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Unexpected epidemic thresholds in heterogeneous networks: The role of disease transmission

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We reformulate several recent analyses of infection processes on highly heterogeneous networks (e.g., scale-free networks) which conclude that diseases will spread and persist even for vanishingly small transmission probabilities. The results of these latter studies contrast with conventional epidemiological models where there are clear threshold effects, namely, should the transmission probability fall below a critical threshold level the disease is expected to die out. Here we show that epidemic propagation depends equally on the infection scheme as well as the network structure. Connectivity-dependent infection schemes can yield threshold effects even in scale-free networks where they would otherwise be unexpected.

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The notion of thresholds forms a central part of classical and current epidemiological theory and carries important implications for disease eradication and vaccination programs. Recent modeling studies [1–5] demonstrate that social and spatial structures governing the connectivity of individuals in networks have major control over the spread of infections and the emergence of intrinsic disease thresholds. For example, these thresholds are believed to be absent in scale-free networks where vanishingly small infection probabilities suffice for a disease to spread and perpetuate. To date most of the effort has focused on studying the effects of network architecture, paying little attention to disease transmission. Here we show that transmission mechanisms are decisive factors in determining epidemic thresholds. This has implications not just for human populations, but also for applications such as the spreading of computer viruses across the internet.

When modeling epidemics, it is convenient to formulate the population in terms of its underlying graph structure with nodes representing individuals, and potential contacts or connections between pairs of individuals as edges of the network. The connectivity of the graph naturally controls to a great degree the spread of an infection through a network. It is well understood that for a large and completely random graph, there is a threshold level of connectivity [6]. Below the threshold, individuals are essentially disconnected from one another all direct and indirect pathways being considered. However, for a critical number of edges the graph forms a "giant component" whereby nearly all individuals are connected to one another. One of the goals in this paper is to distinguish between this threshold effect due to the giant component, and other thresholds that are usually overlooked and are due to the intrinsic dynamics of the disease itself.

We use the simplest *SIS* model to explore the disease dynamics on a network. In this model, individuals are either in a Susceptible (S) state or an Infected (I) state. Let λ be the rate at which a susceptible individual changes from S to I upon contact with a single infected individual. The parameter δ is taken to be representative of the recovery rate of an individual from the infected state, and thus its return to the susceptible state.

Models of this type demonstrate clear infection threshold effects on random and small-world networks [2,3] both of which are characterized by their relatively homogeneous network connectivity (i.e., with all nodes having approximately an equal number k of connections). In the presence of a threshold, disease eradication requires reducing the infection rate below a critical level $\lambda=\lambda_c$ where a stable infection-free equilibrium is guaranteed. In epidemiological terminology, the infection threshold may be expressed in terms of the basic reproductive number R_0, the average number of infections produced by a single infected individual in a population of susceptibles. Crossing the threshold reduces the basic reproductive number R_0 below unity and the infection is prevented from propagating. In practice, the same result can be achieved by random immunization of a proportion of the population [5], since it reduces the effective R_0.

It is important to realize that the above results are for homogeneous network structures such as random and small world topologies, where the number of connections per node changes little over the whole graph. However, this formulation needs modification for heterogeneous networks where the number of connections per node is highly variant. May and Anderson [1,4,5] have shown that heterogeneity in network structure acts to increase the effective R_0 as follows:

$$R_0 = \hat{R}_0[1 + C_V(k)^2]. \tag{1}$$

Here $\hat{R}_0$ is the basic reproductive rate obtained for a homogeneous network having the average connectivity $\hat{k}=\langle k\rangle$ and $C_V(k)$ is the coefficient of variation of the heterogeneous network. That is, $C_V(k)$ is a measure of the variability in the number of edges k at each node over the entire graph. Consequently, an increase in $C_V(k)$ could raise the basic reproductive number above the threshold level $R_0=1$, increasing the probability of an epidemic outbreak.

Epidemic threshold dependence upon heterogeneity in connectivity introduces an analogy to percolation thresholds. It is known that highly heterogeneous networks are extremely resilient and even for high level of dilution (for example through vaccination), nodes are still connected in a giant component [7]. In particular, if the network has an infinite $C_V(k)$, then the giant component is always present for every level of dilution. According to Eq. (1), on these networks $R_0>1$ and there is always a chance of an epidemic outbreak.

