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Risk perception and disease spread on social networks

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Abstract

Social networks have become vital in the modeling of disease spread in populations, but so far these models have failed to take into account human risk perception: the fact that human beings, when aware of a disease outbreak, will take precautions to reduce their susceptibility. We study an existing model of risk perception on a social network and note the changes in the efficiency of the disease spread to previous studies that failed to take human behavior into account. We also discuss the accounts that need to be taken into consideration in the construction of accurate models of the population.

Keywords: epidemiological modeling, disease dynamics, scale-free networks, Barabási-Albert model, risk perception

1. Introduction

The field of epidemiological modeling has evolved considerably over the past couple of decades. Originally concerned with compartmental models [1] and assuming homogeneous mixing between all individuals of the population concerned, it has evolved into considering networks of contacts between which the disease may spread. This new form of modeling allowed accounting for the fact that people tend to have only a limited number of contacts to whom they may pass an infection [2]. This allowed for the more accurate modeling of the spread of disease at the individual, as opposed to population-level. Initially homogeneous networks (homogeneous in their connectivity properties) were studied extensively, such as the Erdös-Rényi [3] and Watts-Strogatz [4] models, the latter in particular for its 'small-world' property. Following on from the work of Barabási and Albert [5] in the late 90's, it became increasingly common to model the underlying population as a scale-free network.

A scale-free network is a network whose degree distribution obeys a power law $P(k) \sim k^{-\gamma}$, where usually $2 < \gamma \leq 3$. An important property of these networks when it comes to epidemiological modeling is that their mean connectivity $\langle k \rangle$ is finite, where as the variance is dominated by the second moment $\langle k^2 \rangle$ which tends to infinity as the network grows. In their work Pastor-Satorras and Vespignani [6, 7, 8] studied extensively the spread of disease, following the classical Susceptible-Infected-Susceptible (SIS) model on scale-free networks and discovered, as the network size tends to infinity, the absence of an epidemic threshold: a spreading rate below which the virus will not be able to spread and become extinct. Even for extremely low spreading rates the disease would be able to diffuse through the population and prevail, and this was due to the diverging variance of the network's connectivity. Further work by

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May and Lloyd [9] confirmed this, and pointed out that reason for this is that the most highly connected nodes play a major role in both spreading the infection and containing it. A major conclusion was that scale-free networks are in particular prone to the spread of infection. This finding has had a massive impact on how we model certain diseases: for example, most sexually transmitted diseases (STD) models assume that the main spread of the disease originates from a small set of 'high-activity' nodes, since many network of sexual contacts have a degree distribution that can be fit by power laws with exponents that would agree with the theoretical findings of Pastor-Satorras and Vespignani (for example, see [10]). The reason why scale-free networks are so important for epidemiology is not just for their properties concerning the disease dynamics, but also because many real-world networks are thought to be scale-free, and some have been fitted by power law degree distributions [11]. Furthermore, it has been shown that the degree distributions of locations where people interact and infections may occur also obey power law distributions [12, 13].

Recently, there has been growing interest in discovering ways to model aspects of human reactions to disease outbreaks into epidemiological models. Ferguson [14] argues that people, when aware of an infectious disease outbreak, will sometimes change their behavior in order to reduce the risk of infection. This human reaction can have a dramatic effect on the disease spread. From an economics point of view, Klein et al [15] have also called for an incorporation of human behavior, based on simple economic principles, to improve the accuracy of existing models. So far progress in this direction has been limited although recent work has been done in modeling human behavior under voluntary vaccination campaigns [16], and considering a second network over which spread of disease awareness occurs [17].

