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Adaptive Network Dynamics - Modeling and Control of Time-Dependent Social Contacts

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***Abstract*—Real networks consisting of social contacts do not possess static connections. That is, social connections may be time dependent due to a variety of individual behavioral decisions based on current network connections. Examples of adaptive networks occur in epidemics, where information about infectious individuals may change the rewiring of healthy people, or in the recruitment of individuals to a cause or fad, where rewiring may optimize recruitment of susceptible individuals. In this paper, we will review some of the dynamical properties of adaptive networks, and show how they predict novel phenomena as well as yield insight into new controls. The applications will be control of epidemic outbreaks and terrorist recruitment modeling.**

Keywords: Adaptive networks, Terrorist recruitment, Social Networks, Epidemics

I. Introduction

With the invention of high speed computing, data analysis on large spatial scales coupled with models for individual social behavior have greatly refined the understanding of the dynamics of populations. Models currently used are based on various population decompositions which describe social interactions of groups and individuals. Examples come from information spreading dynamics, epidemiology, and terrorist cell analysis [1]–[5].

Much previous work on social dynamics assumed homogeneous populations, where real social structure was lacking. Most of these models were compartmental and were similar to mean field models of stochastic simulations. Modeling of social interaction is done by mass action, which effectively allows all individuals to contact all others. It is most evident in the modeling of epidemics, where infection spread in a population arises from direct contact between healthy and sick individuals. Another mass action modeling class of interest to defense is that of recruitment of susceptible individuals by the terrorist cell networks.

Population models have been improved by incorporating demographic information, such as age structure and gender, as well as spatial characteristics, such as geography. These models have included a range of detail, from the level of the individual to coarse graining spatial regions by coupling various local patches. Typically, the connections among individuals or patches are modeled as networks, and the epidemics are dynamically propagated on these networks.

One current and future trend of network modeling is to consider adaptive behavior, or social response in the population to information about a current or future threat [6]–[9]. Here we briefly discuss some of the adaptive network models used in infectious disease and terrorist recruitment modeling, and how individual social adaptation may change the dynamics of the networks, which in turn alters the progression of disease or recruitment.

II. Epidemics on Adaptive Networks

In the presence of a natural or man-made biological threat, it is highly probable that people will change their behavior. Such an assumption holds if the population of susceptible individuals knows the existence of infectious individuals and adapts their behavior to avoid contact with disease [6]. Methods such as increased hand washing, wearing of a mask when contagious, and self-imposed isolation all are examples of adaptive behavior in the presence of a disease. The implication is that not only does the disease status of individuals change in time, but so do the contacts. In fact, the change in disease status causes a change in contact behavior.

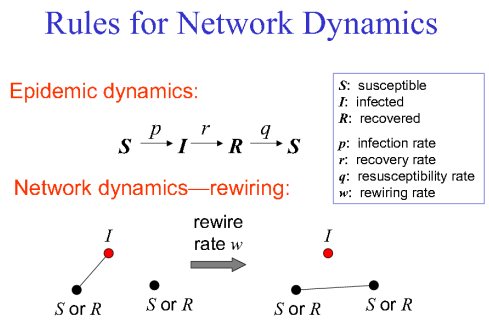


Figure 1. Adaptive network dynamical rules for an SIRS model.

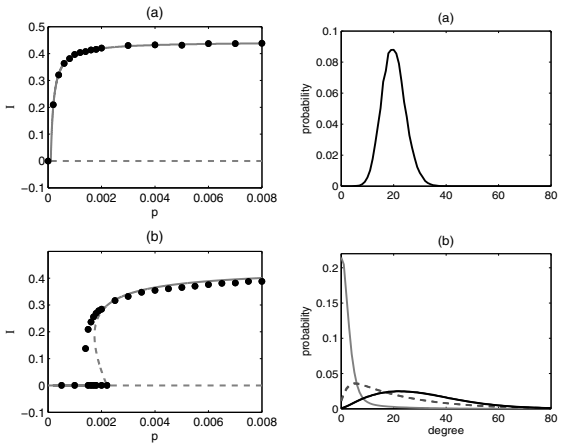


Figure 2. The effect of rewiring in an adaptive network using avoidance behavior. The left panels show the fraction of infectious individuals I in a finite population as a function of the infection rate p. (a) Fixed contact network (no rewiring). (b) Rewiring is turned on, causing the emergence of bistable behavior. Solid (dashed) lines are stable (unstable) mean field predictions, and dots correspond to averaged Monte Carlo runs. The right panels show the degree distribution (a) without rewiring and (b) with rewiring. Light grey denotes infectious, black susceptible, and dashed recovered individual fractions. Reprinted from [7].

