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Why scale-free networks are a good thing for controlling disease transmission

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Abstract—The observation of a scale free degree distribution in real disease incidence data tends to suggest that such diseases would be difficult, or in fact impossible to control. Moreover, it has been shown that the fat tail degree distribution for scale free networks implies that (SIS type) disease transmission cannot be eradicated for any nonzero level of infectivity. Nonetheless, we have found that when one considers a voluntary immunisation strategy, or even, disease transmission in multiple waves, the presence of hub nodes actually becomes an advantage. Highly connected nodes will be immunised earlier (or conversely, quickly encounter an early and less virulent strain of infection). As a consequence of the frailty of scale-free networks, the removal of hub nodes will actually reduce the infectivity of the disease. In this paper we consider disease transmission on scale free networks and on more stratified networks, motivated by structures observed in society. We find that the level of infection for scale free networks is actually only moderately higher than for equivalent structured networks. Nonetheless, in finite size networks extinction occurs at a higher threshold for structured (non-scale free) networks. Conversely, these structured networks exhibit broader (in time) peaks in the disease outbreak.

1. Introduction

Since the recent rediscovery of small world and scale free networks by Watts and Strogatz [7] and Barabasi and Albert [1] transmission of infectious agents on such networks have been one of the doctrinaire examples. Indeed, it is natural to consider that for diseases for which personal contact is required to support transmission (as opposed to air borne pathogens) that the degree of connectivity between individuals will have an effect on the transmission of the disease.

The importance of this idea was given weight by the work of Boguñá and colleagues who showed that disease transmission on a scale free network will persist for any non-zero value of transmission rate [2]. This is in direct contrast with our experience of homogeneous mixing (the standard differential equation based model of disease transmission) for which there always exists a finite non-zero threshold below which an infection will decrease to zero, and above which it will remain endemic.

Nonetheless, it is important to examine the result of Boguñá *et al.* a little more closely. Essentially, the result considers the case of SIS disease transmission on a perfect scale-free network. That is, there are two disease states, susceptible (S) and infected (I). Susceptible individuals become infected with some probability λ if they have an infected neighbour. Without loss of generality, infected individuals recover and become susceptible with rate 1. If one assumes full connectivity then we have the case of disease transmission on a homogeneous system and there exist a critical rate $\lambda_c > 0$ such that if $\lambda < \lambda_c$ the disease will be eradicated. The remarkable result of Bogñá *et al.* is that if one examines disease transmission on a scale free network (i.e. less than full connectivity) then the exists no such $\lambda_c > 0$. However, the fundamental requirement for this to occur is that one must consider a infinite perfect scale free network (in [2], the authors also only consider the case where the degree exponent is between 2 and 3 — but this restriction is not entirely necessary [5]). That is, the network is infinitely large and consists of nodes such that the probability P of a node having degree k is given by

$$P(k) \approx Ck^{-\gamma} \tag{1}$$

for $2 < \gamma \leq 3$.

Boguñá and colleagues show that λ_c may be calculated exactly

$$\lambda_c = \frac{< k >}{< k^2 >} \tag{2}$$

and, of course, for degree distribution (1) both $< k >$ and $< k^2 >$ are infinite (hence, $\gamma < 3$ is actually sufficient to ensure their result). Note, however, that this presumes that the network is uncorrelated (in obtaining (2) the authors assume that the degree distribution (1) applies equally to all nodes). Moreover, the network must be infinite (for otherwise a finite sample variance exists).

In [5], Small, Walker and Tse presented the first evidence that such behaviour could arise in the real world. They showed that the degree distribution of a network inferred from the global spatial temporal distribution of avian influenza outbreaks (in animal populations) cases actually did follow a scale free distribution — in fact, the scale exponent of that distribution was about 1.2) [5]. However, it was not clear that the result of Boguñá et al. could be extended to this situation. We had observed that the degree distribution was highly assortative (high degree nodes tend to connect to one another) [6] and we proposed a mechanism by which such a network could arise [9]. Moreover,

population	N	10^6
proportion of school children	p_{kids}	0.3
mean number of children	N_{kids}	2
class size	c	30
school size	s	30
mean degree (children)	ℓ	5
links outside their own class (children)	$\ell_{\overline{\text{class}}}$	2
links outside their own school (children)	$\ell_{\overline{\text{school}}}$	1
mean degree (adults)	γ	5
links to adults within the same school	a_{school}	2

Table 1: Model parameters used in the community structured complex network model.