Scale-free networks exemplify this point well. These

networks are extremely heterogeneous with some individuals being highly connected, but the great majority having only few connections. The probability of a node having exactly k connections is given by the power-law distribution $p_k \sim k^{-\gamma}$. When $\gamma \leqslant 3$ the variance (and $C_V(k)$) of the node-connectivity approaches infinity for a large number of nodes (N). In this regime, all nodes are connected in a giant component where there are a vast number of pathways by which an infection can propagate or percolate through the network. Thus, it is assumed that on these networks the lack of a percolation threshold is the reason for the absence of an infection threshold. In practical terms this would make random vaccination ineffective. But although it is obvious that on a disjoint network an infection will not propagate, we show that surprisingly, it is not necessarily true that in the percolative phase a disease will spread.

We claim that the analogy between infection and percolation threshold holds for a limited range of cases only. In all studies until now where this analogy has been made, it was supposed that the infection rate λ is constant for all individuals. Doing so implies two assumptions. The first is that the infection rate of a node is independent of the number of connections it has (i.e., connectivity independent), and the second is that infection rate is symmetrical. By symmetrical we are referring to the assumption that the transmission probability between a highly connected infected individual to its poorly connected susceptible neighbor is equal to the transmission probability in the converse situation, where the poorly connected individual is the infected and the highly connected neighbor is the susceptible. In our opinion, these assumptions are too restrictive and fall short when trying to describe the vast spectrum of disease propagation strategies. Introducing a connectivity correlated transmission rate $\lambda(k)$ enables us to relax these assumptions and to broaden the scope of the *SIS* model. However, once λ is connectivity dependent, Eq. (1) no longer holds in its present form, and we explore the consequences below.

Mean field models have been described for analyzing heterogeneous graphs having arbitrary node distribution. Let I_k be the density of infective nodes within the class of nodes having exactly k connections. Hence the mean-field approximation for the general infection scheme takes the following form [1,3,8]:

$$\dot{I}_k = \tilde{\lambda}(k)k(1 - I_k)\Theta - \delta I_k. \tag{2}$$

The first term on the right-hand side of Eq. (2) represents the assumption that the average density of newly infected nodes $\dot{I}_k$ is proportional to the density of susceptible nodes from class k (i.e., $1-I_k$), the number of edges (k) emanating from each one of those nodes, the rate at which an infection is admitted through an edge ($\tilde{\lambda}(k)$), and the probability that a given edge is connected to an infected node and can transmit the infection (Θ). The second term represents the infected nodes recovery rate δ (or dilution rate in the language of percolation theory). Since we are free to scale time, without loss of generality we can set $\delta = 1$.

By taking $\tilde{\lambda}(k)$, it becomes possible to model diseases which have a connectivity correlated (i.e., k-dependent) infection rate. One can interpret the correlated infection rate in the context of edge percolation as $\tilde{\lambda}(k) = \lambda A(k)$, where $A(k)$ is the probability that a susceptible node would actually admit an infection through an edge connected to an infected node. If so, it is assumed that the infection is transmitted at rate λ and the edge is referred to as occupied. Thus each susceptible from class k has on average $kA(k)$ occupied edges from which it could be infected. Following the same interpretation, an infective node has on average $kT(k)$ occupied edges from which it can transmit the disease. Thus the probability Θ is approximated as the proportion of infective occupied edges over the entire network [9]:

$$\Theta = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} kT(k)p_k I_k \tag{3}$$

where p_k is the probability that a node has exactly k connections and n is the largest degree of any node in the network.

It is easy to see that system 2 always has an infection free steady state ($I_k^* = 0$). The system may also have a positive steady state with $I_k^* > 0$ which will be referred to here as the endemic steady state. It was analytically shown [8,9], that a constant infection rate $\lambda(k) = \lambda$ on a scale free network with only a percolative phase always generates a positive steady state. It was also assumed that on those networks the infection would always prevail implying that the existence of an endemic steady state also implies its stability. However to our knowledge, no formal proof has ever been given. Our aim is to find conditions on $A(k)$ and $T(k)$ for the emergence of a threshold level λ_c on those networks. In doing so we also present a rigorous stability analysis.