We do note that many of the agent based and multi-scale models take into account distributions of human motion, thereby causing contacts that are time dependent and heterogeneous. In contrast to the models of a static network or models with externally applied changes in structure, a new class of models based on endemic populations on an *adaptive* network has been recently introduced [6]. For a recent review, see [3]. Changes to the network structure are made in response to the epidemic spread and in turn affect future spreading of the epidemic. Here, a new parameter is one that describes the rewiring rate of the network, which governs changes in the fraction of susceptible (S) to infective (I) links. The network alters dynamically when there are contacts between S and I, and social pressures (the desire to avoid illness) rewire the contacts, replacing them with contacts between S and S. Infections are reduced due to isolation, and a new phenomenon occurs: for appropriate choices of parameters, bistability between the disease free equilibrium and endemic state has been observed. This is in contrast to static networks in a large population, where there is typically only a single attracting endemic or disease free state.

When modeling adaptive networks, one needs to describe the disease status of the individual as well as contact behavior for each individual. Both nodes (people) and links (contacts between people) must be modeled as functions of time. Suppose we have S, I, and R states available for each node. If a node in an infected state is linked to a susceptible node, the avoidance behavior says the S node should rewire by changing its link to a non-infectious node. Since the model is a finite population with random transitions between contacts and states, we assume there is a rewiring rate at which the new contacts are made probabilistically [7]. The rules of evolution for a susceptible-infectious-recovered-susceptible (SIRS) model with adaptation are summarized in Fig. 1, following [7], [9].

Comparing the model with adaptive social behavior to the fixed contact models, the results are quite dramatic. First, new attractors leading to bistability appear for small rewiring rates [6], [7], [10], [11]. An example of the behavior is shown in Fig. 2. In addition, the size of the fluctuations increases, which may lead to higher probabilities of disease extinction [7].

A complication is the possibility that individuals may not have full knowledge of their own and others' infection status. The presence of asymptomatic infectious individuals can occasionally lead to disease avoidance behavior being counterproductive [11]. Co-spreading of an epidemic and awareness of the epidemic has been considered, where the network structure was held fixed but the connection strength reduced for nodes that were aware of the need to protect themselves from the disease [12]. This behavioral response increased the epidemic threshold and was most effective when the awareness was transmitted on the same contact network as the infection.

III. Terrorist recruitment modeling

Background

In recent years our society has witnessed a growing concern over existence and spread of terrorist networks. The ability of a government to efficiently counteract this phenomenon strongly depends on understanding the structure as well as dynamical properties of the terrorist networks. Many papers discuss terrorist networks as optimal structures that balance communication efficiency with maintaining the secrecy/security of the networks (e.g, [13]–[15]), while ignoring the dynamical processes that take place on such networks. Another class of work [16], [17] models the dynamics of terrorist recruitment within a well-mixed population, where the existence of network structure is excluded from the discussion, and therefore any network changes that may arise as a result of node dynamics are ignored. Models that include both terrorism recruitment dynamics and network structure are, to our knowledge, extremely rare [18], and no such model has considered adaptive changes of the social network structure.

We present a model that captures the time evolution of the underlying network due to the interaction dynamics of the individual network members. In our model, we study a society (represented by a network) where some of the members of the society belong to a terrorist organization. We postulate a simple procedure by which a regular citizen may be recruited into a terrorist organization. In order to improve their ability to recruit new members, the terrorists follow a simple algorithm for how they change their interactions with the rest of the society.

