Topology independent SIS process: an engineering viewpoint

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Abstract

A Susceptible-Infected-Susceptible (SIS) spreading process, occurring on complex networks that are characterized by a special form of contact dynamics called "acquisition exclusivity", is studied. Assuming statistical independence of joint events, we find analytic solutions for the stationary probabilities that network nodes are infected, and more importantly, find that these solutions are independent of the network topology. We explore the possibilities to use the studied set-up as an engineering solution for controlled spreading on technological networks. In that context, an example for controlled sharing of viral countermeasures in networks is presented. Considering the high epidemic threshold that characterizes the process, "acquisition exclusivity" is suggested as a method for eradication of viral infections from networks.

Keywords: Networks, Stochastic spreading, SIS, Threshold values, Infection eradication

1. Introduction

Stochastic spreading processes, such as epidemic, rumor, information spreading, have been in the focus of the scientific community in the last half century, following the rapid development of technological, social, transportation and communication networks. The interest in spreading processes

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primarily originates from their descriptive power of potentially catastrophic events such as cascading failures and blackouts in electrical networks or epidemics with new strains of deadly viruses [1–6], as well as their potential use as mechanisms for diffusion of innovations [7, 8] and information spreading [9, 10].

In this paper, following our previous work [11], we focus on some engineering aspects related to stochastic spreading, primarily on exploring the possibilities to use these processes as a basis for development of applications for controlled spreading. For that purpose, we investigate a SIS type of process, occurring on networks characterized by a special form of node contact dynamics that we call *acquisition exclusivity*. The term node contact dynamics refers to the method by which a node communicates or exchanges content with its neighbors within one time step of the dynamics on the network. In that sense, the term *acquisition exclusivity* is used for a contact behavior where at each instance a node contacts and acquires some form of material from exactly one of its neighbors. Similar set-ups may be found in the literature, as special cases in the analysis of virus sprading [12], as well as for some theoretical models, for example the Voter model [13] in statistical mechanics. Unlike the Voter model, which is a two state sharing process, the SIS process is characterized by node self-induced transition from status Infected to status Susceptible. Other technological aspect have been of interest as well. As shown here, SIS spreading process (as might be expected for other re-occurring spreading processes) taking place on networks working in acquisition exclusivity mode, is characterized by an epidemic threshold much higher then the one occurring on regular networks [14]. As a consequence, acquisition exclusivity may be used as an alternative contact dynamics in engineering networks when an epidemic occurs, in order to remove the epidemic from a network.

When studying spreading processes on networks, some of the relevant questions are: What is the fraction of nodes in the network that will become infected? Is there an epidemic threshold (i.e. critical point) for the spreading rate which separates the regime when there is no epidemic in the network from the one where a non-zero fraction of the nodes will become infected? What does it take to eliminate an epidemic from the network? In order to answer these questions different authors have taken different approaches [15–21]. Due to network heterogeneity and the stochastic character of the spreading processes, the developed mathematical models have always been built around certain assumptions. In our work we adopt the approach of

Wang et al. [15] and *Chakrabarti et al.* [16], which is based on the assumption of statistical independence of joint events (the point estimate approximation [16]). Using this approach, we show that the SIS spreading process, under the analyzed circumstances, is analytically solvable, i.e. an analytical solutions for the nodes status probabilities may be found. Furthermore, the obtained results are topology independent.

Note should be taken on the point estimate approximation [16]. Numerical simulations have shown that this assumption, in networks characterized by the analyzed contact dynamics, is valid under several constrains. First, a large minimal node degree is required. This prerequisite is easily met in networks where the cost per link is relatively low. Virtual networks built around different applications (for example social networks, sharing networks e.t.c.) are a typical example. In wireless networks, and primarily sensor networks [22], this condition may be fulfilled with minimal extra costs. Next, as indicated by the numerical simulations in Section IV, statistical independence of joint events is emphasized for large values of the infection rate β. In that sense, in engineering solutions one should consider taking $\beta \to 1$. The last, together with acquisition exclusivity mode of contact dynamics, is typical for the *Voter model* [13].

2. Description of the analyzed process

Let $A = [a_{ij}]$ be the network adjacency matrix, and $p(i \leftarrow j)$ the contact probability, i.e. the probability that node i will contact node j at each instance and, possibly, acquire some form of spreading content. Due to *acquisition exclusivity*, $\sum_{j=1,N} p(i \leftarrow j) = 1$. It is considered that the analyzed graph is unweighed, connected and bidirectional, i.e. the network adjacency matrix is symmetric and irreducible. Next, the contact probability $p(i \leftarrow j) > 0$ is strictly positive when $a_{ij} = 1$, and in general the contact probabilities $p(i \leftarrow j) \neq p(j \leftarrow i)$. Therefore, the matrix $B = [b_{ij}]$, where $b_{ij} = p(i \leftarrow j)a_{ij}$ is irreducible and asymmetric, satisfying $\sum_{j=1,N} b_{ij} = 1$. It is assumed that no instantaneous reinfection may occur within one time step.

Having in mind the stated above and adopting the approach of [15, 16], the studied SIS type process is described with the following set of probability

difference equations:

$$\begin{aligned} p_i^S(t+1) &= p_i^S(t)\left(1-\sum_{j=1}^{N}\beta b_{ij}p_j^I(t)\right)+\delta p_i^I(t) \\ p_i^I(t+1) &= p_i^S(t)\sum_{j=1}^{N}\beta b_{ij}p_j^I(t)+(1-\delta)p_i^I(t) \\ 1 &= p_i^S(t)+p_i^I(t), \end{aligned}$$

where $p_i^S(t)$ and $p_i^I(t)$ denote the probabilities that node i is susceptible and infected at time t, respectively. Parameter β, usually referred to as the "infection rate", is the probability that a susceptible node connected to an infected one will become infected itself, while δ the probability that an infected node will become susceptible again, both in one time step.

It is standard practice, using the normalization constrain $p_i^S(t) = 1-p_i^I(t)$ to transform the system above into:

$$p_i^I(t+1) = (1-p_i^I(t))\sum_{j=1}^{N}\beta b_{ij}p_j^I(t)+(1-\delta)p_i^I(t), \tag{1}$$

and study the process in question by analyzing the system (1).

