Network Formation by Contagion Averse Agents: Modeling Bounded Rationality with Logit Learning

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Abstract—**Economic and convenience benefits of interconnectivity drive current explosive emergence and growth of networked systems. However, as recent catastrophic contagious failures in numerous large scale networked infrastructures demonstrated, these benefits of interconnectivity are inherently associated with various risks, including risk of undesirable contagion. Current research on network formation by contagion risk averse agents, which analyzes Nash or some other game-theoretic equilibrium notion of the corresponding game, suffers from interrelated problems of intractability and oversimplification. We argue that these problems can be alleviated with dynamic view, which assumes logit responses by strategic agents with utilities quantifying multiple competing incentives. While this approach naturally incorporates practically critical assumption of bounded rationality, it also allows for leveraging a vast body of results on network formation, e.g., preferential attachment in growing networks.**

Keywords-Network formation; contagion risk averseness; bounded rationality; preferential attachment; SIS contagion.

I. INTRODUCTION

Modeling network formation by strategic agents/nodes having dual incentives for interconnectivity and contagion risk avoidance is a highly relevant and challenging problem. The relevancy is due to numerous recent systemic failures in various networked infrastructures. The challenges are due to non-trivial interactions between interconnectivity and contagion: depending on network density and contagion mechanism, interconnectivity may either alleviates or suppress contagion [1]. We follow a conventional modeling of rational agents with multiple and probably competing incentives by the corresponding utility function. However, instead of identifying the result of agents' behavior with the corresponding game-theoretic equilibrium, e.g., Nash equilibrium, we propose learning/evolutionary dynamic modeling.

Our contention is that this approach overcomes interrelated limitations of oversimplification and intractability of the conventional approach. We illustrate this thesis by demonstrating tractability of incorporating practically important aspect of bounded rationality into agent behavior. Considering growing network, we follow conventional modelling of boundedly rational agents by logit response dynamics [2]. Tractability of this approach is due to the logit response dynamics takes form of generalized preferential attachment [3], which allows one to leverage an extensive body results on preferential attachment models of growing models. Note that the same is true for rewriting models for networks of fixed size. Section II proposes a model for network formation by contagion averse agents, and section III briefly discusses the interplay between the contagion and network evolution.

II. MODEL

Consider a growing network, where nodes arriving at discreate moments $t = 1, 2, ..$ make decision on connecting to an existing node n of degree d in attempt to maximize utility

$$u_d(\delta_n) = (1 - h\delta_n)\ln\varphi_d, \quad (1)$$

where $\delta_n = 1$ if node n is infected and $\delta_n = 0$ otherwise, $\varphi_d \geq 1$ is an increasing function of $d = 1, 2, ..$, and parameter $h > 1$ characterizes tradeoff between incentive for connectivity and contagion avoidance. Due to our assumption $h > 1$, decision of an arriving node to connect to an existing infected node of degree d results not only in loss of the utility of connectivity but also in additional loss $(h-1)\ln\varphi_d$. Thus, connectivity to a high degree node is a high risk high potential reward for an arriving node.

For brevity we assume time scale separation: contagion develops much faster than new nodes arrive, and thus network formation is driven by the averaged utility (1): $u_d(\bar{\delta}_n)$, where $\bar{\delta}_n = E[\delta_n]$ is node n probability of being infected, given the network. We also assume that prior to linking to some node n, an arriving node is aware of the infection expectation $\bar{\delta}_n$, but not of the actual infection status δ_n. Following conventional approach, we model bounded rationality by assuming that an arriving node immediately develops a fixed number $m \geq 1$ of connections to already existing nodes $n_1, .., n_M$. All these nodes $n = n_1, .., n_m$ are selected independently from each other with logit probabilities [2]: $\alpha_n \sim \exp[T^{-1}u_{d_n}(\bar{\delta}_n)]$, where d_n is node n degree, and "temperature" $T > 0$ characterizes agents' rationality: $T \downarrow 0$ corresponds to

complete rationality and $T \uparrow \infty$ corresponds to complete randomness.

In the spirit of logit response, we assume that if all m initial links resulted in connection to uninfected nodes $\delta_n = 0$, $n = n_1, .., n_m$, with probability α_+ one more connection is established to an existing node n_{m+1} selected with probabilities (2). Otherwise, i.e., if $\prod_{n=n_1,..,n_m}(1-\delta_n) = 1$, the process of developing new connections for the arriving node stops. If $\delta_{n_{m+1}} = 0$, then with probability α_+ one more connection is established to an existing node n_{m+2} selected with probabilities (2). Otherwise, i.e., $\delta_{n_{m+1}} = 1$, the process of developing new connections for this arriving node terminates. This process continues until the number of connections reaches its maximum for a given arriving node $M \geq m$. Since we are interested in asymptotic behavior as number of nodes $N \to \infty$, we assume $N >> M$.

