SIS Contagion Avoidance on a Network Growing by Preferential Attachment *

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Abstract

The economic and convenience benefits of interconnectivity drive the current explosive growth in networked systems. However, as recent catastrophic contagious failures in numerous large-scale networked infrastructures have demonstrated, interconnectivity is also inherently associated with various risks, including the risk of undesirable contagions. This paper reports on a work-in-progress on network formation subject to Susceptible-Infected-Susceptible (SIS) contagion risk mitigation. As opposed to existing research, we concentrate on network growth. Using a generalized form

of preferential attachment, we consider evolving networks where each incoming node to the network combines a preference for connecting to high degree nodes with an aversion for contagion risk. Our initial simulation results indicate that contagion risk aversion significantly alters the topology and contagion propagation for an emerging network. We also discuss the computational aspects of our simulation and our future plans to extend this model.

1 Introduction

The economic and convenience benefits of interconnectivity drive the current explosive emergence and growth of networked systems. However, interconnectivity is inherently associated with various risks, including the risk of an undesirable contagion [1]. Network formation by strategic agents/nodes is affected by numerous competing incentives, including the incentives to directly connect to the "central" nodes and also to reduce risks of undesirable contagions. The effect of these two competing incentives on the network evolving by rewiring has been investigated in recent papers on adaptive and active networks [2, 3]. However, due to economic or contractual constraints, e.g., in the physical or financial networked infrastructure, breaking old and establishing new links may be difficult. In such cases, the effect of competing incentives on network growth plays a primary role in understanding of the structure and function of the emerging networked systems.

While in our future work we plan to investigate effect of competing incentives

on networks simultaneously evolving by growing and rewiring, in this short paper we concentrate on growing networks subjected to an undesirable Susceptible-Infected-Susceptible (SIS) infection. We model multiple competing incentives by utility function and assume that network growth follows logit response dynamics [4]. This model describes bounded rational agents who attempt to maximize their utility with controlled level of rationality. This approach overcomes interrelated limitations of oversimplification and intractability of the game-theoretic approaches [5]. The tractability of this approach is due to the fact that logit response dynamics take the form of generalized preferential attachment [6], which allows one to leverage an extensive body of results on preferential attachment models for growing networks. Note that the same is true for rewriting models for networks of fixed size.

This paper is organized as follows. Section 2 proposes a model for network formation by contagion averse agents. Section 3 describes the simulation setup and computational challenges. Section 4 discusses the initial simulation results. Finally, Section 5 briefly summarizes and outlines directions for future research.

2 Model and Problem Statement

Consider a growing network, where nodes arriving at discrete moments $t = 1, 2, \ldots$ make decisions to connect to an existing node i with degree d in order to attempt

to maximize the utility function

$$u_d(\delta_i) = (1 - h\delta_i) \ln \varphi_d, \tag{1}$$

where $\delta_i = 1$ if the node i is infected and $\delta_i = 0$ otherwise, and where $\varphi_d \geq 1$ is an increasing function of $d = 1, 2, \ldots$. The parameter $h \geq 0$ characterizes the trade-off between the incentives for connectivity and for contagion risk avoidance. If $h > 1$, then the decision of an arriving node to connect to an existing infected node of degree d results in not only the loss of the utility of connectivity, but also in an additional loss $(h-1) \ln \varphi_d$. Thus connecting to a high degree node has a high-risk but also a high-potential for reward to the arriving node.

Following the conventional approach, we model bounded rationality by assuming that an arriving node immediately develops a fixed number $m \geq 1$ of connections to a subset of m existing nodes. All the nodes in this subset are selected independently from each other using logit probabilities [4]

$$g_i \sim \exp[T^{-1} u_{d_i}(\delta_i)], \tag{2}$$

where d_i is the degree of i, and the "temperature" $T > 0$ characterizes the agents' rationality. A value $T \to 0$ corresponds to complete rationality, while a value $T \to \infty$ corresponds to complete randomness. For the utility (1), the logit probabilities are:

$$g_i \sim (\varphi_{d_i})^{\frac{1-h\delta_i}{T}}. \tag{3}$$

Further in the paper we consider a specific function, $\varphi_d = d^{\gamma}$, where $\gamma > 0$, when attachment probabilities (3) take the following form:

$$g_i \sim d_i^{(1-h\delta_i)\alpha}, \tag{4}$$

where $\alpha = \gamma/T$. Since we are interested in the effect of infection averseness on network formation, we contrast two extreme cases: the case of infection risk indifference for the agents/nodes ($h = 0$), and the case of a very high infection risk aversion ($h \gg 1$).