In this work we would like to explore the effect of risk perception on the SIS-model disease spread over the classical BA model scale-free network. We define risk perception as information concerning the disease which drives an individual to take measures in order to reduce his susceptibility to infection. The model of risk perception that we describe below has been introduced and studied before on homogeneous 'small-world' networks [18] and on directed scale-free networks of exponent $\gamma = 2$ [19]. It is based on two factors: a global level of awareness and a personal one, the latter being based on the individual observing known disease symptoms in their neighbors. We have chosen the model and network in order to explore the effects that a simple incorporation of risk perception can have on the disease dynamics in terms of the classic studies done by Vespignani and Pastor-Satorras [7]. Even if many real-world networks exhibit power law degree distributions with $2 < \gamma \leq 3$, epidemic outbreaks are actually quite rare, even for highly infectious diseases, and smallpox has even been eradicated. Furthermore, recent studies by Piccardi and Casagrandi [20] have discovered that, under certain conditions, scale-free networks would be less efficient at spreading and maintaining a disease, than their homogeneous counterparts. We develop a mean-field model of the rate of infection occurring per unit time and also run agent-based simulations to test our model. Our findings suggest that incorporating risk perception greatly reduces the number of transmissions that occur, and that, at high levels of risk perception, the disease only propagates between the high-connectivity (central) nodes, while the majority of their low degree neighbors and the periphery of the network are largely unaffected. We conclude that our model of risk perception has caused a great reduction in the scale-free network's efficiency of spreading the virus and, at very high levels, can dramatically reduce its prevalence.

This work proceeds as follows: we start by briefly describing the network model and introducing our definition of risk perception. We then define a mean field model and compare its behavior to the average of a large number of agent-based simulations. We conclude with discussions on the importance of the underlying network model and with suggestions for future work on risk perception.

2. Model

We model the underlying population as a scale-free network, constructed using the classical Barabási-Albert (BA) model of growth and preferential linkage. To briefly summarize: we start with a small network of m connected nodes, and we would like to construct a scale-free network of size N nodes ($N >> m$). At every time step we introduce a new node with degree m, connected to the existing nodes. We keep doing so until we have allocated all N nodes. When choosing the nodes to which the new node will connect we consider the degrees of the existing nodes and are more likely to connect to a high degree node than to a low degree one. The probability of connecting to a node i with degree k_i is:

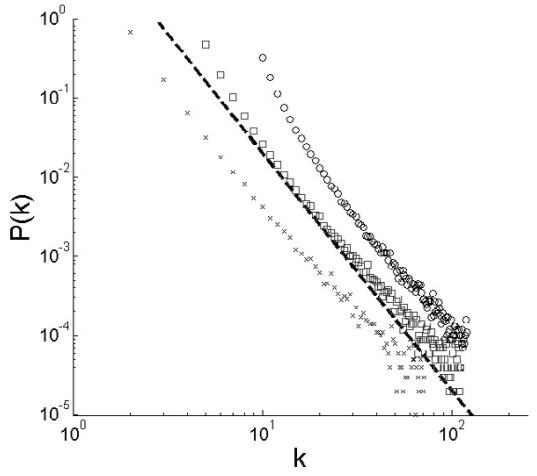

Figure 1: Distribution of the nodes in our network construction for different values of m (= 5 crosses; = 10 squares; = 15 circles). The dashed line has gradient −3. $N = 1x10^5$ nodes.

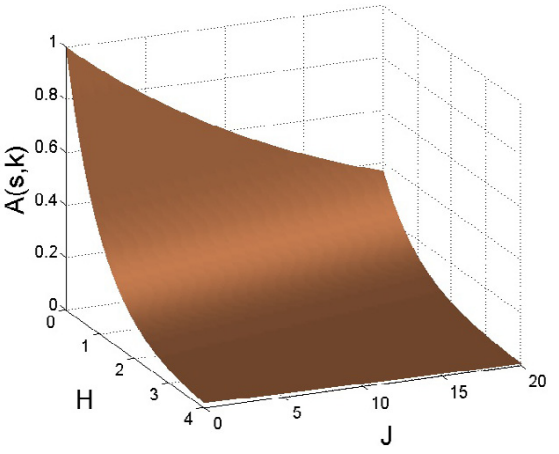

Figure 2: A 3D plot of $A(s,k)$ for varying H and J and s/k set to 1/20.