We find it more convenient to present system 2 in matrix form:

$$\dot{\boldsymbol{I}} = \left[\frac{\lambda}{\langle k \rangle} (\hat{K}_A \cdot (\mathbf{1} - \boldsymbol{I})) \otimes (\hat{K}_T \cdot \boldsymbol{p}) - \hat{1} \right] \cdot \boldsymbol{I}, \tag{4}$$

where $\boldsymbol{I}$, $\boldsymbol{p}$, and $\mathbf{1}$ are the vectors $\{I_k\}_1^n$, $\{p_k\}_1^n$ and $\{1\}_1^n$, respectively. $\hat{K}_x$ are n on n matrices which obey $k_{i,i} = ix(i)$ and $k_{i,j} = 0$ for every $i \neq j$. $\hat{1}$ is the n on n unity matrix.

Perturbing the steady state $\boldsymbol{I}^*$ so that $\boldsymbol{I}(t) = \boldsymbol{I}^* + \boldsymbol{\epsilon}(t)$ and omitting higher powers of $\boldsymbol{\epsilon}(t)$ gives the stability matrix in the vicinity of the steady state:

$$\boldsymbol{\epsilon} = [B(I^*) - \lambda \Theta^* \hat{K}_\alpha] \cdot \boldsymbol{\epsilon}, \tag{5}$$

where

$$B(I) = \frac{\lambda}{\langle k \rangle} [\hat{K}_A \cdot (\mathbf{1} - \boldsymbol{I})] \otimes [\hat{K}_T \cdot \boldsymbol{p}] - \hat{1}. \tag{6}$$

The eigenvalues (μ_n) of B may be determined from the characteristic equation $|B(I^*) - \mu\hat{1}| = 0$ which is equivalent to:

$$0 = (-\mu - 1)^{n-1} \left(-\mu - 1 + \frac{\lambda}{\langle k \rangle} \sum_k k^2 A(k) T(k) (1 - I_k^*) p_k \right). \tag{7}$$

From the fact that at the endemic steady state $\Theta^* > 0$, it follows that a nonpositive leading eigenvalue (μ_{max}) of $B(I^*)$ is a sufficient condition for the stability of the endemic steady state (Eq. (5)). Introducing the equality

$kA(k)(1-I_k n^*)=I^*/\lambda\Theta^*$ (Eq. (2)) into Eq. (7) we find that the leading root is $\mu_{max}=0$, and we can deduce that if a positive steady state exists (i.e., $\Theta^*>0$) it must be stable.

The existence of the positive steady state is established by following the same argument as was introduced by Pastor-Satorras *et al.* [9] for the uncorrelated infection rate $\lambda(k)=\lambda$. Note that from Eq. (2) the nonzero steady state must satisfy: $I_k^*=\lambda A(k)k\Theta^*/(1+\lambda A(k)k\Theta^*)$. This imposes a self-consistency expression on Θ^* as expressed in Eq. (3), and yields a necessary and sufficient condition for the existence of a positive steady state, namely $\Theta^*>0$, or:

$$\frac{\lambda}{\langle k\rangle}\sum_k A(k)T(k)k^2p_k>1. \tag{8}$$

On the other hand in the vicinity of the infection free steady state $\Theta^*=0$ and a negative leading eigenvalue (μ_{max}) of $B(I^*)$ is imperative for its stability (Eq. (5)). Setting $I^*=0$ in Eq. (7) gives rise to the leading eigenvalue:

$$\mu_{max}=\frac{\lambda}{\langle k\rangle}\sum_k A(k)T(k)k^2p_k-1. \tag{9}$$

By defining the effective basic reproductive number as

$$R_0=\frac{\lambda}{\langle k\rangle}\sum_k A(k)T(k)k^2p_k \tag{10}$$

we see that the infection free steady state is stable if and only if $R_0<1$. Note that this condition is the converse from the condition establishing the existence (and hence the stability) of the positive steady state. Thus R_0 as expressed in Eq. (10) indeed serves as the bifurcation point distinguishing the endemic from the infection free steady state.