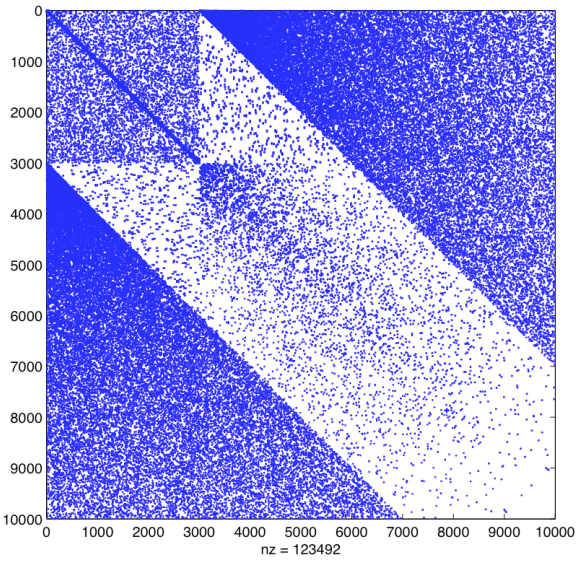

Figure 1: Adjacency matrix for the community structured complex network model. For ease of visualisation this system has $N = 10^4$, $c = 10$ and $s = 5$. Other parameters are the same. Children appear first (upper left most). The clustering within schools and between schools is evident along the main diagonal in the upper right. But, note that links are otherwise relatively sparse (the axes of this figure exaggerate the density of links in this region). Links between adults appear to follow a scale free like distribution — note that the connections between these adult hubs and the population of children are random.

the network was certainly finite, so the question of whether this was a good sampling of an underlying infinite network, or whether the sample mean and variance would imply a finite $\lambda_c > 0$ was unresolved. Moreover, we must note that this network is only inferred from time series data: we can not be certain that this necessarily captured the relevant structure in the true transmission pathways.

In addition to the connectivity model of [2] being rather idealised, the simple SIS dynamics also lacks some of the complexity of the real world. In [8] Zhang and colleagues consider the case where nodes can choose to receive vaccination, and that the decision to vaccinate is affected by node degree (that is, those with a high degree have more risk and therefore more reason to vaccinate). Under this situation, the system behaviour is the opposite of that described in [2]. That is, scale free networks, with hub nodes being more ready to vaccinate actually inhibit the spread of disease when compared to homogeneous mixing.

In this paper we report out recent attempts to address this problem from another angle. Rather than pursuing the pure scale free networks popular in the physics literature we attempt to construct a communal model of connectivity within a city — the basic defining characteristic of which is the localisation of children within schools — and we study the transmission dynamics within this system. This network is structured in such a way that it contains distinct layers (adults and children) and has both characteristics of scale free and small world networks. We examine the behaviour of disease transmission on this network to determine whether the results from computational physics can also be applied to this somewhat more realistic model.

2. The model

The purpose of this paper if to compare the behaviour of networks with more realistic transmission topologies to standard scale free complex networks in the vein of the Barabasi-Albert (BA) model [1]. We assume two distinct populations in the community: adults and children. Each child attends a school and their network connections are constrained by that school structure. Each adult is connected in a scale-free fashion to a certain number of other adults. Some adults are also parents, and in these cases they are connected to their children and to fellow parents within the same school. We assume that parents always occur in pairs, and that each adult can be a member of at most one such pairing.