Two stationary solutions of (1) are obvious: $p_1^I = p_2^I = ... = p_N^I = p^I = 0$ and $p_1^I = p_2^I = ... = p_N^I = p^I = 1-\frac{\delta}{\beta}$. The second solution has no meaning if $\frac{\delta}{\beta} > 1$.

3. Validity of the obtained solutions

In this section we prove that the obtained fixed points as stationary solutions of the process described with (1). For that purpose an stability analysis is performed on the system (1) in two parametric regions: $\frac{\delta}{\beta} < 1$ and $\frac{\delta}{\beta} > 1$.

Theorem 1. *For the system described with the set of equations (1) and $\delta > \beta$, the point $p^I = (p_1^I, p_2^I, ..., p_N^I) \in [0,1]^N$, such that $p_1^I = p_2^I = ... = p_N^I = p^I = 0$, is unique and globally (on $[0,1]^N$) asymptotically stable fixed point.*

Proof. The system (1), may be written in a matrix form, as follows:

$$\mathbf{p^I}(t+1) = ((1-\delta)\mathbf{I}+\beta\mathbf{D}(t)\mathbf{B})\,\mathbf{p^I}(t) = \mathbf{S}(t)\mathbf{p^I}(t), \tag{2}$$

with $\mathbf{D}(t)$ being a diagonal matrix, with diagonal elements $d_{ii} = 1 - p_i(t)$. Notice that for any $t \geq 0$:

$$\mathbf{S}(t) \;\; = \;\; (1-\delta)\mathbf{I} + \beta\mathbf{D}(t)\mathbf{B} \leq (1-\delta)\mathbf{I} + \beta\mathbf{B} = \mathbf{S} \tag{3}$$

Each of the matrices $\mathbf{S}(t) \geq \mathbf{0}$, with $\mathbf{0}$ being $N \times N$ zero matrix, is non-negative. This further implies that the product of any of the matrices $\mathbf{S}(t)$ is non-negative as well; one may also claim that exists $d \in Z^+$, such that for $k > d$, the product of k matrices $\mathbf{S}(t)$ is strictly positive. The last conclusion follows from the fact that the graph is strongly connected which makes the matrices $\mathbf{S}(t)$ irreducible. Furthermore, since $\mathbf{B}$ is row stochastic, the largest eigenvalue of the matrix $\mathbf{S}$ is $\lambda_{1_S} = 1 - \delta + \beta < 1$. Then:

$$\lim_{t\to\infty} \mathbf{p}^{\mathbf{I}}(t+1) \;\; = \;\; \lim_{t\to\infty} \mathbf{S}(t)...\mathbf{S}(0)\mathbf{p}^{\mathbf{I}}(0) \leq \lim_{t\to\infty} \mathbf{S}^{t+1}\mathbf{p}^{\mathbf{I}}(0) = \mathbf{0}_{N\times 1},$$

yielding $\limsup_{t\to\infty} \mathbf{p}^{\mathbf{I}}(t) = \mathbf{0}_{N\times 1}$. On the other hand, having in mind the previous discussion, one may claim that $\liminf_{t\to\infty} \mathbf{p}^{\mathbf{I}}(t) = \mathbf{0}_{N\times 1}$ (the product of non-negative matrices is non-negative). Finally, one obtains that:

$$\lim_{t\to\infty} \mathbf{p}^{\mathbf{I}}(t) \;\; = \;\; \mathbf{0}_{N\times 1}, \tag{4}$$

that proves the Theorem statement. □

Lemma 1. *For the system described with the set of equations (1) and $\beta > \delta$, the point $p^I = (p_1^I, p_2^I, ..., p_N^I) \in [0,1]^N$, such that $p_1^I = p_2^I = ... = p_N^I = p^I = 0$, is unstable fixed point.*

Proof. By perturbing the equation (1) in the area around the point $p_1^I = p_2^I = ... = p_N^I = p^I = 0$, one obtains:

$$\delta\mathbf{p}^{\mathbf{I}}(t+1) \;\; = \;\; [(1-\delta)\mathbf{I} + \beta\mathbf{B}]\delta\mathbf{p}^{\mathbf{I}}(t) = \mathbf{S}\delta\mathbf{p}^{\mathbf{I}}(t)$$

Since the largest eigenvalue of the matrix $\mathbf{S}$, $\lambda_{1_\mathbf{S}} = 1-\delta+\beta\lambda_{1_\mathbf{B}} = 1-\delta+\beta > 1$, the origin is an unstable fixed point. The proof is completed. □

Lemma 2. *For the system described with (1), assuming that $p_i^I(0) > 0$ for at least one i, then there exist t', such that $0 < p_i^I(t) < 1$ is a strict inequality, for every $t > t'$ and $i = \overline{1,N}$.*

Proof. Both sides of the inequality will be proved separately.

a. Assume that at time t, $p_i^I(t) = 0$. Then from (1), $p_i^I(t-1) = 0$, as well as $p_j^I(t-1) = 0$, for all $j \propto i$. Using similar arguments, and by going backwards in time, one may conclude that exists $d < dia$, with dia being the diameter of the graph, such that $p_i^I(s-d) = 0$, for all $i = \overline{1,N}$. But then $p_i^I(0) = 0$ as well, for all $i = \overline{1,N}$, that contradicts the assumption in the Lemma definition.

b. Assume that $p_i^I(t+1) = 1$, at an instance in time $t+1$; then, for the preceding value $p_i^I(t)$ one obtains:

$$p_i^I(t) = \frac{1 - \sum_{j=1}^N \beta b_{ij} p_j^I(t)}{1 - \sum_{j=1}^N \beta b_{ij} p_j^I(t) - \delta}, \tag{5}$$

which is either negative or greater then one, when $\delta > 0$, and therefore not a valid solution for $p_i^I(t) \in [0,1]$, $i = \overline{1,N}$. The proof is completed. □