$$\prod(k_i) = \frac{k_i}{\sum_j k_j}$$

As this network grows it evolves into a scale-free network with exponent $\gamma = 3$. Fig. 1 shows the log-log degree distribution of the nodes of our network for varying m, following the BA model. For the BA model it is known that the mean connectivity $\langle k \rangle = 2m$ and that the degree distribution $P(k) = 2m^2k^{-3}$ [9, 21, 22]. Each node in our model represents an individual and the links between them represent contact sufficient for the propagation of the disease. Each node has a state, which is either susceptible or infected.

In [15] the authors state that the way people respond to the threat of infectious disease "depends on their perception of risk, which is influenced by public and private information". Our model of risk perception seeks to incorporate both these types of information, with public information being global, or shared between all nodes, whereas the level of private information depends on the number of infectious neighbors that the node has: the more infected people an individual observes around him the more concerned he becomes with preserving his personal well-being. Both of these are separate as they come from different sources and have a different effect on the level of risk perception. We model the risk perception or alertness of an individual, with connections k of which s are in the infected state, as the following function, which reduces his susceptibility:

$$A(s,k) = \exp\left[-\left(H + J\frac{s}{k}\right)\right] \tag{1}$$

where H is the parameter denoting global awareness and J is the private perception based on the infected neighbors. Fig. 2 shows a plot for the value of $A(s,k)$ for varying values of H and J, for a constant s/k ratio. Note that for high H the contribution of the value of J diminishes. As an individual becomes aware of a disease through increasing government or media attention he or she is already in a heightened state of alert, and the effect of observing the illness in neighbors has a lower effect. Given that the rate of infection/transmission rate for a particular disease is τ, then any individual faces the probability of becoming infected of $A(s,k)\tau$, and the probability that an individual receives the infection from at least one of its neighbors is:

$$p(s,k) = 1 - [1 - A(s,k)\tau]^s \tag{2}$$

We would like to point out to the reader that the parameters H and J have different effects on the network. They both result in dampening the infectivity τ, however the way they affect the network is different. H is a global parameter – changing this parameter results in the entire network's exposure to the disease being dampened. J also has this effect on the network, with the subtle difference that the effect of J is pronounced differently at each node, because it is scaled according to that individual's connectivity and number of infected neighbors. Thus, J causes the disease to propagate differently at different points in the network. This variation in the spread of infection is equally important to the disease spread as is the underlying model [23]. In this study H is kept at 0, unless stated otherwise.

3. Results

3.1. Mean field approximation

In [6] Pastor-Satorras and Vespignani define the classical mean-field equation for scale-free networks, which considers the infectivity present in nodes of varying degrees and combines these equations to receive the overall fraction of infected. Considering the probability (2) of receiving an infection from at least one of your infected neighbors we can describe rate of change in the fraction of infected individuals i of degree k at time t as:

$$\frac{di_k}{t} = -\gamma i_k + (1 - i_k)g(k) \tag{3}$$

where γ is the rate of recovery, such that the expected time spent sick is $1/\gamma$ and $g(k)$ is a function approximating the number of infected neighbors and the exposure to them according to (2), derived as follows. For any node the degree distribution of any of its neighbors (see [22]) is

$$q_k = \frac{kP(k)}{\langle k \rangle}$$

From this we can estimate the number of infected neighbors to be

$$\tilde{i} = \sum_{k_{min}}^{k_{max}} q_k i_k \tag{4}$$

and this leads us to the definition of $g(k)$:

$$g(k) = \sum_{s=0}^{k} \binom{k}{s} p(s,k)\tilde{i}^s(1-\tilde{i})^{k-s} \tag{5}$$