As an application of our new adaptive networks tools, we are developing a model that captures the time evolution of a social network as terrorists recruit individuals from a pool that is susceptible to radical ideas. Adaptive changes in the network structure may occur in a variety of ways. For example, newly recruited individuals may change their social interactions as a result of their changing status, terrorists may adjust their social behavior to improve recruiting, or non-radical individuals may attempt to avoid extremists. The resulting network structure, and its susceptibility to control measures, are likely to depend on the form of network adaptation taking place.

Recruitment Model

We model the evolution of a social network with members belonging to one of three classes: *non-susceptible* (N), *susceptible* (S), and *terrorist* (T), following the categories used in [16] in the absence of network structure. By treating radical ideas as a phenomenon that can spread person-to-person along social contacts, we build on previous studies of epidemic spread in networks.

The non-susceptible class corresponds to the portion of the population whose members do not participate in any terrorist activity, and whose mentality is sufficiently far from radicalization that they cannot instantaneously become terrorists. The class of terrorists consists of people involved in terrorist activity, and it is assumed that once a person joins this class s/he remains a member until death. We assume that in order for a regular, non-susceptible person to become a terrorist, s/he must first become susceptible to terrorist recruitment. Non-susceptibles may spontaneously become susceptible. The people in the susceptible class are not terrorists, but there is a probability that they will join that class. The rate at which susceptibles become terrorists depends on their contacts with terrorists who can recruit them.

We study the above society model as a network consisting of M nodes (on average), with some initial connectivity. Each node in the network corresponds to a person from one of the classes N, S, T. People in each class can change their affiliation according to the following transition rules:

- N can become S at a rate λ_1
- S can go back to being N at a rate λ_2
- S can become T at a rate proportional to the number of contacts that exist between the S and T class, with proportionality constant γ
- Birth process replenishes numbers of N a rate μ
- Death rate of T, $\delta_1 + \delta_2$, is potentially greater than the death rate in N and S classes, δ_1
- The rewiring rate w allows T-nodes to rewire their connections from N-nodes to S-nodes (Terrorists rewire their connections to increase their connectivity with the susceptible portion of the population, improving their chances to recruit.)

To take into account the dynamic nature of the real social networks, we allow the nodes to adaptively change their connectivity with the rest of the network. Here, we allow terrorists to rewire their connections to increase their connectivity with the susceptible population, or equivalently non-susceptible nodes avoid contact with terrorists and the terrorists are forced to find other contacts. Adaptive behavior may depend on global properties of the system, such as the level of terrorism in the system or media coverage of terrorist attacks.

Using a mean-field approach, we study the statistical properties of the network as it evolves in time. Let the functions $N_{\mathrm{N}}(t)$, $N_{\mathrm{S}}(t)$, $N_{\mathrm{T}}(t)$ describe the total number of N, S and T-nodes in the network respectively. The evolution of these functions is governed by the following

system of equations:

$$\frac{dN_N}{dt} = \mu - \lambda_1 N_N + \lambda_2 N_S - \delta_1 N_N \quad (1)$$

$$\frac{dN_S}{dt} = \lambda_1 N_N - \lambda_2 N_S - \delta_1 N_S - \gamma N_{TS} \quad (2)$$

$$\frac{dN_T}{dt} = \gamma N_{TS} - (\delta_1 + \delta_2) N_T. \quad (3)$$

The term $N_{\rm TS}$ corresponds to the interaction of susceptibles with the terrorists, and therefore characterizes the level of infiltration of the terrorists into the rest of population. In order to close the above system of equations, we introduce equations describing the evolution of the possible links present in the network. With the three types of nodes, N, S, T, there are six types of undirected links that must be accounted for. Let the functions $N_{\rm TT}$, $N_{\rm TS}$,$N_{\rm TN}$, $N_{\rm SS}$, $N_{\rm SN}$ and $N_{\rm NN}$ describe the number of edges that connect two nodes with types indicated by the subscript, i.e. $N_{\rm TS}$ is the number of edges connecting a terrorist node to a susceptible node.