Let $row_i(\mathbf{A}) = \sum_{j=1,N} a_{ij}$ denotes the sum of the elements of row i of a matrix $\mathbf{A}$ and $maxrow(A) = max_i row_i(\mathbf{A})$ be the maximal value of a row sum for that matrix. Consider $\mathbf{A}_1 = [a_{ij}^{(1)}]$ and $\mathbf{A}_2 = [a_{ij}^{(2)}]$ be two non-negative square matrices of size N, and $\mathbf{C} = [c_{ij}] = \mathbf{A}_2\mathbf{A}_1$ their matrix product. It is obvious that $\mathbf{C}$ is non-negative matrix as well. Now:

$$\begin{aligned} row_i(\mathbf{C}) &= \sum_{j=1}^N \sum_{k=1}^N a_{ik}^{(2)} a_{kj}^{(1)} = \sum_{k=1}^N a_{ik}^{(2)} \sum_{j=1}^N a_{kj}^{(1)} \\ &\leq \sum_{k=1}^N a_{ik}^{(2)} maxrow(\mathbf{A}_1) = row_i(\mathbf{A}_2) maxrow(\mathbf{A}_1), \end{aligned}$$

with the equality holding only if $row_i(\mathbf{A}_1)$ is the same, for every i. From the last relation:

$$maxrow(\mathbf{C}) \leq maxrow(\mathbf{A}_2) maxrow(\mathbf{A}_1). \tag{6}$$

Following (6), it may easily be proven, by induction, that for an arbitrary block product of non-negative matrices $\mathbf{A}_M \dots \mathbf{A}_2\mathbf{A}_1$, $M \in Z^+$:

$$maxrow(\mathbf{A}_M \dots \mathbf{A}_2\mathbf{A}_1) \leq maxrow(\mathbf{A}_M) ... maxrow(\mathbf{A}_2) maxrow(\mathbf{A}_1). \tag{7}$$

Theorem 2. *For the system described with the set of equations (1) and $\beta > \delta$, the point $p^I = (p_1^I, p_2^I, ..., p_N^I) \in [0,1]^N$, such that $p_1^I = p_2^I = ... = p_N^I = p^I = 1 - \frac{\delta}{\beta}$, is globally asymptotically stable fixed point.*

Proof. We will introduce the following transformation of variables:

$$p_i(t) \;=\; p_i^I(t) - 1 + \frac{\delta}{\beta},$$

that transforms the equation (1) into:

$$p_i(t+1) \;=\; (1-\beta)p_i(t) + \left(\frac{\delta}{\beta} - p_i(t)\right)\sum_{j=1}^{N}\beta b_{ij}p_j(t). \tag{8}$$

Note that (8) is defined for $p_i(t) \in [-1+\frac{\delta}{\beta}, \frac{\delta}{\beta}]$. In what follows, the global asymptotic stability of the translated system (8) is being proved.
Let $\mathbf{p}(t) = [p_1(t)\ p_2(t)\ ...\ p_N(t)]^T$ be a node status probability vector and $\mathbf{D_p}(t) = [d_{ij}(t)]$ an $N \times N$ diagonal matrix, such that $d_{ii}(t) = \frac{\delta}{\beta} - p_i(t)$. System of equations (8) may be written in matrix form as:

$$\mathbf{p}(t+1) \;=\; \left((1-\beta)\mathbf{I} + \beta\mathbf{D_p}(t)\mathbf{B}\right)\mathbf{p}(t) = \mathbf{S}(t)\mathbf{p}(t), \tag{9}$$

with

$$\mathbf{S}(t) = (1-\beta)\mathbf{I} + \beta\mathbf{D_p}(t)\mathbf{B} \tag{10}$$

Assume that at time $t = 0$ system is in some initial state $\mathbf{p}(0)$. Then, at time $t = s$ the state of the system may be described with the probability matrix equation:

$$\mathbf{p}(s) \;=\; \prod_{t=0}^{s-1}\mathbf{S}(t)\mathbf{p}(0) = \prod_{t=0}^{s-1}\left((1-\beta)\mathbf{I} + \beta\mathbf{D_p}(t)\mathbf{B}\right)\mathbf{p}(0). \tag{11}$$

Following Lemma 2, there exists t' such that for $t > t'$, $-1+\frac{\delta}{\beta} < p_i(t) < \frac{\delta}{\beta}$ is a strict inequality, and consequently $0 < d_{ii}(t) < 1$, as well. In regard to the matrices $\mathbf{S}(t)$, for $t > t'$ one may summarize that:

i. $0 \le s_{ij}^{(t)} \le s_{ij}$, where $\mathbf{S} = [s_{ij}] = (1-\beta)\mathbf{I} + \beta\mathbf{B}$, $\mathbf{S}(\mathbf{t}) = [s_{ij}^{(t)}]$, satisfying: if $s_{ij} > 0$, then $0 < s_{ij}^{(t)} < s_{ij}$.

ii. Matrices $\mathbf{S}(t)$, $\mathbf{S}$ are irreducible.

iii. $\sum_{j=1,N} s_{ij}^{(t)} < 1$ and $\sum_{j=1,N} s_{ij} = 1$.

From iii., it follows that for $t > t'$, $maxrow(\mathbf{S}(t)) < 1$. Consider the sequence of matrices $\mathbf{S}(t'+s), \mathbf{S}(t'+s-1), ..., \mathbf{S}(t'+1)$ starting at $t'+1$ with length s. It is obvious that:

$$\mathbf{S}(t'+s)\mathbf{S}(t'+s-1)...\mathbf{S}(t'+1) \geq \mathbf{0}_{N\times N}, \tag{12}$$

with the relation surely being strict inequality for $s \geq dia$. Adequately:

$$maxrow(\mathbf{S}(t'+s)\mathbf{S}(t'+s-1)...\mathbf{S}(t'+1)) > 0 \tag{13}$$

On the other hand, from (7):

$$maxrow\left(\prod_{t=t'+1}^{s}\mathbf{S}(t)\right) \leq \prod_{t=t'+1}^{s} maxrow(\mathbf{S}(t)). \tag{14}$$

Consider the series $..., a_s, a_{s-1}..., a_{t'+1}$, defined with:

$$a_s = \prod_{t=t'+1}^{s} maxrow(\mathbf{S}(t)) = maxrow(\mathbf{S}(s))a_{s-1}, \tag{15}$$

for $s > t'+1$, with $a_{t'+1} = maxrow(\mathbf{S}(t'))$. It is obvious that $.. < a_s < a_{s-1} < ... < a_{t'+1}$, with $a_s > 0$. Assume that a positive lower bound $lb > 0$ exists, such that:

$$lb = \lim_{s\to\infty} a_s = \lim_{s\to\infty} maxrow(\mathbf{S}(s-1))a_{s-1}.$$