One can render Eq. (10) in a way that resembles the expression for R_0 as introduced in Eq. (1):

$$R_0=\hat{R}_0\left[1+\frac{\mathrm{cov}(kA,kT)}{\langle kA\rangle\langle kT\rangle}\right]. \tag{11}$$

where $\hat{R}_0$ is the basic reproductive rate obtained for a random uncorrelated homogeneous network having the average effective connectivity $\langle kA\rangle\langle kT\rangle/\langle k\rangle$. It is easy to see that for $A(k)=1$ and $T(k)=1$ we retrieve May's formula Eq. (1), for the effective R_0, and the infection free steady state for an infinite scale free network (i.e., $C_V(k)\to\infty$) is never stable.

We now illustrate the profound effect correlated infection rates bear upon the emergence of the infection threshold by way of three simple examples.

(1) In the first example, we set $A(k)=1$ and $T(k)=1/k$ implying that the potential transmission rate is connectivity correlated. In contrast a susceptible's potential admission rate is independent of its degree of connectivity. This example would have relevance for macroparasite propagation where infected host individuals have a limited pathogen reservoir and thus transmissibility must be limited within the infection period. In such a case, the more the host's contacts (k) the less would be the chance of transmission per contact. An infection rate that reduces with connectivity k is much more appropriate.

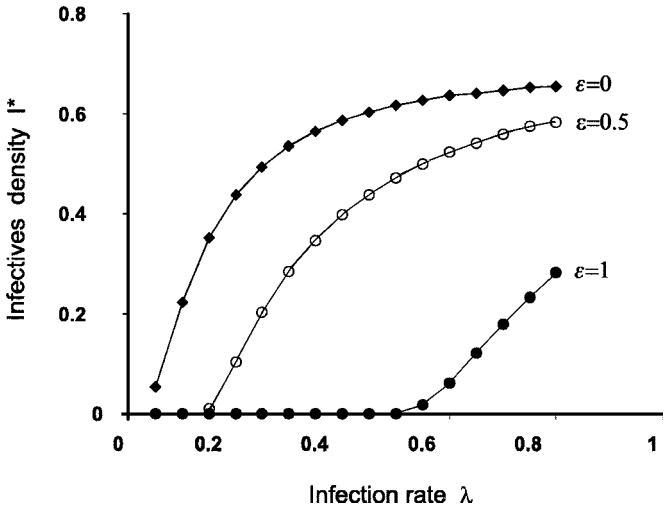

FIG. 1. Infective density steady state I^* vs infection rate λ, for different scaling schemes $\epsilon=0$ (kites), $\epsilon=0.5$ (open circles), $\epsilon=1$ (closed circles) where $A(k)=k^{-\epsilon}$, $T(k)=1$ and $\delta=0.5$. Numerical simulations were implemented on an Albert-Barabasi scale-free network [12] with $\gamma=3$, $N=10^6$ nodes. Each point is the average of 5 simulations run with parallel updating until convergence to equilibrium. Each run began with $I_0=10^4$ initial infectives randomly placed on a uniquely realized network. Thresholds are slightly shifted to the right from the expected values ($\lambda_c=0.5$ when $\epsilon=1$) due to a distribution cutoff effect on a finite size network.

Substituting the above to Eq. (10) yields $R_0=(\lambda/\langle k\rangle)\Sigma_k A(k)T(k)k^2p_k=\lambda$ and $\lambda_c=1$ as the infection threshold level. This scheme gives birth to an infection threshold regardless of the network's connectivity distribution (p_k). Thus reducing λ below unity would prevent pathogen propagation even when applied to a scale free network. Note that this contrasts with the usual understanding of epidemic dynamics on scale free networks where it is believed that there is never an infection threshold and random vaccination could never prevent disease propagation.

We note that Newman [10] also studied "the physically plausible case in which the transmissibility T depends inversely on the degree of the infective individual: $T_k=1/k$." He showed that with this transmission scheme disease cannot spread on networks regardless of their degree distribution (p_k). This would seem to be in contradiction to our own results outlined above. The discrepancy arises from the different schemes for scaling transmissibility. While we directly scaled the exact transmission rate by a factor $1/k$, Newman scales a specially derived "transmission probability" T_k. These two quantities are not equivalent and can yield different conclusions.