Our network model necessitates a moderate number of parameters, all of which can be given reasonably "realistic" values: nonetheless, we would like to stress that our results do not sensitively depend on these choices (for a "sensible" range of values). The parameters are listed, along with our chosen values in Table 1. The network has a total of N nodes, $p_{\text{kids}}N$ children (all assumed to be of school age) and the remaining $(1 - p_{\text{kids}})N$ adults. Each family has two adults and a mean N_{kids} children (following a Poisson distribution). Each child is linked to exactly ℓ other children of which $\ell_{\overline{\text{class}}}$ are in classes other than their own and $\ell_{\overline{\text{school}}}$ are outside their school (that is, most children are connected only within that same school, and largely within the same class).

This creates a network of children which is both hierarchical and clustered. Children are arranged in classes, with dense links within classes. The classes are then arranged in

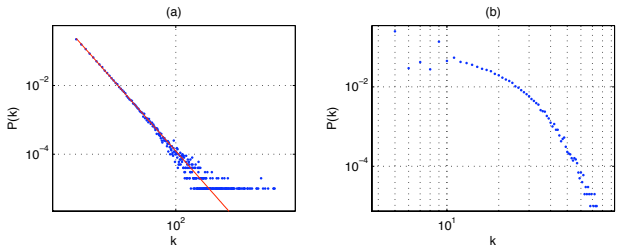

Figure 2: Degree distribution for the community structured complex network model and for the standard Barabasi-Albert scale free network model.

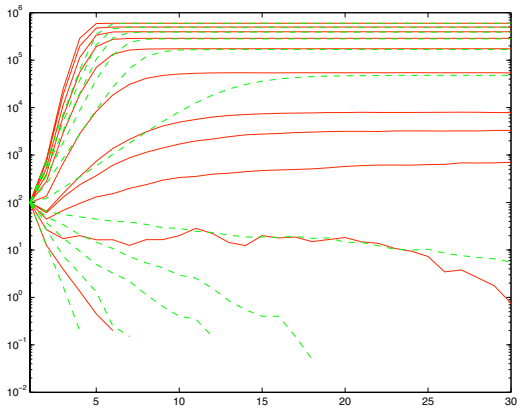

Figure 3: Comparative rate of spread of disease on a BA network (red, solid) and the community structured complex network described in the text (green dashed) for $\lambda = 0.01, 0.02, 0.03, 0.04, 0.05, 0.1, 0.2, 0.3, 0.4, 0.5$ and $N = 10^6$. We plot the mean of 20 realisations. For sufficiently low rate of transmission $\lambda = 0.01, 0.02$ diseases on both networks become extinct. For moderate transmission $\lambda = 0.03, 0.04, 0.05$ disease on the community network becomes extinct, but on the scale free network remains endemic. For larger $\lambda \geq 0.1$ transmission on both networks is endemic and at similar levels (although the scale free network is slightly larger).

schools, with moderate density of links between schools. Links between schools are relatively sparse, but sufficient to ensure that the resulting network is small world.

Each adult contributes γ links with preferential attachment — leading to a scale free distribution. For parents a_{school} of those links are restricted to be chosen from among fellow parents of the same school attended by their children. Hence, viewed in isolation from the children, the parents form an independent scale free network. However, for the adults that are also parents they are then connected directly to the hierarchical small world network of children. A typical adjacency matrix of a network constructed with this scheme is depicted in Fig. 1.

In Fig. 2 we depict the degree distribution for this network and for an equivalent BA network. The societal network has mean degree of 12.4. To construct a preferential attachment scale free network with an equivalent mean degree we add 7 links with each new node — doing so yields a scale free network with a mean degree of 14. Similarly the assortativity for the society network is 0.029; and for the preferential attachment network is −0.0050. The mean path length (computed from a random sampling of pairs of points on each network) is 5.57±0.54 and 4.48±0.59 (mean ± standard deviation of 100 samples) respectively.

3. SIS disease dynamics

For each of the networks described in the previous section we simulate the effect of SIS transmission dynamics for various rates of infectivity λ. In Fig. 3 we report the mean infected population as a function of time over 20 realisations.

Despite the fact that both networks have similar connectivity, similar degree and similar mean path lengths (the scale free network being a little more highly connected) we do see (as anticipated by the theoretical results of [2]) that the scale free network supports a high incidence of infection. Nonetheless, the effect is rather less marked than one might have expected. For moderate of large values of λ the difference is only that the preferential attachment network reaches the steady state infectivity level somewhat quicker.