Then $\lim_{s\to\infty} maxrow(\mathbf{S}(s)) = 1$. Considering that from (8), one can obtain that $maxrow(\mathbf{S}(s)) = 1-\beta+\beta(\delta/\beta - p_l(s))$, with l being the node with minimal infection probability at time s, the previous relation yields:

$$\lim_{s\to\infty} p_l(s) = -1 + \frac{\delta}{\beta}, \text{ or } \lim_{s\to\infty} p_l^I(s) = 0 \tag{16}$$

Assume (16) holds. Then exists $s(\epsilon)$, for ϵ arbitrary small, such that $p_l^I(s) < \epsilon$. Then, following (1):

$$\begin{aligned}
\epsilon &> p_l^I(s) = \left[1-p_l^I(s-1)\right]\sum_{j=1}^{N}\beta b_{ij}p_j^I(s-1) + (1-\delta)p_l^I(s-1) > \\
&> (1-\delta)p_l^I(s-1), \\
\epsilon &> p_l^I(s) > \left[1-p_l^I(s-1)\right]\sum_{j=1}^{N}\beta b_{ij}p_j^I(s-1) > \\
&> \left[1-p_l^I(s-1)\right]\beta b_{ij}p_j^I(s-1), \text{ for j} \propto \text{l}
\end{aligned}$$

yielding:

$$\begin{aligned} p_l^I(s-1) &< \frac{\epsilon}{1-\delta}, \\ p_j^I(s-1) &< \frac{\epsilon}{(1-p_l^I(s-1))\beta b_{ij}} < \frac{\epsilon}{c\beta b_{ij}}, \text{ for j} \propto \text{l}. \end{aligned}$$

In the second relation $0 < c < 1 - \frac{\epsilon}{1-\delta}$ may be chosen arbitrary. Using similar arguments, and going backward in time, as done in the proof of Lemma 2, one may show that for $d < dia$, $p_i^I(s-d) < \epsilon g_i(\beta, \delta, b_{ij}, d)$, with $g_i(\beta, \delta, b_{ij}, d)$ taking a finite value, for all $i = \overline{1, N}$. Consequently, exits s such that all $p_i^I(s-d)$ may be made arbitrarily small, and therefore:

$$\lim_{s\to\infty} \mathbf{p}^I(s) = 0 \text{ , and } \lim_{s\to\infty} \mathbf{p}(s) = -1 + \frac{\delta}{\beta}.$$

But the last relations are in contradiction with the proved Lemma 1, and therefore (16) can not hold. Consequently $lb = 0$, yielding:

$$\lim_{s\to\infty} maxrow\left(\prod_{t=t'+1}^{s} \mathbf{S}(t)\right) \leq \lim_{s\to\infty} \prod_{t=t'+1}^{s} maxrow(\mathbf{S}(t)) = 0. \tag{17}$$

From (13) and (17) one may conclude that when $s \to \infty$, $maxrow(\mathbf{S}(t'+s)\mathbf{S}(t'+s-1)...\mathbf{S}(t'+1))$ is bounded both from bellow and above by zero, i.e equals zero in the limiting case. Considering the last conclusion, and (12):

$$\lim_{s\to\infty} \mathbf{S}(t'+s)\mathbf{S}(t'+s-1)...\mathbf{S}(t'+1) = \mathbf{0}_{N\times N} \tag{18}$$

Finally, from (11):

$$\lim_{s\to\infty} \mathbf{p}(s+1) = \lim_{s\to\infty} \prod_{t=t'+1}^{s} \mathbf{S}(t) \prod_{t=0}^{t'} \mathbf{S}(t)\mathbf{p}(0) = \mathbf{0}_{N\times 1},$$

that proves the Theorem. □

From the previous discussion, we may define the average number of susceptible nodes in the network, $N_S = \lim_{t\to\infty} \sum_{i=\overline{1,N}} p_i^S(t)$, and the average number of infected nodes in the network, $N_I = \lim_{t\to\infty} \sum_{i=\overline{1,N}} p_i^I(t)$, for different parametric regions:

$$N_S = \begin{cases} N, & \frac{\delta}{\beta} > 1 \\ \frac{\delta}{\beta} N, & \frac{\delta}{\beta} < 1 \end{cases}, \; N_I = \begin{cases} 0, & \frac{\delta}{\beta} > 1 \\ (1 - \frac{\delta}{\beta})N, & \frac{\delta}{\beta} < 1 \end{cases},$$

with N being the number of nodes in the graph. As one may notice, the obtained result for the average number of nodes that are in one of the two possible statuses, does not depend on the network topology, but only on the parameters that describe the infection process (β and δ).

4. Numerical simulations, analyzes and possible applications

In this section we present the results of the numerical (stochastic) simulations of the studied set up and compare these results, which we consider the reality, with the analytical results obtained in the previous Sections. We aim towards determining the region in which the system (1) most adequately represents the reality, in order to derive protocols for development of applications for controlled spreading in networks. The simulations are initially performed on Barabási - Albert graphs, where the minimal node degree is being altered by changing the parameter m. We focus on this type of graphs since they are synthetic networks whose structure is most similar to real world networks. We use three networks:

- Barabási - Albert network with $N = 1000$ nodes, $m_0 = 4$, $m = 3$, total of $L = 2991$ links, and the largest eigenvalue of the network adjacency matrix $\lambda_{1_A} = 13.8983$; we refer to this network in the further text as BA(3,4,1000).

- Barabási - Albert network with $N = 1000$ nodes, $m_0 = 5$, $m = 4$, total of $L = 3985$ links, and the largest eigenvalue of the network adjacency matrix $\lambda_{1_A} = 16.6121$; we refer to this network in the further text as BA(4,5,1000).

- Barabási - Albert network wit $N = 1000$ nodes, $m_0 = 8$, $m = 7$, total of $L = 6951$ links, and the largest eigenvalue of the network adjacency matrix $\lambda_{1_A} = 27.1275$; we refer to this network in the further text as BA(7,8,1000).