To summarize, equation (6) represents an approximation of the number of infected nodes that any node of degree k is exposed to, and all the possible scenarios under which transmission may occur. Note that if we wish to obtain the total fraction of infected in the population we sum over all k: $i = \sum_k p_k i_k$. At $t = 0$ i is set to 0.2. We can compare the behavior of our mean field approximation to that of actual numerical simulations to check for correctness. The results for two cases of the fraction of infected i after a sufficiently large period of time of the mean field and the numerical simulations are shown in Fig. 3. Note that although they are mostly in agreement, there is a slight variation for some values of J in which the mean field overestimates the size of the infected population. This is because the mean field can only approximate the number of infected nodes that are in our vicinity, where as the numerical simulations, running on an actual network, would be able to take account of cases where some individuals exist in the periphery of the network and are not exposed to the 'super-spreading' high connectivity nodes. As expected H has the effect of greatly dampening the disease's infectivity and brings down i much more efficiently, whereas J causes variations across nodes as its impact relies heavily on their connectivity. As J grows an increasing fraction of the nodes become less susceptible to the disease, resulting in a decreasing number of infected individuals. For the disease to stop propagating to the high-connectivity nodes J would have to grow to extremely large values, comparable to k_{max}. However, even at low values J already has had a large impact on the disease dynamics by protecting the lower degree nodes from the infection.

We now proceed by examining additional properties of this model incorporating risk perception by averaging over a large number of agent-based simulations.

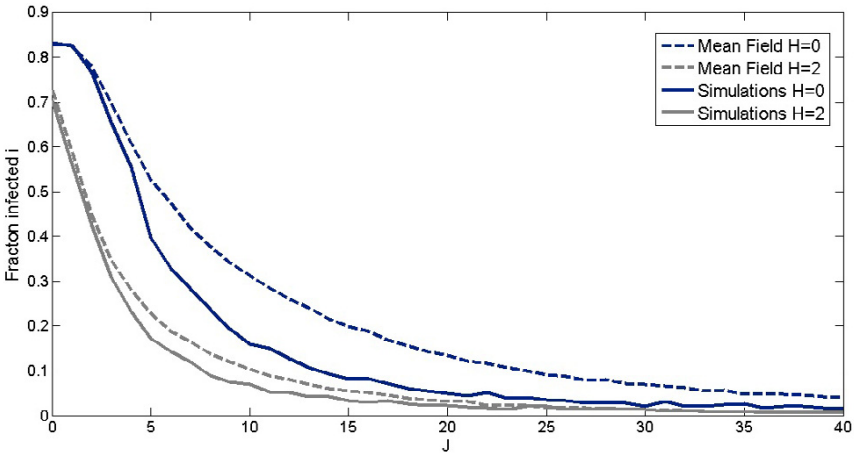

Figure 3: A plot of the mean field and microscopic simulation results of fraction of infected i over J, for two cases $H = 0$ and $H = 2$. For both cases $N = 2500$ nodes were considered with $m = 5$, $\tau = 1$.

3.2. Numerical simulation results

As mentioned in the previous section although the mean field approximation is an important method for evaluating the dynamics of our model, it does not in fact take into account the effect of varying network topologies. The results in Fig. 3 allow us to check the correctness of the implementation of our agent-based simulations so that we can derive additional data from them. We generate a network topology using the BA model and then infect a fraction of c nodes at random. We have noted that for long simulations the statistics explored here tend to converge to the same values, as the model eventually settles to a certain behavior, regardless of the initial number of infected individuals. However we have kept c single valued throughout at $c = 0.2$. At every time step each node can become infected (if they have at least one infected neighbor) with probability $A(s, k)\tau$. Once infected a node can recover from the disease in any subsequent time step with probability γ.

In the following studies we mainly examine the individual (J) parameter or a combination of it and the global (H) awareness parameter. We do not consider the case in which $J = 0, H > 0$. This is because this is equivalent to running the simulations with a disease infectivity τ reduced by a factor of e^{-H}. This would would be equivalent to simply running the same simulations for a different effective infectivity and will not produce interesting results. On the other hand, the case $J > 0, H > 0$ is included to illustrate the impact of a model where an individual's awareness is influenced both by private and public factors.