(2) The independency of infection dynamics on connectivity distribution becomes even more transparent when considering the reciprocal infection scheme where $A(k)=1/k$ and $T(k)=1$ (see Fig. 1). Although, as Eq. (10) makes clear, the ultimate outcome will be similar to the example above, nevertheless it allows us to give an alternative complementary and intuitive analysis. The scheme expresses asymmetrical infection rates where an infected node can transmit the infection from all of its edges with the same rate, while a

susceptible admits the infection at a rate which is inversely correlated to its connectivity. This reflects the infection dynamics of some sexually transmitted diseases. For example, the WHO reported that 55% of HIV adult infections in Africa are in women [11]. A bipartite network would thus be a suitable framework for modeling HIV propagation in Africa. Since females and males differ greatly in their susceptibility to HIV [11], a projection of the bipartite network onto the male population would be more tangible for mathematical investigation. In this example nodes from type *a* (males) infect nodes from type *b* (females) very efficiently. On the other hand the infection is less efficient when transmitted from females (type *b*) toward males (type *a*) who can receive the disease only after a sustained close interaction. Thus if a susceptible node from type *a* is a member of a highly connected class, the efficiency of each one of its interactions with an infected *b* type node is reduced.

Introducing $A(k)=1/k$ and $T(k)=1$ into Eq. (2) engenders the k-dependent infection rate to counteract connectivity so that the force of infection $(k\Theta\lambda(k))$ does not vary between connectivity classes. Thus for each connectivity class the number of infected nodes are determined solely by the initial conditions. Moreover for approximately homogeneous initial conditions we get $I_k \approx I$ for every k and $\Theta=I$, which renders Eq. (12) to the known *SIS* model on random networks

$$\dot{I}_k = \lambda(1-I_k)I_k - I_k. \tag{12}$$

A simple stability analysis shows that the model has $R_0=\lambda$ an infection threshold at $\lambda_c=1$. Hence this alternative analysis shows the presence of an infection threshold regardless of the network's connectivity distribution (see Fig. 1 where $\epsilon=1$).

(3) We now return to the general case given by Eq. (10). It follows that a necessary and sufficient condition for the existence of a threshold on an infinite network is the convergence of the sum $\Sigma_k A(k)T(k)k^2p_k$. An equivalent mathematical condition, but a weaker one from an epidemiological point of view, would be that the summation tail on $[\hat{k},\infty)$approaches zero for large enough $\hat{k}$. Thus for threshold emergence it is imperative that $A(k)T(k)p_k<k^{-(3+\epsilon)}$ for every $k>\hat{k}$ and $\epsilon>0$.

Applying this condition on a scale free network with a connectivity distribution of $p_k<k^{-3}$ suggests that the infection rate for the high connectivity classes (say for every $k>\hat{k}$) must scale as:

$$A(k)T(k) < k^{-\epsilon}. \tag{13}$$

The condition is sufficient for having an infection threshold even though the network itself possesses no percolation threshold. Since we are free to choose $\hat{k}$ and ϵ the scaling effect is small in the majority of connectivity classes, yielding an epidemiologically reasonable scenario with almost no effect on infection rates but with an imprint on immunization strategies decisions.

Our results also have relevance to the field of computer network security. One could think of the following plausible scenario: the spread of an infected file in a peer to peer network (for example: in a file sharing network like Kazza). Since every peer (node) has a finite upload rate, the larger his connectivity the slower each one of his neighbors would be able to download. In reality, the probability a node would complete a download from a specific source is inversely correlated to the download rate. This yields a connectivity correlated transmission scheme with a probability of $T(k)=k^{(-\epsilon)}$, which needs to be taken into account via the methodology we have set out above.

For scale-free networks, when the infection probability is not k dependent, it has been proved that a connectivity dependent vaccination policy is the only strategy for disease eradication. For implementation it is imperative that the social connectivity pattern of each individual is determined prior to inoculation. This is usually highly impractical especially in cases of unexpected epidemic outbreaks (i.e., bioterrorism, computer viruses). On the other hand, if based on detailed knowledge of the infection, a scaling effect is noted and a threshold emerges, random vaccination might prove sufficiently efficient.

The threshold phenomenon described here, which stems from alternative transmission schemes, demonstrates that epidemic propagation depends equally on the infection scheme as well as the network structure. Proper formulation of optimal vaccination campaigns requires taking both of these important factors into account.

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