From Krapivsky, Redner, and Leyvraz [6], if $h = 0$ then the resulting structure of a growing network depends on whether the parameter α is smaller than, greater than, or equal to unity. If $\alpha < 1$, this mechanism results in a stretched exponential node degree distribution while when $\alpha > 1$, then one "central node" is connected to nearly every other node in the network. When $\alpha > 2$, this "winner takes all" phenomenon is so extreme that the number of connections between "non-central" nodes is finite even in an infinite network. Finally, if $\alpha = 1$, then a power-law node degree distribution, $N_d \sim d^{-\nu}$, emerges. In this case, the parameter ν, where $2 < \nu < \infty$, controls the finer details of attachment probability.

A fundamental and still to a large degree open question is why numerous existing networked infrastructures are characterized by power law node degree distribution. In the context of the above generalized preferential attachment mechanism, the question is identifying additional mechanisms responsible for the tuning parameter ν, ensuring the specific value of the preference parameter $\alpha = 1$. We

suggest that one of such mechanisms may be contagion risk avoidance by systemic risk averse strategic agents. Indeed, node infection probability among other things depends on node degree, and thus contagion risk avoidance affects the attachment probability.

We simulate network growing by generalized preferential attachment and subjected to SIS infection. Once node n becomes infected, it spreads infection to each of its neighboring nodes j at fixed rate $\lambda > 0$. Node recovery time is distributed exponentially with the same recovery rate μ for all nodes. We define the effective infection rate ρ as the ratio between the infection and recovery rates,

$$\rho \stackrel{def}{=} \frac{\lambda}{\mu}. \tag{5}$$

Alós-Ferrer and Netzer [4] showed that an SIS model is infection free if and only if

$$\rho \leq \frac{1}{\Gamma}, \tag{6}$$

where Γ is the Perron-Frobenius (P-F) eigenvalue of the adjacency matrix $A = \left(X_{ij}\right)_{i,j=1}^{N}$, where $X_{ij} = 1$ if nodes i and j are connected by an edge, and $X_{ij} = 0$ otherwise. Thus, the value $\rho = 1/\Gamma$ defines the threshold boundary for the infection-free region.

Ferreira, Castellano, and Pastor-Satorras [7] showed that an SIS contagion results in higher infection probabilities for higher degree nodes, at least in a case of random uncorrelated network. Thus, one may expect that SIS contagion aversion of strategic agents forming network growing by generalized preferential attach-

ment *suppresses* the "winner takes all" phenomenon for $\alpha > 1$. Our goal in this paper is to confirm and quantify this conjecture by simulations.

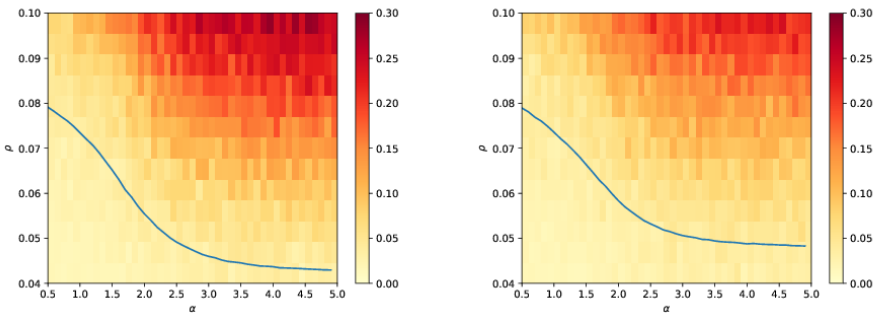
3 Simulation Setup and Challenges

In order to examine the effects of SIS contagion aversion, we simulate the growth of a number of networks across varying values of α and ρ. For our simulations, we fix the recovery rate at $\mu = 0.1$ and vary the infection rate λ by varying ρ.