We start by examining the amount of time spent sick during a long (time $t = 10,000$ steps) set of simulations. We group the results according to the individuals' connectivity k to observe what effect the connectivity has on an individual's exposure to an infectious disease, and how it varies as we increase the perception. The results are shown in Fig. 4. An important aspect to note here is that the variance of the time spent sick for nodes of low connectivity is much greater than that of those of high connectivity (although, over a large period of time and many simulations they should converge to the actual mean value). This is because there are many times more nodes of low k, and there are only extremely few highly connected nodes. When generating network topologies this means that low degree nodes can have a wide range of different neighbors, whereas for high degree nodes this variation is much less. As Christakis and Fowler [24] have pointed out, when studying disease spread over complex networks, visualization of the network topology plays an important role in understanding the process taking place. Positioning the high connectivity nodes, and those closest to them in the center of the network (and the rest in the periphery) would allow us to better visualize the spread of disease in a network, since it is not only the connectivity of the nodes that matters but also their position in the social network or more precisely: to whom they are actually connected. The nodes in the periphery, especially in the case of increasing risk perception, may be largely unaffected by the disease since they are being 'shielded' by

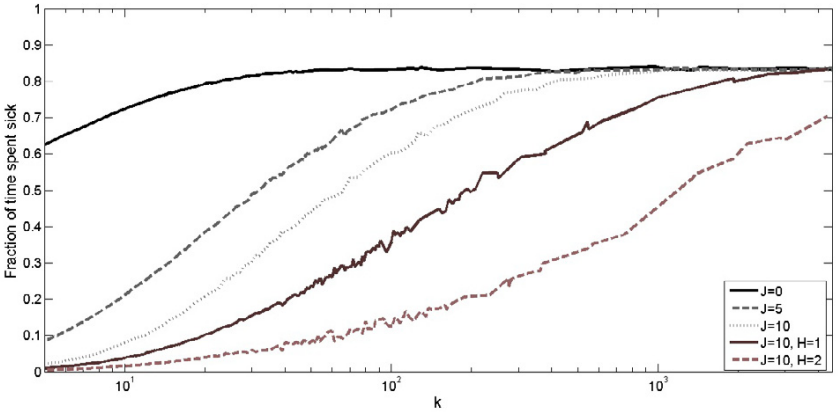

Figure 4: Fraction of time spent sick as a function of the degree k. Number of nodes used in this simulation $N = 1x10^5$, $t = 1x10^5$, $\tau = 0.1$.

their neighbors that are nearer to the center and thus are closer to the 'source' of infection.

Despite these variations due to the varying topologies Fig. 4 is still representative of the average time we would expect a node of connectivity k to be ill. We note that as we increase J the low connectivity nodes experience a dramatic decrease in the average time of illness, however the higher k ones are affected much less, with the highest nodes being unaffected at all. Increasing H however has the effect of decreasing the time throughout the whole range of connectivities, as we assumed when we introduced our model of awareness. It is interesting to note that the curves seen in Fig. 4 take on a sigmoid or a Gompertz curve like shape. Since we are considering a very large network of 100, 000 nodes there are very few nodes with extremely high connectivity. In a realistic model the maximum number of contacts (sufficient for the propagation of the virus) that an individual has in a given time period (whether it is days, weeks, months, etc) is strictly limited, and an individual with approx. 4000 contacts may seem quite unrealistic. In our study nodes of such high degree are included for completeness. In later studies we reduce the number of nodes for more realistic maximum connectivity. The following studies consider a scale free network of 2500 individuals.