In the analysis, connection probabilities $p(i \leftarrow j)$ have been chosen randomly for each existing link.

The stochastic simulations are performed as follows: first the system network-infection, starting from arbitrary initial conditions, is iterated for 2000 time steps in order the system reaches the stationary regime. Then, the system is further iterated for another 8000 time steps, after which the results

are averaged. The analysis is repeated several times for different values of the parameters β and δ. The results of the analysis are presented in Figs. 1, 2 and 3.

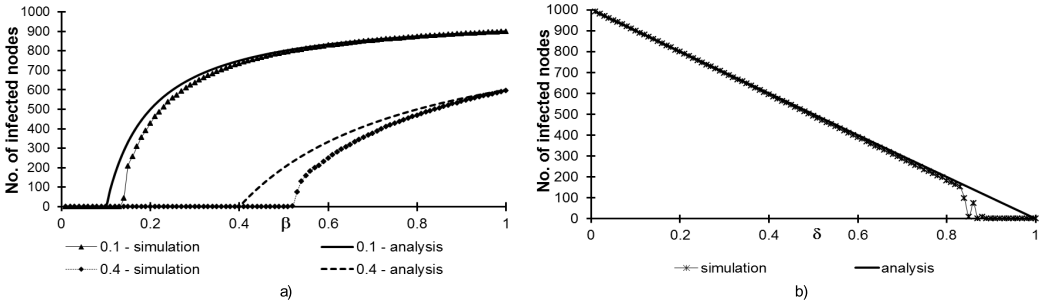

Figure 1: Number of infected nodes for the SIS type process occurring on the BA(3,4,1000) network: a) as a function of β for $\delta = 0.1$ (black full line - analytic result, black line with triangle markers - simulation); $\delta = 0.4$ (black dashed line - analytic result, black dashed line with rhomboid markers - simulation); b) as a function of δ for $\beta = 1$ (black full line - analytic result, black line with X markers - simulation).

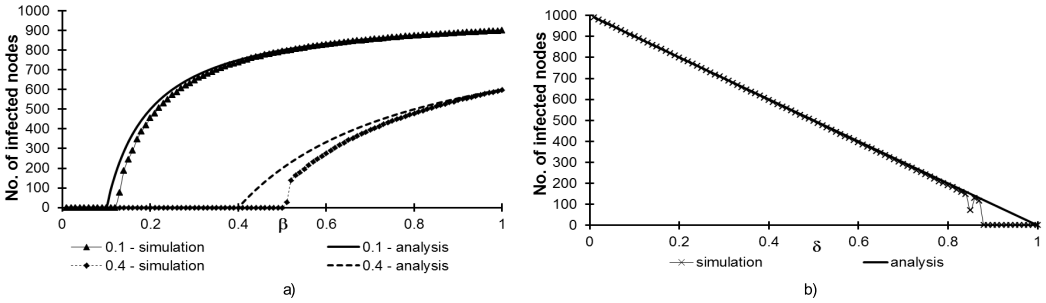

Figure 2: Number of infected nodes for the SIS type process occurring on the BA(4,5,1000) network: a) as a function of β for $\delta = 0.1$ (black full line - analytic result, black line with triangle markers - simulation); $\delta = 0.4$ (black dashed line - analytic result, black dashed line with rhomboid markers - simulation); b) as a function of δ for $\beta = 1$ (black full line - analytic result, black line with X markers - simulation).

From the figures, one may conclude as follows:

- The validity of the analytical results increases as the minimal node degree in the network increases.
- There is a good degree of accuracy for large β/δ ratios.

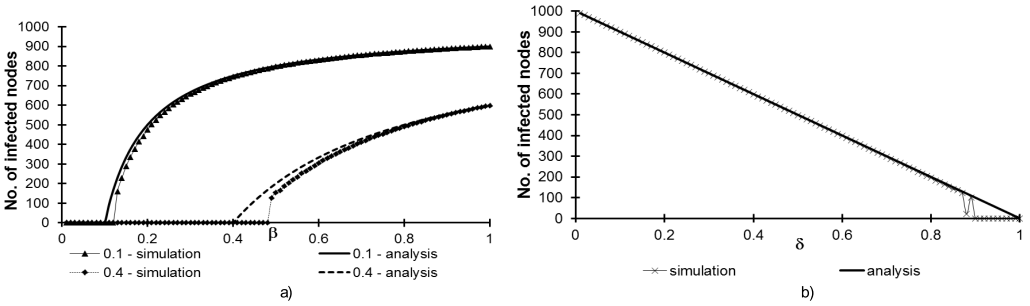

Figure 3: Number of infected nodes for the SIS type process occurring on the BA(7,8,1000) network: a) as a function of β for $\delta = 0.1$ (black full line - analytic result, black line with triangle markers - simulation); $\delta = 0.4$ (black dashed line - analytic result, black dashed line with rhomboid markers - simulation); b) as a function of δ for $\beta = 1$ (black full line - analytic result, black line with X markers - simulation).

- The accuracy is much higher (and almost perfect match-up might be expected) for larger values of β (β close to one).

We believe that the last conclusion is the most important from an engineer's point of view, and use it as a basis for the discussion in the following section.

To re-evaluate the results obtained from the simulations on the Barabási - Albert graphs, as well as to analyze the effect that network clustering has on the studied process, we have further conducted numerical simulations on several Holme-Kim networks (HK) [23]. The analyzed networks defer both in respect to the minimal node degree, as well as the level of clustering. Similar as before, the minimal node degree is being altered by changing the parameter m, while the clustering coefficient by changing the parameter p_t. Simulation procedure is identical as in the case of BA graphs. The results of the simulations for four of the studied networks are presented in Fig. 4.

The analysis on the HK networks confirms the conclusions previously drawn from the analysis conducted on the BA networks. Minimal node degree, in respect to the topology, predominantly affects the validity of the analytic results obtained in the previous sections. The level of clustering has minimal effect on the results, which is detectable only near the epidemic threshold.

