Relating Network Structure to Diffusion Properties through Stochastic Dominance

by Matthew O. Jackson and Brian W. Rogers *

Draft: December 15, 2006 Forthcoming in Advances in Economic Theory[†]

Abstract

We examine the spread of a disease or behavior through a social network. In particular, we analyze how infection rates depend on the distribution of degrees (numbers of links) among the nodes in the network. We introduce new techniques using first- and second order stochastic dominance relationships of the degree distribution in order to compare infection rates across different social networks.

JEL Classification Numbers: D85, A14, C71, C72.

Keywords: Diffusion, Infection, SIS, Networks, Social Networks

^{*}Jackson: Department Stanford Unversity, ofEconomics, ifornia 94305, USA; Rogers: Managerial Economics and Decision Sciences (MEDS), Kellogg School of Management, 2001 Sheridan Rd., Jacobs Center 5th Floor, Evanston, IL 60208-2009, USA; emails: jacksonm@stanford.edu and brogers@kellogg.northwestern.edu; web sites: http://www.stanford.edu/~jacksonm and www.kellogg.northwestern.edu/faculty/rogers_b/personal/. We gratefully acknowledge financial support under NSF grant SES-0316493, the Lee Center for Advanced Networking, and a SISL/IST fellowship. We thank Dunia Lopez-Pintado for helpful comments and conversations.

[†]This paper appeared previously as part of "Search vs Random Attachment and the Formation of Large Networks" by the same authors. That paper was split, with part becoming this paper and the other part being Jackson and Rogers [4].

1 Introduction

Social and economic networks exhibit widely different characteristics across different applications. In particular, they differ both in the average numbers of links that individuals have as well as how those links are distributed across the population. The patterns of links across the population can have a significant impact on the transmission of diseases, ideas, beliefs, and other behaviors through the group. Applications in which network structures have been shown to influence diffusion dynamics include the spreading of infectious disease, computer viruses, rumors and information, and individual consumer and strategic choices. How do the specific characteristics of interaction systems map into the likelihood and prevalence with which an infection or behavior spreads through a population?

Pastor-Satorras and Vespignani [9], [10], [11], Newman [8], Sander et al [13], and Lopez-Pintado [7], among others, have addressed this question by enriching models of infection (e.g., Bailey [1]) to explicitly include network structure as modeled through degree distributions.¹ These approaches often work through solving for, estimating, or simulating infection rates for particular network structures.

Here, we provide a new approach that allows us to order infection rates based on stochastic dominance relationships of the degree distributions. This allows us to derive very general orderings over the rates of the adoption of a behavior or infection rates as related to degree distributions, without having to specify the details of the network structure.

The stochastic process governing diffusion is modeled as one where the behavior or state of an individual changes over time depending on the behavior or state of an individual's neighbors. Strictly speaking, we examine the "SIS" model that has been used to investigate spread of diseases. In this model, a node changes from being "susceptible" to "infected" with a probability that is proportional to the number of neighbors who are infected, and recovers from being "infected" to being "susceptible" at random. While the study of diseases is clearly important, these dynamics can also be interpreted as a model of behavior where an individual adopts an action, such as using a new product, with a probability proportional to the number of neighbors currently using it, with a possibility of reverting to a status quo behavior. In this sense our results provide a method of analyzing the diffusion of behavior in strategic settings. Despite the importance of such applications, we stress

¹The degree of a node is the number of links that it has, and the degree distribution is simply the frequency distribution of the degrees of the nodes in the network.

that the techniques we have developed to analyze the diffusion model should be useful more generally.

The main tools that we use are stochastic dominance relationships. Our results analyze how infection rates and thresholds for persistent infection vary as the network structure is changed in the sense of stochastic dominance. When one increases a degree distribution in the sense of first order stochastic dominance, there are more connections in the network, thereby facilitating infection. If one holds average connectivity constant and increases a network's heterogeneity in degrees in the sense of a mean-preserving spread (which is inverse to second order stochastic dominance), then there are more nodes with high and low degrees. The high degree nodes, or "hubs," serve as conduits for infection which increases the chance that a given node will become infected by any one of its neighbors. How this translates into average infection rates is more subtle, and depends on the transmission process in ways that we cleanly document for some families of distributions.

