all applied curing rates and concluded that global optimum in this respect is equal to the number of links in the complete bi-partite graph. For the case of fixed δ_1 and δ_2, the optimal solution is on the boundaries of $(p, q) \in [0, 1] \times [0, 1]$.

References

1. N. T. J. Bailey, *The Mathematical Theory of Infectious Diseases and its Applications*, Charlin Griffin & Company, London, 2nd ed., 1975.
2. D. M. Cvetkovic, M. Doob, H. Sachs, *Spectra of graphs, Theory and Applications*. Johan Ambrosius Barth Verlag, Heidelberg, third edition, 1995.
3. J. Guckenheimer, P. Holmes, *Nonlinear oscillations, dynamical systems, and bifurcations of vector fields*. Applied Mathematical Sciences, Vol. 42, Springer-Verlag, New York, 1983.
4. D. J. Daley, J. Gani, *Epidemic modelling: An Introduction*, Cambridge University Press, 1999.
5. A. Ganesh, L. Massoulié and D. Towsley, *The Effect of Network Topology on the Spread of Epidemics*, Proc. IEEE INFOCOM'05, Miami, March 2005.
6. M. Garetto, W. Gong, D. Towsley, *Modeling Malware Spreading Dynamics*, Proc. IEEE INFOCOM'03, San Francisco, April 2003.
7. J. O. Kephart, S. R. White, *Direct-graph epidemiological models of computer viruses*, Proc. IEEE Computer Society Symposium on Research in Security and Privacy, pp. 343-359, May 1991.
8. F. G. Coffman Jr., Z. Ge, V. Misra and D. Towsley, *Network resilience: exploring cascading failures within BGP,* Proc. 40th Annual Allerton Conference on Communications, Computing and Control, October 2002.
9. D. Chakrabarti, J. Leskovec, C. Faloutsos, S. Madden, C. Guestrin, M. Faloutsos, *Information Survival Threshold in Sensor and P2P Networks,* Proc. IEEE INFOCOM'07, Anchorage, May 2007.
10. P. Th. Eugster, R. Guerraoui, A. M. Kermarrec, L. Massoulié, *From Epidemics to Distributed computing*, IEEE Computer, Vol. 37, No.5, 60-67, May 2004.
11. A. Shwartz, A. Weiss, *Large Deviations for Performance Analysis*, Chapmann & Hall, London, 1995.
12. Y. Wang, D. Chakrabarti, C. Wang, C. Faloutsos, *Epidemic spreading in real networks: An eigenvalue viewpoint*, 22nd Symposium in Reliable Distributed Computing, Florence Italy, Oct. 6-8, 2003.
13. Y. Wang, C. Wang, *Modeling the Effects of Timing Parameters on Virus Propagation*. ACM Workshop on Rapid Malcode, Washington, DC, Oct. 27, 2003.
14. R. Pastor-Satorras and A. Vespignani, *Epidemic Spreading in Scale-Free Networks*, Physical Review Letters, Vol. 86, No. 14, April, 3200-3203.
15. P. Van Mieghem, *Performance Analysis of Communication Systems and Networks*, Cambridge University Press, 2006.
16. P. Van Mieghem, J. Omic and R.E. Kooij, *Virus Spread in Networks*, accepted for IEEE Transactions on Networking.
17. J. Omic, A. Orda and P. Van Mieghem, *Protecting Against Network Infections: A Game Theoretic Perspective*, submitted to IEEE INFOCOM'09.
18. P. Van Mieghem, J. Omic, *In-homogeneous Virus spread in Networks*, Technical report 20080801, http://www.nas.its.tudelft.nl/people/Piet/TUDelftReports.html
19. J. Omic, R.E. Kooij and P. Van Mieghem, *Virus Spread in Complete Bi-partite Graphs*, Proc. BIONETICS 2007, Budapest, December, 2007.