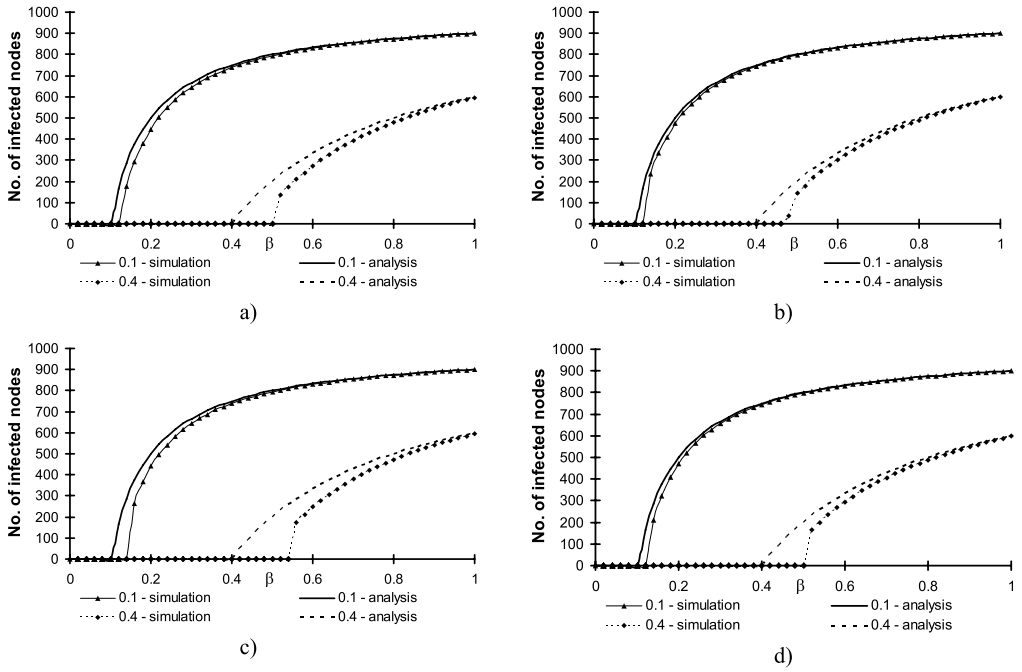

Figure 4: Number of infected nodes for the SIS type process occurring on the HK(m,p_t,N) networks as a function of β for $\delta = 0.1$ (black full line - analytic result, black line with triangle markers - simulation) and $\delta = 0.4$ (black dashed line - analytic result, black dashed line with rhomboid markers - simulation): a) HK(4,0.3,1000) network, b) HK(7,0.3,1000) network, c) HK(4,1.0,1000) network, d) HK(7,1.0,1000) network.

4.1. Controlled spreading of content

Using the conclusions above, we consider the possibility for development of distributed applications for controlled spreading of content in complex networks, primarily technological (computer) networks. We base our suggestion on the fact that by making an adequate choice of the process parameters β and δ, using the principles presented in this paper, one may fine-tune the number of nodes (application users) that will posses a certain content at an instance in time.

Possible operational scenario for information spreading on computer networks may be as follows:

- The application for controlled spreading (sharing) of content is deployed on users personal computers. Upon installation, connections to a number of other application users are made, therefore creating a virtual

network. A minimal number of neighbors for each user ($d_{min} >> 1$, $d_{min} > 3$ is in order) is pre-defined. The choice of neighbors can be made either by the user, or by the application by randomly connecting the new to a number of existing users. Each user may additionally add links to other application users.

- The virtual network functions in acquisition exclusivity mode, and the content through the virtual network is shared as a SIS type of a process: at each time step a user (deployed application) contacts exactly one of the neighbors and acquires the spread content (file, document, book, movie etc) with probability β ($\beta = 1$ recommended). The acquired content is stored on the users machine, in a dedicated folder created by the application. The stored content is available to be acquired by each of the neighbors. At each following time step the application decides to delete the content from the application folder with probability δ. Once erased, the same content might be reacquired in a subsequent step.

Note should be taken that the virtual network uses the physical resources of an existing data-communication network, for example the Internet.

Assuming conditions that provide statistical independence of joint events are satisfied (d_{min} sufficiently large, $\beta \to 1$), one may claim that the expected number of nodes that posses the spread content in the stationary regime is $(1 - \delta/\beta)N$, with N being the number of application users (number of nodes in the virtual network). Parameter β will be pre-set for the application by the developer (distributer). Parameter δ will be different for each sheared item and the information about its actual value might be acquired together with the content in question (encapsulated in the shared content). Therefore, δ will be the controlling variable that will determine the actual number of copies of each item shared trough the application in the virtual network.

4.2. Application for virus eradication from complex networks

On the other practical aspect, the SIS process, under the circumstances described in the paper, is characterized with an epidemic threshold $\beta = \delta$ (similar result is obtained for the contact process [24, 25]). This is significantly higher threshold value, than the one for the SIS infection occurring on networks with regular contact dynamics [14] ($\frac{\beta}{\delta} = \frac{1}{\lambda_{1_A}}$ [15, 16, 26, 18]). In reality, the epidemic threshold for spreading processes on networks characterized with acquisition exclusivity will be even greater then the one obtained

in this paper (in regard to the effect that point estimate approximation has on the epidemic threshold, one may refer to [27]).

Acquisition exclusivity, will surely eradicate any infection for which $\beta \leq \delta$. This is significantly more than what can be achieved by other purely topology manipulative algorithms [28–30]. The process is accompanied by preservation of the graphs topology, under no additional cost. On the down side, acquisition exclusivity will affect the flow of normal data through the network, significantly reducing the information exchange as well. Therefore, we suggest that acquisition exclusivity should be considered only as an alternative contact dynamics in regular networks, when an epidemic outbreak occurs. The following mechanism of action might be considered: when no infection exists, every node in the network exchanges data with each of its neighbors, in every time frame; when infection occurs, the network is switched to acquisition exclusivity mode of work; once the infection is eradicated, the network is switched back to normal regime [14].

For $\beta > \delta$, acquisition exclusivity might be insufficient to complete infection eradication by itself. In this case some form of node immunization or vaccination [31–35] should be considered as well; however, acquisition exclusivity will severely reduce the costs of the additional protective measures. Note, that same effect, as one considered, may be obtained if the network is set to work in a "contact" mode, i.e. if information from one node is transmitted exclusively to one of its neighbors, chosen randomly.