The system of three equations described above, together with equations for the six link variables derived using a moment closure approximation, presents a closed system of equations that we solve numerically and confirm the mean-field predictions with direct Monte-Carlo simulations. The solution of this system will answer questions about the effectiveness of the recruitment methods, discover existence of steady state solutions, and allow characterization of the statistical properties of networks that could result as a result from such dynamical system. For example, $N_{\rm TT}$ will contain the statistical description of the terrorist network.

The derivation of the link equations will be given in detail in another paper. The link equations, which include the birth/death processes as well as rewiring of nodes, also assume that the newborn non-susceptible nodes are attaching themselves to the network via σ links to N-nodes:

$$\partial_t N_{\rm NN} = \lambda_2 N_{\rm SN} - 2\lambda_1 N_{\rm NN} + \sigma\mu - 2\delta_1 N_{\rm NN} \quad (4)$$

$$\partial_t N_{\rm SN} = -\lambda_1 N_{\rm SN} - \lambda_2 N_{\rm SN} + 2\lambda_1 N_{\rm NN} + 2\lambda_2 N_{\rm SS} - \gamma \frac{N_{\rm SN} N_{\rm TS}}{N_{\rm S}} - 2\delta_1 N_{\rm SN} \quad (5)$$

$$\partial_t N_{\rm SS} = -2\lambda_2 N_{\rm SS} + \lambda_1 N_{\rm SN} - 2\gamma \frac{N_{\rm TS} N_{\rm SS}}{N_{\rm S}} - 2\delta_1 N_{\rm SS} \quad (6)$$

$$\partial_t N_{\rm TN} = -\lambda_1 N_{\rm TN} + \lambda_2 N_{\rm TS} + \gamma \frac{N_{\rm SN} N_{\rm TS}}{N_{\rm S}} - (2\delta_1 + \delta_2) N_{\rm TN} - w N_{\rm TN} \quad (7)$$

$$\partial_t N_{\rm TS} = -\lambda_2 N_{\rm TS} + \lambda_1 N_{\rm TN} - \gamma \left(\frac{N_{\rm TS}^2}{N_{\rm S}} + N_{\rm TS} \right) + 2\gamma \frac{N_{\rm SS} N_{\rm TS}}{N_{\rm S}} - (2\delta_1 + \delta_2) N_{\rm TS} + w N_{\rm TN} \quad (8)$$

$$\partial_t N_{\rm TT} = \gamma \left(\frac{N_{\rm TS}^2}{N_{\rm S}} + N_{\rm TS} \right) - 2(\delta_1 + \delta_2) N_{\rm TT}. \quad (9)$$

Here the $\sigma\mu$ is the source term, describing the creation of new links. The $wN_{\rm TN}$ term corresponds to the rewiring process, where a terrorist node may decide to rewire its link to an S-node in favor of an N-node, at a rate w. All the other linear terms are due to nodes changing their status from N to S and back, as well as due to the death of links as a result of one of the end nodes dying. The nonlinear terms describe the links changing their types as a result of one of the end nodes becoming a T-node.

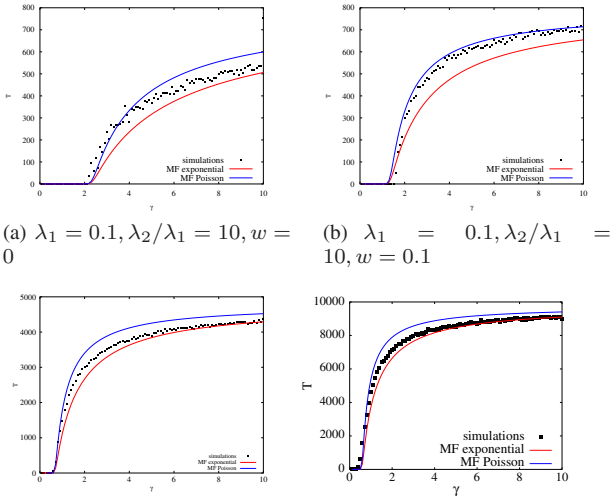
Simulation results

First, we rescale time t by δ_1. This allows us to discuss the rates of the participating events as they compare to the death rate. Thus, the ratio of μ to the death rate determines the characteristic size of the population. Second, we consider three regimes of the behavior based on the characteristic number of times any particular node becomes susceptible to recruitment: a) $\lambda_1 \gg \delta_1$, b) $\lambda_1 \sim \delta_1$, c) $\lambda_1 \ll \delta_1$. Finally, we assume that any given node spends most of the time being non-susceptible, that is the following holds: $\lambda_2/\lambda_1 \gg 1$. The other parameters used in the simulations are $\delta_1 = 1.3, \mu = 1.3 \times (11,000), \sigma = 50$, $\delta_2 = 0$. Note that with this choice of birth and death rates the total population is $11,000$.