We examine two cases: one where we avoid connecting to any node that is currently infected, in other words, preferential attachment with infection avoidance (IA) (where the value $h \gg 1$ for equation (4)). For the second case, we ignore the infection state of a node when choosing where to connect ($h = 0$ for equation (4)). This case is simple generalized preferential attachment (GPA).

In constructing the graphs used in our simulations, in each case we began with a path graph of 5 nodes. Nodes were then sequentially added to the graph using the generalized form of preferential attachment. In our simulations, we fixed the number of attached edges to $m = 5$. The associated α for each simulation run is reflected in the attachment weights ω for selecting edges as a new node is introduced.

The probability that an introduced node forms an edge with an existing node i



(a) Without infection avoidance (GPA) (b) With infection avoidance (IA)

Figure 1: This figure shows the average portion of infected nodes across a number of α and ρ values.

is proportional to its weight ω_i. The weight ω_i for the node i is given by

$$\omega_i = \begin{cases} d_i^{\alpha} & \text{for pure preferential attachment (GPA)} \\ (1-\delta_i)\cdot d_i^{\alpha} & \text{for infection avoidance (IA).} \end{cases} \tag{7}$$

In order to attach the m edges of a new node, we select m unique nodes using these weights to define the probability distribution.

Between adding new nodes, we allow the SIS infection to reach a steady-state. In order to try to avoid correlation between infected nodes when we add new nodes, we allow the infection to run for 20 rounds after each node addition. Our analysis indicated that this value allowed the infection to reach a steady state before adding a new node.

For simulation runs where the value of ρ falls into the infection-free zone of (6), we risk the infection dying out after each SIS infection round. In order to prevent our infection avoidance simulations from degenerating into the simple

case of preferential attachment, we maintain the infection state of the previous round. If at any time step the infection dies out of the network, we then simply backtrack to the last time step and rerun the simulation step to assure that the infection continues.

Each data point in our simulations is the average of 100 samples for a 100 node networks with the given α and ρ. Our simulation code is written in C++ using GSL [8] for the numerical computation library.

4 Initial Simulation Results

Localization of the principal eigenvector controls the spread of infection in a network [9, 10]. A network with some level of localization tends to maintain an infection over a subset of the nodes. Networks created using GPA show localization for their hub nodes. We are interested in whether IA networks also show localization, and how IA affects the spread of infection in these networks.

As expected, infection avoidance has a profound effect on the topology and infection sustainability for an evolving network. In Figure 1, we see the how infection is dampened when using infection avoidance. The intensity of the average infection is shown from 0 % to 30 % of the nodes for the ranges of α and ρ. Figure 1a shows simulation results while using general preferential attachment for growing the networks, while Figure 1b shows the results for networks grown using infection avoidance. The blue lines represents the boundary of the infection-free region given by $\rho = 1/\Gamma$. Each point in this line is computed by averaging the

values for $1/\Gamma$ across all networks grown with a given α.

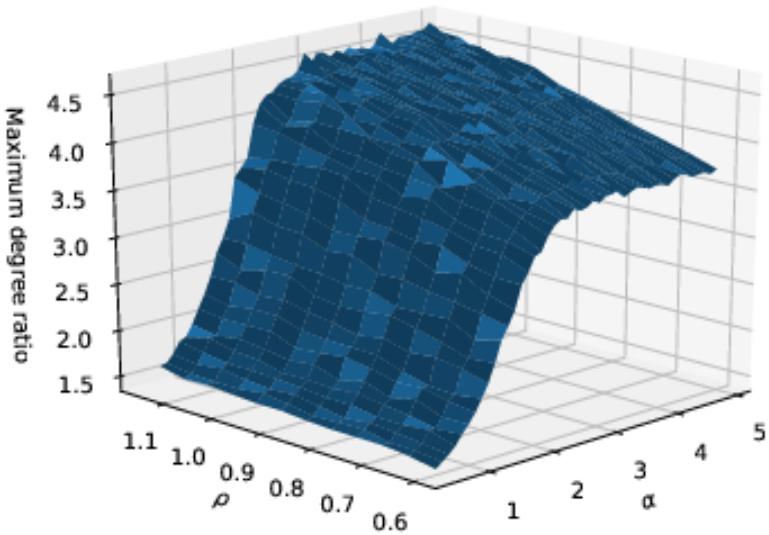


Figure 2: Ratio of maximum degrees between the GPA and IA graphs.