5. Application on spreading virus countermeasures in networks

In this section we give a simple example of an application for controlled spreading of content, as described in Section 4.1. We consider an application for controlled spreading of virus countermeasures, and adequately for malware eradication from networks.

Consider a computer network defined on the graph $G = G(V, E)$, where V is the sets of vertices and E is a set of edges. The graph G represents a real world network and follows a regular contact dynamics [14]. An application that spreads viral countermeasures (and therefore acts as an antivirus package) is installed on each computer (node). The application forms a virtual network $G_1 = G_1(V, E_1)$, that comprises of all vertices of the graph G (the set V) and a set of virtual edges (contacts) E_1. For G_1 a minimal node degree $d_{min} >> 1$ (taking $d_{min} > 3$ is in order) is predefined. The content (countrameasures) is spread through the virtual network as a SIS type of

process, while the virtual network is set to work in acquisition exclusivity mode of contact dynamics, as described in Section 4.1.

Assume that an epidemic of an arbitrary type (SI, SIS, SIRS, SEIRS etc) breaks on the network G. Once the epidemic is recognized, adequate response in form of viral countermeasures is prepared by one or several of the network users. These countermeasures are disseminated using the installed application, by spreading over the network G_1 as a SIS type of process characterized by $\beta^c \to 1$ (in practice $\beta^c = 1$ should be considered) and δ^c. If node i possess countermeasures, the node is "protected" i.e it is neither infected, nor in any of the statuses that characterize the infection occuring on G. A protected node from which countermeasures are removed (deleted) becomes susceptible. Unprotected nodes will be in one of the stages that characterize the infection type and are prone to undergo adequate status changes.

In what follows, we analyze a situation in which the infection occurring on G is of SI type. We consider an infection of SI type due to two reasons. First, this is the worst case scenario for the network - each node will eventually be permanently infected as time elapses. Second, one may argue that virus spreading processes on technological networks are predominantly of SI type. Processes where additional stages exist, like SIS or SIRS for example, are usually a result of additional protective measures (immunization, vaccination, implementation of quarantines).

Let $\mathbf{A} = [a_{ij}]$ denote the adjacency matrix that characterizes the graph G. Further, let $\mathbf{A}' = [a'_{ij}]$ denote the adjacency matrix of the network G_1, and let $p(i \leftarrow j)$ be the contact probabilities. We denote with $\mathbf{B} = [b_{ij}] = [p(i \leftarrow j)a'_{ij}]$ the matrix of contacts for G_1. Having in mind the arguments stated above, and using the approach of *Wang et al.* [15] and *Chakrabarti at al.* [16], the system comprising of the infection and viral countermeasures spreading on networks G and G_1 respectively, can be described with the following set of probability equations:

$$p_i(t+1) = (1-p_i^c(t))\left((1-p_i(t))(1-\prod_{j\propto i}[1-a_{ij}\beta p_j(t)]) + p_i(t)\right) \tag{19}$$

$$p_i^c(t+1) = (1-p_i^c(t))\sum_{j=1}^{N}\beta^c b_{ij}p_j^c(t) + (1-\delta^c)p_i^c(t), \tag{20}$$

with $p_i(t)$ and $p_i^c(t)$ denoting the probabilities that the node i is infected and

possesses countermeasures (is protected) at time t, respectively.

Theorem 3. *For the system described with (19,20) and $\beta^c > \delta^c$, if:*

$$\frac{\delta^c}{\beta^c} < \frac{1}{1+\beta\lambda_{1_A}} \tag{21}$$

the point described with $p_i(t) = 0, p_i^c(t) = 1 - \frac{\delta^c}{\beta^c}$, $i = \overline{1,N}$ is globally asymptotically stable fixed point.

Proof. First, notice that the system of equations (20) (the countermeasure spreading through a virtual network) is independent of the system (19) (spreading of the viral infection through network G). Therefore, in respect to (20), Theorem 2 applies.

In respect to the global asymptotic stability of (19), one may follow the approach of *Chakrabarti et al.* [16]. From (19) and the Wierstrass product inequality, one obtains:

$$\begin{aligned} p_i(t+1) &\leq (1-p_j^c(t))\left((1-p_i(t))\sum_{j\propto i} a_{ij}\beta p_j(t) + p_i(t)\right) \\ &\leq (1-p_j^c(t))\left(\sum_{j\propto i} a_{ij}\beta p_j(t) + p_i(t)\right) \end{aligned}$$

From (21), let $\frac{\delta^c}{\beta^c} = \frac{1}{1+\beta\lambda_{1_A}} - \zeta$, where $0 < \zeta < \frac{1}{1+\beta\lambda_{1_A}}$. From Theorem 2 exists t' such that for every $t > t'$, $\epsilon(t') < \zeta$, where $\|p_j^c(t) - 1 + \frac{\delta^c}{\beta^c}\| < \epsilon(t')$. Accordingly, for $t > t'$ the last inequality takes the following form:

$$\begin{aligned} p_i(t+1) &< (1-(1-\frac{\delta^c}{\beta^c} - \epsilon(t')))(\sum_{j\propto i} a_{ij}\beta p_j(t) + p_i(t)) = \\ &= (\frac{\delta^c}{\beta^c} + \epsilon(t'))(\sum_{j\propto i} a_{ij}\beta p_j(t) + p_i(t)) = \\ &= (\frac{1}{1+\beta\lambda_{1_A}} - (\zeta - \epsilon(t')))(\sum_{j\propto i} a_{ij}\beta p_j(t) + p_i(t)) \end{aligned}$$

or in matrix form:

$$\mathbf{p}(t+1) < (\frac{1}{1+\beta\lambda_{1_A}} - (\zeta - \epsilon(t')))(\beta\mathbf{A} + \mathbf{I})\mathbf{p}(t) = \mathbf{D}(t')\mathbf{p}(t),$$

with $\mathbf{D}(t') = (\frac{1}{\beta\lambda_{1_A}} - (\zeta - \epsilon(t')))(\beta\mathbf{A} + \mathbf{I})$. Considering that $\mathbf{D}(t')$ is non-negative and, from the problem statement, irreducible, the Perron-Frobenius Theorem applies. Adequately, for its largest eigenvalue $\lambda_{1_\mathbf{D}}(t')$ one obtains:

$$\begin{aligned} \lambda_{1_\mathbf{D}}(t') &= (\frac{1}{1+\beta\lambda_{1_A}} - (\zeta - \epsilon(t')))(\beta\lambda_{1_A} + 1) = \\ &= 1 - (\zeta - \epsilon(t'))(\beta\lambda_{1_A} + 1) < 1 \end{aligned} \tag{22}$$