In Fig. 3 we study the dependence of the total number of T-nodes in the steady state regime on the recruitment rate γ. Figs. 3(a), 3(c) and 3(d) correspond to three regimes of λ_1. Two different mean field models are used with differing distributions in derivations, and results are plotted for both of them. We can see that far from

the *growth threshold* the total number of T-nodes in the network can reach large fraction of the total population in the cases of $\lambda_1 \sim \delta_1$ and $\lambda_1 \gg \delta_1$. On the other hand, for $\lambda_1 \ll \delta_1$ the fraction of the population recruited can be only a small fraction of total population. Furthermore, comparing the Fig. 3(a) with Fig. 3(b), we see that the rewiring can increase the total T-node population and bring the growth threshold closer to zero. The result is that increased interaction between terrorist and susceptible groups due to adaptive rewiring causes terrorist group onset for earlier levels of parameter γ.

The solid curves in Fig. 3 show that the mean-field we derived does not match well with the simulations. We suspect that the reason for the deviation is a poor approximation for the distribution of number of T-nodes neighboring each S-node. This is being explored in a longer paper.



(a) $\lambda_1 = 0.1, \lambda_2/\lambda_1 = 10, w = 0$ (b) $\lambda_1 = 0.1, \lambda_2/\lambda_1 = 10, w = 0.1$

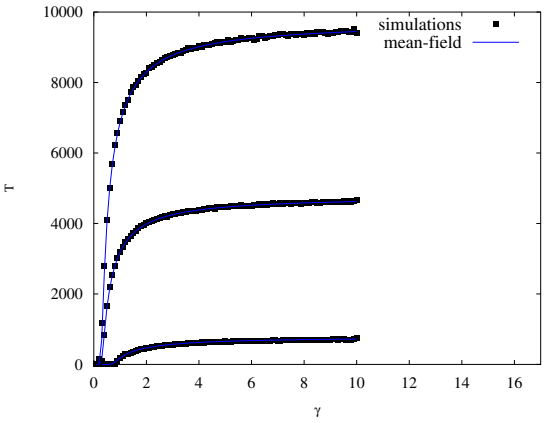
(c) $\lambda_1 = 1.0, \lambda_2/\lambda_1 = 10, w = 0$ (d) $\lambda = 10, \lambda_2/\lambda_1 = 10, w = 0$

Figure 3. Dependence of the number of terrorist nodes on the level of recruitment γ. Black squares: direct simulations, Blue curve: mean field where distribution of number of T-nodes neighboring an S-node is approximated by Poisson distribution (Eqs. 4-9), Red curve: mean field where distribution is approximated by exponential distribution.

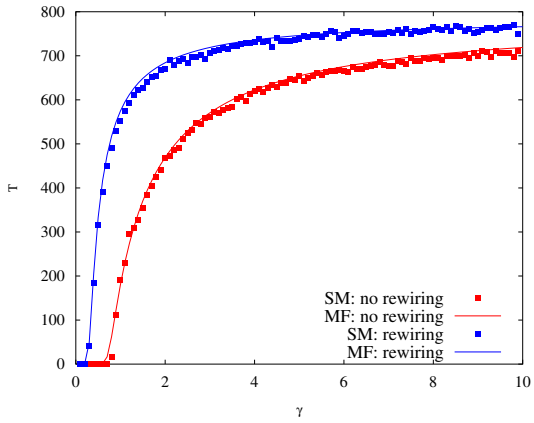
We introduce another birth process, where the new nodes can be attached to a randomly selected node, independent of the node's current status. The mean-field equations are modified by distributing the term $\sigma\mu$ in Eq. (4) proportionally among the equations for NN, SN, and TN links. As shown in Fig. 4, the simulations agree well with the mean-field theory.