In comparing the upper right hand corners of the two graphs, the percentage of infected nodes in the resulting networks significantly decreases when using infection avoidance. We are also interested in the effect on the infection-free region, between using and not using infection avoidance growth. In comparing the two figures, we see that the threshold boundaries are raised in networks grown using infection avoidance. We note that the strongest effect on the resulting graphs occurs for α when $1 \leq \alpha \leq 2.5$. In the region $\alpha > 2.5$, localization begins to affect both regimes leveling out the threshold boundary. For the region $\alpha \leq 2.5$,

infection avoidance slows the descent of the threshold boundary.

Since localization for the eigenvector of Γ depends on the maximum degree in a network, we plotted how the maximum degree is affected in Figure 2. In this figure, we show the ratio between the average of the maximum degrees from GPA networks over the IA networks. This ratio shows that in GPA graphs, the maximum degree grows much faster than in IA graphs when $\alpha \geq 1$. We also see that the strongest dependency for the maximum degree ratio is on the value of α with a smaller effect from the value of ρ. Put together, these two figures imply that IA network growth limits contagion spread by limiting the amount of localization that occurs.

To test this idea we further examine the amount of localization that arises in IA networks by computing the inverse participation ratio (IPR) [9, 10]

$$IPR(\Gamma) = \sum_i \mathbf{f}_i^4(\Gamma), \tag{8}$$

where $\mathbf{f}(\Gamma)$ represents the eigenvector associated with the eigenvalue Γ. The IPR for a set of networks is usually studied as a function of the size of a network n by fitting it to a power-law distribution,

$$IPR(\Gamma) \sim n^{-\psi}. \tag{9}$$

Examining the slope ψ for a family of graphs helps us to understand the amount of localization occurring in these graphs. If the slope ψ converges to 1, then no localization is occurring for the networks, else if the slope converges to

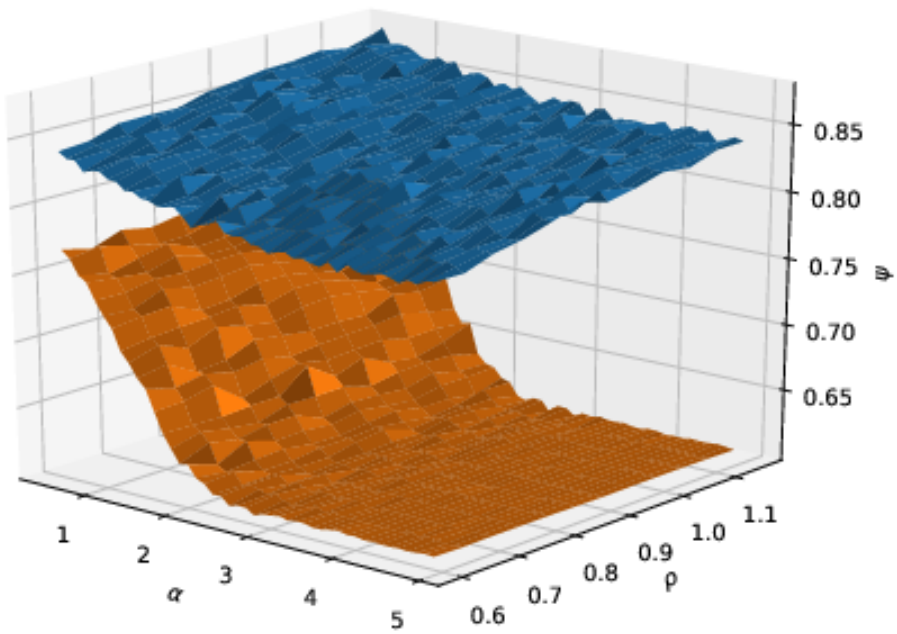


Figure 3: Slope ψ of inverse participation ratio between IA (blue surface) and GPA (orange surface) graphs.