In both scale free and the socially structure networks the steady state level is very similar. Certainly, for moderate levels of infectivity, there is a range of values $\lambda = 0.03$, 0.04 and 0.05 for which the preferential attachment scale free network supports a contagion that becomes extinct on the non scale free network. However, this range is quite small and the asymptotic level of infection is rather low (between 0.01% and 0.1% of the population[1]).

Finally, for small values of λ we note that both models become extinct. As noted earlier, this is to be expected, even for scale free networks. Since the networks used in this study are finite one must expect $\lambda_c > 0$.

Now, we turn to the central issue of the current study — what happens when a disease occurs in waves (and successive wave confer resistance)? (Or, equivalently, what is the effect of vaccination?) We modify the above SIS model by assigning an increased resistance to infection for a node based on previous infection. Let α ($0 \leq \alpha \leq 1$) be a constant such that if a node has been infected on k previous occasions, the rate of successive infection is reduced from λ to $\alpha^k \lambda$. Fig 4 illustrated typical results. Note that if $\alpha = 1$ this reduces to the standard SIS model, and if $\alpha = 0$ it is SIR.

As expected, the gradual conferment of immunity means

[1] Admittedly, this value is comparable to the incidence of SARS in Hong Kong in 2003 [4, 3]

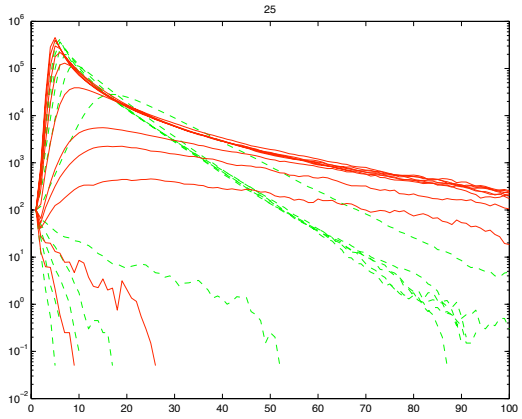

Figure 4: Comparative rate of spread of disease on a BA network (red, solid) and the community structured complex network described in the text (green dashed) for $\lambda = 0.01, 0.02, 0.03, 0.04, 0.05, 0.1, 0.2, 0.3, 0.4, 0.5$ and $N = 10^6$. We plot the mean of 20 realisations. We set $\alpha = 0.25$ so that each infection of a node will mean a 75% lower risk of reinfection.

that the disease eventually becomes extinct. Moreover, the rate of extinction (the total duration of the infection) for the structure community network is consistently less than for the BA network. However, in the structured network we also observe a broader initial peak. That is, the peak of infection is longer for the community network than for the scale free network.

4. Conclusions

The scale free BA network is an appealing model for physicists interested in the study of transport in complex systems, and disease transmission is a useful prototypical application of this model. However, complexities in the real world do not always mirror such neat abstractions. In this work we have focussed on the comparison of this model to (perhaps) more realistic alternatives.

Our results show that the persistence one observes in SIS type dynamics in infinite BA networks is a reasonable proxy for moderately large (but finite) BA simulations. In the case of the community model proposed in this paper, we find that the level of infection is somewhat — but not drastically lower. The most marked distinction is that the disease propagates to and reaches it's equilibrium more slowly. We also observe a somewhat larger threshold λ_c.

These results are also mirror we we consider the case of partial immunisation — a simple proxy for a more complex model of vaccination [8]. Interestingly, in this case, the time course for the disease of both networks is very similar. The main difference being that the BA model exhibits a sharper and narrower peak, while the community model (for comparable parameter values) has a broader and lower maximum. We now need to extend this community model to ensure that it does closely reflect reality. The model structure — with separate communities of adults and children also means that it may be a useful tool to study the transmission of diseases which affect adults and children differently. This may also help us to study disease which typically break out in waves — first affecting mostly children, and later adults.

Acknowledgments

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