IV. Control and Adaptive Rewiring

All finite population models exhibit randomness resulting in observed fluctuations. As components, such as



(a)



(b)

Figure 4. Dependence of number of terrorists on γ in a system with homogeneous attachment of newborn nodes. Fig. 4(a) shows the three different parameter regimes. Fig. 4(b) shows the dependence on presence of rewiring.

infectives or recruiting individuals, evolve in large populations, there is the possibility of finite time extinction. In disease propagation, extinction occurs when the number of infectives becomes so small that there is insufficient transmission to keep the disease in its endemic state. Fluctuations cause the extinct state to be reached in a finite time. Populations based upon adaptive networks further complicate the problem, since social dynamical situations, such as disease avoidance strategies, can cause both the endemic and extinct states to be bistable [6], [7].

A major characteristic of fluctuation-induced extinction in globally connected stochastic models for large populations is the extinction rate. Viewing disease fadeout as coming from systems far from thermal equilibrium, finite population extinction rate laws have been derived in SIS [19], [20] and SIR [21] models. Recently,

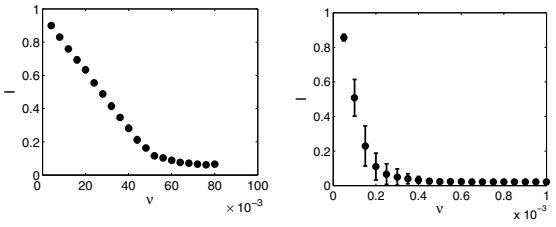


Figure 5. Average infected fraction I versus mean vaccine frequency ν. Left panel: No rewiring of the network. Right panel: With rewiring of the network. Two orders of magnitude less vaccine is needed to suppress infection in the adaptive network [8]. Reprinted from [9].

non-Gaussian vaccination has been used to derive enhanced extinction rates in such stochastic models [22].

Vaccine strategies have also been considered for epidemics spreading on static contact networks. Targeting of high degree nodes has been shown to be more effective than random vaccination for scale free networks [23], [24], small world networks [25], and other social network geometries [26]. Since targeting the highest degree nodes requires full knowledge of the network geometry, models based on local knowledge have been developed. For instance, vaccinating a random acquaintance of a randomly selected node tends to favor high degree nodes and is more effective than random vaccination [27]. In the presence of limited vaccine resources, outbreaks can be reduced by fragmenting the network via a graph partitioning strategy that requires less vaccine than targeting high degree nodes [28].

Recently, we used a random non-Gaussian vaccination strategy and found that in conjunction with adaptive rewiring, it is extremely effective. We assumed that pulsed vaccination was a Poisson process with fixed amplitude of susceptibles vaccinated and a mean frequency ν of application to the population. A comparison of the results of the vaccination with and without network adaptivity is shown in Fig. 5. (See [8] for details.) To eliminate disease, vaccination takes advantage of targeting susceptibles in the population. Because of the adaptivity of the network, rewiring leads to susceptibles with higher degree on average. Random vaccination of the susceptible population will automatically tend to target higher degree nodes and is therefore expected to be much more effective than when applied to a static network, where the high degree nodes are likely to be infectious and not selected for vaccination.

V. Conclusions

We considered the dynamics of social networks where the nodes are rewired as a function of the state of the system. As a result of rewiring, the links change in time, inducing a change in node dynamics. The entire system is expected to fluctuate, which causes a nonzero probability for one or more components to go extinct.

We considered two distinct models of adaptive networks: an infectious disease model and a terrorist recruitment model. In the disease model, susceptibles rewire away from infectious individuals to other non-infectious individuals, while in the recruitment model, terrorist recruiters try to connect to those who are susceptible, thereby optimizing their numbers.

Finally, we considered one example of control in the disease model. We incorporated a simple vaccine procedure and saw that as a result of both rewiring and vaccination, two orders of magnitude less vaccine was needed to extinguish the disease in a large population.

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