The results in Fig. 4 agree with the findings of many other works, such as [8, 9] on the role of high connectivity nodes in maintaining the infection by acquiring it easily. A further finding is that these high connectivity nodes also are responsible for the transmissions that maintain the disease's prevalence. That is, the high degree nodes are also responsible for the majority of transmissions taking place in the scale-free network. Although we do not expect this to change in our model incorporating risk perception, we would like to explore how changes in the awareness affect their role. A plot of the number of average transmissions originating from a node of degree k is shown in Fig. 5. The values have been normalized so that they correspond to the same number of total transmissions. We discover that, although increasing the personal perception J reduces the number of transmissions from high connectivity nodes much more than their low degree counterparts, the overall shape of the curve remains unchanged. That is, despite the introduction of risk perception, high connectivity nodes will maintain their status as the main spreaders of disease. Meanwhile, increasing H has the effect of decreasing the number of transmissions equally across all connectivities, as expected. We would like to draw the readers' attention to the inset of Fig. 5, which shows the average total number of transmissions occurring over $t = 5000$ time steps of the simulations. We observe that the total number of transmissions occurring in that time window decreases dramatically, and would argue that, with the incorporation of risk perception, the number of transmissions occurring becomes more realistic for the population than it is if risk perception is not taken into account at all. As mentioned previously, even highly infectious diseases hardly ever grow into pandemics, which would not be true given the efficiency of scale-free networks at propagating disease [6, 7, 8, 9, 22]. There can be many causes for these discrepancies between the real world and the theoretical findings, but in our view two important ones are:

- Even if most real-world networks are scale free they are much more sophisticated than the classical BA model,

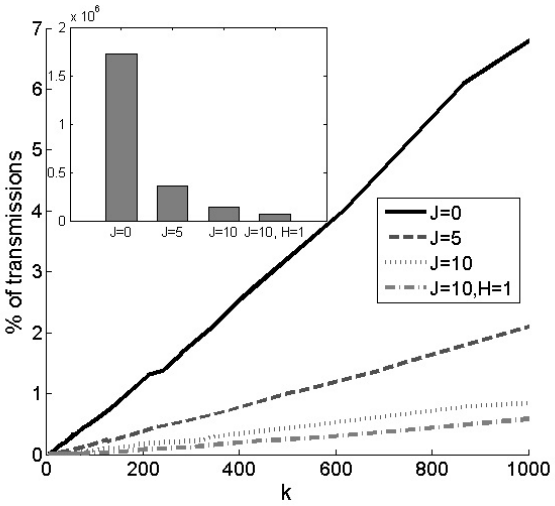

Figure 5: Percent of total transmissions attributable to a node of degree k, normalized over a common number of transmissions. Inset: total transmissions for each case. $N = 2500$, $t = 5000$, $\tau = 0.1$.

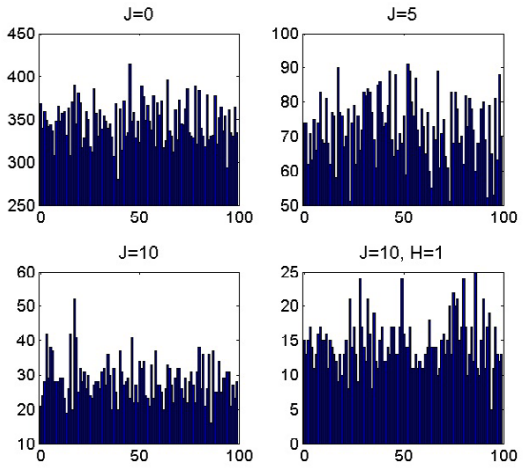

Figure 6: Number of new cases at time t for varying levels of risk perception within a 100 step window. $N = 2500$, $\tau = 0.1$.

as the processes that result in their creation are complicated and not limited to growth and preferential attachment (see [25])

- Risk perception plays an important role in the disease dynamics as people who are sick are likely to remain at home and healthy individuals are likely to avoid contact with people they perceive to be sick.