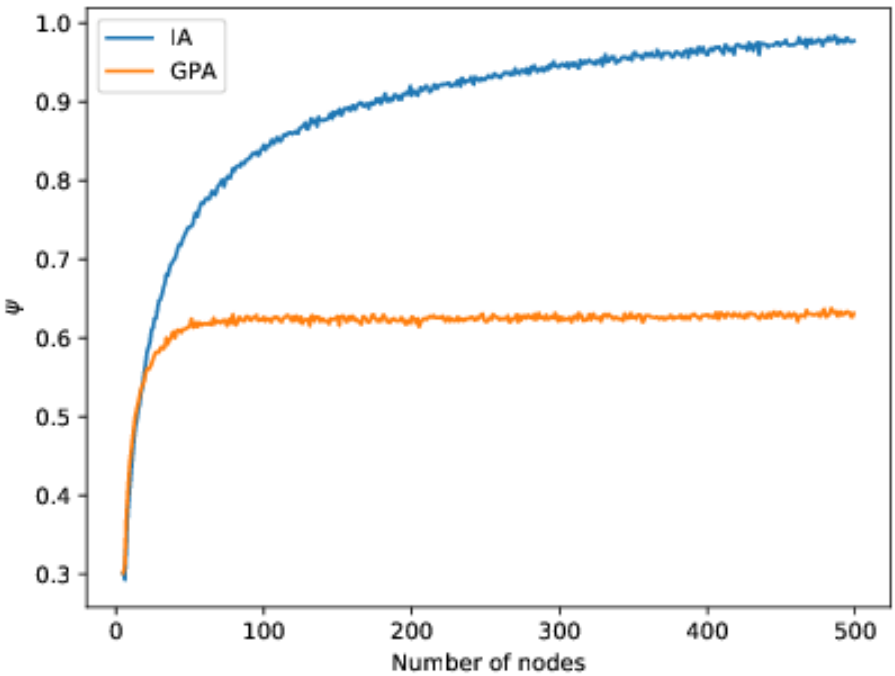


Figure 4: Slope ψ of the inverse participation ratio between IA and GPA graphs. Here the values $\alpha = 2$ and $\rho = 1$ are fixed.

value less than 1, then some localization occurs over a subset of the nodes.

In Figure 3, we see the that slope ψ is much higher for IA graphs than for GPA graphs. This figure shows that less localization is occurring in the IA graphs over all values of α and ρ. In Figure 4, we see a stronger indication that IA graphs do not show localization as they grow. Here we see that the value $\psi \to 1$, implying that IA graphs do not demonstrate localization as the networks grow. Since $\psi \approx .6$ the GPA graphs do show some degree of localization.

5 Conclusion and Future Research

This paper reports on work-in-progress on the effect of SIS infection avoidance on a network growing by preferential attachment [3]. Our simulation-based investigation characterizes competing effects of the preferential attachment parameter α quantifying the propensity of an incoming node to connect to a high degree existing node on the one hand and effective SIS infection rate ρ on the other hand. Our initial simulation results suggest a possible explanation of the consistency of the structure of observed real networks with preferential attachment model.

The problem is that while typically observed networks are characterized by a power law node degree distribution, the preferential attachment model produces this node degree distribution only for the specific "preferential attachment parameter" $\alpha = 1$. For the example of SIS infection avoidance, we suggest a possible explanation for this phenomenon: even when the parameter $\alpha \neq 1$, preference for connectivity to high degree nodes combined with other incentives produces

preferential attachment model with "effective preferential attachment parameter", $\alpha^{eff} \approx 1$. Indeed, infection avoidance counteracts the incentive to connect to a high degree node since high degree nodes are more likely to be infected.

Currently we are attempting to verify our conjecture that for $\alpha > 1$, infection avoidance keeps the network on the verge of eigenvector localization regime [9]. This conjecture may have important practical implications for infection propagation and mitigation on real networks since eigenvector localization implies persistent infection presence in network [10]. Our future plans include enhancing our model by allowing (a) connectivity decisions based not only on the current infected/non-infected node status, but also on the perceived likelihood of being infected, (b) rewiring of the already existing connections, (c) node investments in the infection risk mitigation, and (d) other types of contagion, e.g., threshold-based contagion.

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