III. Some Implications

Assuming $\varphi_d = d^\gamma$, $\gamma > 0$ in (1), we obtain the following logit attachment probabilities to a node of degree i:

$$\alpha_i = N_i(1-h\bar{\delta}_i)i^{\gamma/T} \Big/ \sum_{j\geq 1} N_j(1-h\bar{\delta}_j)j^{\gamma/T}, \qquad (2)$$

where N_d is the number of existing nodes of degree d. In a case of contagion indifferent agents $h = 0$, attachment probabilities (2) produce conventional preferential attachment model, which favors connectivity to high degree nodes and promotes "rich get richer" phenomenon [3]. As parameter $h > 0$ increases, attachment probabilities (2) also promote low risk of infection. Network evolution, driven by attachment probabilities (2), depends on the specific contagion mechanism.

To illustrate the proposed model, consider growing network vulnerable to Susceptible-Infected-Susceptible (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 average τ_i, where i is node degree. It is known [4] that SIS model is infection free if $\lambda \leq 1/\Gamma$, and has finite portion of nodes persistently infected otherwise, where Γ is the Perron-Frobenius (P-F) eigenvalue of matrix $\mathrm{T} = (\tau_n \chi_{nk})_{n,k=1}^N$, and $\chi_{nk} = 1$ if nodes n and k are connected by a link, and $\chi_{nk} = 0$ otherwise.

Figure 1 shows phase diagram of joint network evolution and infection spreading in variables (d_{ave}, ω), where d_{ave} is the average node degree, and ω is the average portion of infected nodes. In "fast time scale," SIS infection reaches "metastable equilibrium" 0ABCDEF. Then in "slow time scale" network evolves along curve 0ABCDEF, finally reaching equilibrium (d^*_{ave}, ω^*) at point C.

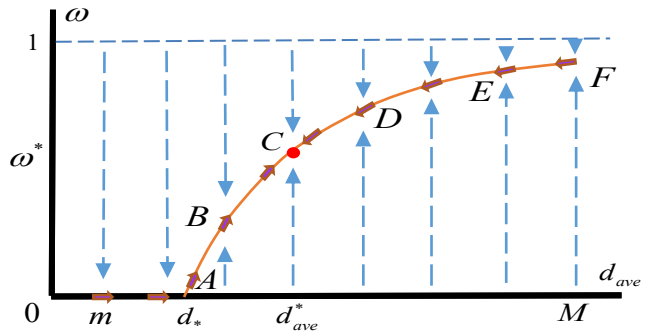

Figure 1. Phase diagram: network density vs. contagion risk

Figure 2 shows result of the network evolution in "slow time scale" vs. infection propagation rate λ.

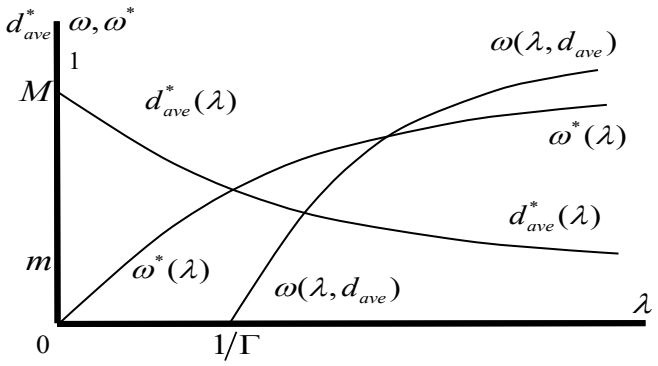

Figure 2. Network density vs. contagion risk tradeoff

Curves $\omega^*(\lambda)$ and $d^*_{ave}(\lambda)$ demonstrate tradeoff between systemic risk of infection on the one hand and network density on the other hand. Increase in the infection propagation rate λ, causes decrease in the equilibrium average node degree $d^*_{ave}(\lambda)$ and increase in the equilibrium average portion of infected nodes $\omega^*(\lambda)$. Note that $\omega^*(\lambda)$ grows slower than average portion of infected nodes in a fixed network $\omega(\lambda, d_{ave})$ due to decrease in the equilibrium density $d^*_{ave}(\lambda)$ with increase in λ. Finally note that models with strategic node aware of actual infection status of existing nodes, may produce more complex evolutionary dynamics.

References

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