Examining Figs. 4 and 5 together we see that, despite the number of transmissions decreasing as a result of increasing risk perception, the higher connectivity nodes do not experience a drop in the time they spent as infected. This suggests that, as risk perception increases, the majority of transmissions in fact occur between the small number of high connectivity nodes, and only few occur in the rest of the network. This would be an important result as it would suggest that, in the presence of risk perception, the vast majority of the population would actually avoid infection, and only a small number of people with many social contacts would suffer from the disease. This means that even a highly infectious disease will not turn into an epidemic, and treatment would only be necessary for a small number of individuals. To test this we have further considered the number of new infections over time.

In epidemiology the force of infection λ is defined as the per capita number of new infections per time step [26]. We now quantify the impact that risk perception has on the force of infection. Fig. 6 shows the number of infections occurring within a small time window of 100 steps of the entire simulation, again for varying levels of risk perception. For increasing levels of risk perception we observe a significant decrease of the mean number of cases occurring as well as their variance, suggesting that the two are linked. Looking at the case of $J = 0$, and using the mean number of cases, $\lambda \approx 345/2500 = 0.138$ or 13.8% of the nodes in the network. Table 1 contains these approximations of all four cases considered in Fig. 6.

Looking at Table 1, for the last two cases we obtain a force of infection which corresponds to 1.08% and 0.6% of the nodes in a network. For such a low force of infection it is very likely that the number of new cases is attributable to the few high connectivity nodes, generated from the long tail of the power law probability distribution. Further steps that would be useful to confirm this is to visualize the network [24] and to determine nodes of which degrees and of what position within the network are involved in this infection spread.

Risk perception level	Mean number of infections/unit time (approx)	λ
J=0	345	0.138
J=5	75	0.03
J=10	27	0.0108
J=10, H=1	15	0.006

Table 1: Average force of infection for varying levels of risk perception.

4. Discussion and Conclusion

The modeling of human behavior contains many aspects and has been largely overlooked in epidemiological modeling. In this work we have studied an existing model of risk perception on a standard scale-free network constructed using the BA model. We have observed the effect that increasing the level of alertness of an individual, both privately and across the whole network, can have on the spread of an infectious disease. Although work has been done on connectivity-dependant transmission [23] and a re-emergence of threshold effects was noted, the method involved in our work depends not only on the connectivity of a node, but also on the state of its neighbors. Similarly to the works of Pastor-Satorras and Vespignani [6, 7, 8], we discover that the most highly connected nodes (sometimes referred to in epidemiology as the cores) are responsible for the majority of transmissions and for the maintenance of the infectious disease. However, with the incorporation of risk perception we now note that scale-free networks are no longer efficient at spreading the disease, and in fact, in some cases, the periphery of the network may remain largely unaffected. As well as causing varying transmission rates across the whole network, the risk perception allows us to reduce the infectivity τ, thus bringing down a potential pandemic in our theoretical model to a mild infection, affecting only a small fraction of the population.

Of course, however, modeling human behavior for epidemiology is not just limited to applying a measure for the level of alertness that an individual might have in the face of an epidemic outbreak. Of equal importance is the underlying social network, which in itself is produced as a result of complex human relationships. Although important to the field, scale-free networks have certain drawbacks that cannot be ignored. One such problem is that they do not allow for homophily, or the 'birds of a feather flock together' principle. In the real world individuals tend to form groups of contacts based on various sociological factors, such as personality, living area or workplace, ethnical origin, religion, income, etc. These groups can potentially be modeled as communities, and each community could be subjected to various levels of alertness or risk perception. In an accurate model the underlying social network should also certainly be dynamic over large periods of time: some people may change their contacts - that is they may lose old 'friends' and make new ones. The factors that result in the construction and evolution of social networks are many and are an active area of research that can greatly benefit epidemiologists.