Therefore:

$$lim_{s\to\infty, t>t'}\mathbf{p}(t+s) < lim_{s\to\infty, t>t'}D(t')^s\mathbf{p}(t) = \mathbf{0}_{N\times 1}.$$

Since $p_i(t) > 0$, then for $t \to \infty$, $p_i(t)$ is bounded by zero both from beneath and above, adequately converges toward zero as time elapses. The proof is completed. □

In practical implementation, one may (should) consider $\beta^c = 1$. Assume that the observed network is either static (non-expandable network), or (more correctly) that an conservative estimation of the maximal value of $\lambda_{1_\mathbf{A}}$ can be made on a long term. It is reasonable to assume that when preparing the countermeasures one may estimate the infective characteristics of the virus (β). Then, from (21), δ^c may be calculated as:

$$\delta^c < \frac{1}{1+\beta\lambda_{1_A}}$$

The examples gives a good insight of the principles and engineering validity of the elaborated concept. Each node i, at an instance in time t, will contact randomly chosen neighbor j from the network G_1, and if necessary and available, acquire the viral countermeasures. Then, it will delete the acquired countermeasures with probability δ^c at each time step. The parameter δ^c will be stated in the countermeasure definition file.

To support the analytical findings, we test the suggested method on the giant component of the Enron e-mail network. It comprises of $N = 33696$ nodes, $L = 180811$ undirected links, and the largest eigenvalue of the graphs adjacency matrix $\lambda_{1_A} = 118.418$. The Enron network was downloaded from Stanford Large Network Dataset Collection [36]. We built an additional virtual network, that in our example is of Barabási - Albert type with $m_0 = 5$, $m = d_{min} = 4$.

Following scenario is observed: a SI type of an infection occurs on the Enron network at $t = 0$ and becomes an epidemic. At $t = 200$ a set of prepared countermeasures are released by a randomly chosen node and disseminated trough the virtual network with $\beta^c = 1$. We monitor both the dynamics of the the probabilistic system (19,20) and the results of the stochastic simulation that mimics the reality, by observing the number of nodes that are infected and the number of nodes that possess the countermeasures at an instance in time. The results of the analysis are presented in Fig. 5.

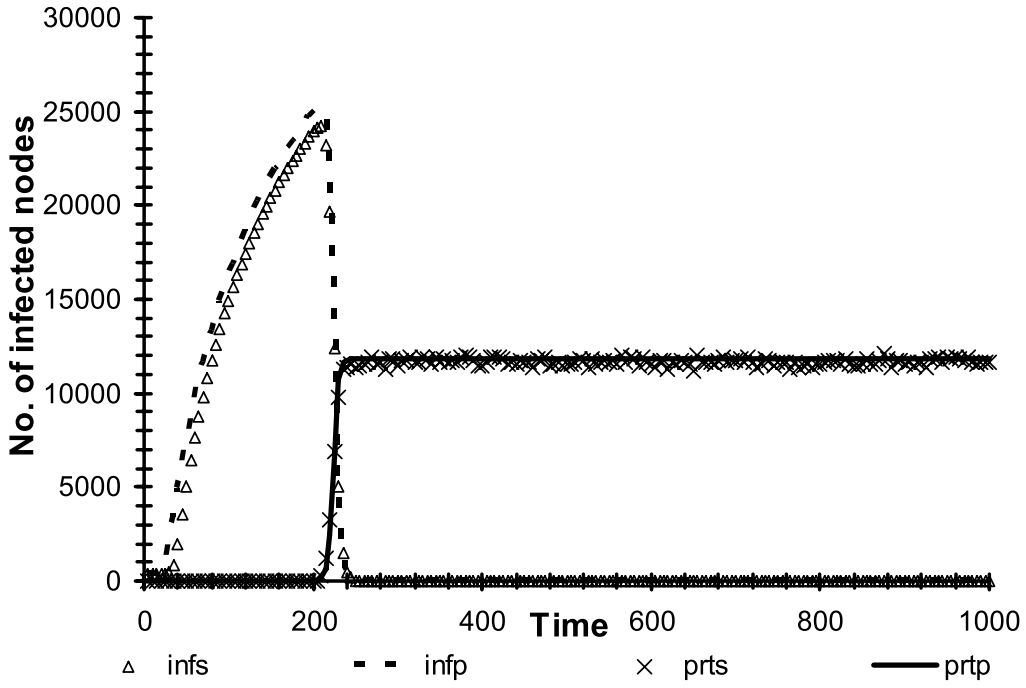

Figure 5: Process of infection and countermeasures spreading on the Enron and the virtual network, with $\beta = 0.004$, $\delta^c = 0.65$; infs-infection status (stochastic simulation), infp-infection probability (19), prts-protection status, prtp-protection probability (20).

Fig. 5 shows the following behavior: In the first 200 time-steps there is nearly quadratic rise in the number of infected nodes; once the viral countermeasures are deployed at time t = 200 the number of infected nodes in the network drops almost immediately to zero.

Commercial security packages today are based on distribution of viral countermeasures (virus definitions) from a centralized database to all the

machines in a network, where they are stored and permanently kept. Analysis shows that countermeasures may freely float through the network, with no permanent presence on each computer, and still be able to eradicate an infection.

6. Conclusion

In this paper a SIS type of spreading processes, characterized by acquisition exclusivity is studied. It is shown that the process, under the imposed restrictions and assumptions is analytically solvable in respect to the status probabilities of nodes. The obtained results indicated that this type of set-up is topology independent. We argue that this could be used as a basis for development of practical applications for controlled sharing of certain contents in complex network. In that sense we give an example of a potential application, that based on principles studied in this paper, is used for controlled sharing of viral countermeasures. In addition, acquisition exclusivity, in a form of an alternative contact dynamics, is suggested as a method for malware removal from technological networks.

Acknowledgement

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