Another important drawback to note is one that has been pointed out by Keller in [27]: even though we may be able to fit a power law to many networks occurring in the real world, regardless of how good or accurate the fit is, the process does not actually improve our knowledge of the actual topology of the network, how it came to be, or how it might evolve. As Christakis and Fowler [24] accurately point out visualizing complex topologies is important in aiding our understanding of the process of disease spread in a given network.

Despite the inaccuracies of scale-free networks their use is in fact very convenient. Gathering data to construct an accurate social network is a very long and complicated process. In many cases egos (the individuals under study) provide incomplete information about their alters (their contacts relevant for the study), either by accident or on purpose and in some cases alters may reside outside of the research area and their contribution may be ignored. When constructing a social network from real data you often need to collect data beyond the set of egos and also collect information from the alters that they have specified to avoid inaccuracies (e.g. 'one-way relationships') [28, 29]. Despite the difficulties however, to develop accurate epidemiological models for certain diseases constructing such empirical networks could be a necessary step.

Let us examine the case of the modeling of STDs as an example. As mentioned earlier, power law curves have been fit to the degree distributions of many networks of sexual partners [10], and one of the main models of spread of STDs involves the concept of a 'core': a group of individuals (e.g. sex workers) who are highly connected to the outside and spread the disease to the periphery or lower-activity part of the network. Some research has even suggested

that to prevent the spread of STDs preventative measures should target only those individuals as opposed to the entire population [30]. Some recent studies on STDs that have constructed empirical networks have however failed to locate such high-activity cores, putting into question the accuracy of this traditional model. Bearman et al [28] studied the sexual network of adolescents in a US high school, and discovered that its topology is that of a spanning tree with no cores. This topology was efficient for the diffusion of disease but very fragile, in that the removal of few links could greatly disjoint the structure. Attempting to reconstruct the network Bearman et al discovered that it was the result of some human (in paritcular, some potentially adolescent) attitudes: example factors are homophily and preventing the loss of social status in front of peers. The authors also point out that, in their case, preventative measures need to be applied across the entire subject population to be effective and that obtaining an accurate network topology is vital for designing efficient intervention techniques.

We would also like to give as an additional example the study of HIV spread in Likoma Island by Helleringer and Kohler [29]. The network that the authors constructed empirically in this study was much different to that of Bearman et al, in that it was very robust and that many individuals in it were members of a bicomponent, i.e. there were two or more independent paths through which they could acquire the infection. The network they discovered was still highly prone to the propagation of infection, despite the lack of high-activity cores. In fact they discovered that HIV prevalence was highest in the sparser regions of the network, contrary to the theoretical finding that the disease is maintained in the denser highly-connected regions [8]. This was in fact due to socioeconomic factors: the individuals in the sparser regions were those that are more likely to have been infected and whose exposure to risk factors was in fact higher. The authors also pointed out that changes in the network topology over time may be responsible for the distribution of HIV that they observed. By studying two completely different groups of people, the authors of those two works discovered network topologies that were not only very different from the traditional models, but were completely different to each other as well. Populations belonging to different social groups can in fact have a very different social network structure. Considering an accurate network to represent the underlying population when constructing an epidemiological model is not only important for obtaining accurate results, but it is also in itself a way of incorporating human behavior into the model.

In this work we have studied how an individual's risk perception can alter the spread of disease in a network topology that had previously been studied extensively without this consideration. We obtained our results through both a mean field approximation and numerical simulations. Our aim was to demonstrate the benefit of incorporating human behavior in epidemiological modeling. Since social network topologies are also largely the product of human behavior we also gave examples of two separate cases in which the network, constructed from real-world data, did not have the expected theoretical or traditional topology or behavior. In conclusion an alertness measure such as the one we have examined here may be insufficient to truly model human behavior. To do so it would be necessary to generate an appropriate network to represent the population, which in itself requires some consideration of the complex processes of human behavior that generated it. When constructing such a network with the aim to study the spread of infectious diseases it may further be necessary to reflect on some of the sociological factors that are relevant to the disease under consideration.

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