While these results are interesting in their own right, much of the usefulness of our methods derives from the observation that many processes governing network formation exhibit stochastic dominance relationships as parameters describing the process are varied. In such cases the above results can be immediately applied, which provides an understanding of how micro-level determinants of linking decisions translate into the probability of reaching an infected node through paths in the network. Indeed, since the first circulation of this paper, the techniques developed here regarding the use of stochastic dominance relations have been applied to analyze diffusion in a variety of settings (e.g., Jackson and Yariv [5], [6], Lopez-Pintado [7], Galeotti et al [3], Galeotti and Vega-Redondo [2]).

2 The Model and Stochastic Dominance Result

2.1 The SIS Model

The SIS model, adapted from the version of Pastor-Satorras and Vespignani [9], is as follows.²

A network has nodes that exist in two different states: "infected" and "susceptible". Nodes randomly transition between states in ways that partly

²In this model, nodes can become infected multiple times, as with catching the common cold. This differs from situations where recovered nodes can no longer be infected, which is generally studied under the alternative "SIR" model.

depend on the states of their neighbors in the social network. A susceptible node i with degree d_i becomes infected in a given period with a probability $\nu(d_i\theta_i+x)$, where $\nu\in(0,1)$ is a parameter describing a rate of infection transmission, d_i is a nonnegative integer representing the degree of the node, $\theta_i\in[0,1]$ is the fraction of i's neighbors that are infected, and x is a nonnegative scalar (which can be set to 0) representing the rate at which infection can occur independently of the social context.³ An infected node recovers to become "susceptible" in a given period with a probability $\delta\in(0,1)$. Let $\lambda=\nu/\delta$ be the ratio of the infection transmission rate to the recovery rate.

This process is a reasonable description of the spread of a virus like the common cold through a population, or a computer virus, etc. As described above, it can also be viewed as a model of the diffusion of behavior in strategic interactions. For example, consider a situation where individuals choose between two behaviors, with one thought of as a status quo and labeled "susceptible." Agents are matched with a randomly chosen neighbor. If the neighbor has adopted the "new" behavior (i.e., become "infected"), then the agent does as well. In addition, they randomly revert to the status quo strategy with some tremble δ . Thus if a small set of individuals perturbs their play to "infected," the behavior could potentially spread through the network. We analyze the long-run implications for the diffusion of such a behavior as a function of the degree distribution of the network.

Let P represent the distribution of degrees across nodes in the society, where P(d) is the probability that a randomly chosen node has degree d. Let $\rho(d)$ denote the average infection rate among nodes with degree d, and ρ be the average infection rate in the society: $\rho = \sum_{d} \rho(d) P(d)$. The probability that a node at the end of a randomly chosen link in the society is infected is then

$$\theta = \frac{\sum_{d} \rho(d) P(d) d}{m},\tag{1}$$

where $m = \sum_{d} P(d)d$ is the average degree, which is presumed to be greater than 0.4 We call θ the average neighbor infection rate.

³For small ν , the first expression is an approximation of becoming infected independently by any infected neighbor. The term x does not appear in previous analyses.

⁴Note that θ differs from the average infection rate across nodes, ρ , as it is degree-weighted. The rate θ conditions on the fact that the node in question lies at the end of a link. The weighting by d captures (via Bayes' rule) the idea that nodes with higher degrees are more likely to be found by tracing a random link. Embodied in this calculation is an independence of degrees across nodes, which requires an infinite network to justify exactly.

2.2 A Mean-Field Approximation and Steady-States

Given a specification of a network and an initial state the above diffusion mechanism is a well-defined Markov process. Since such a process is too complex to analyze directly under arbitrary network structures, we follow the usual technique of analyzing a mean-field approximation to the process. In the mean-field approximations infection rate changes among nodes of a given degree, $\rho(d)$, are approximated by a deterministic continuous time process. Each node is presumed to have a fraction of neighbors who are infected that matches the population average. Steady-states of the system are found by setting the change in average infection rates over time to zero. Specifically, the change in the average infection rate of nodes of degree d per unit time under the mean-field approximation is

$$\frac{\partial \rho(d)}{\partial t} = (1 - \rho(d)) \nu(\theta d + x) - \rho(d)\delta.$$

The first part is the fraction of susceptible nodes that become infected and the second part is the fraction of infected nodes that recover to become susceptible. Steady-state corresponds to $\partial \rho(d)/\partial t = 0$ for all d, which leads to

$$\rho(d) = \frac{\lambda(\theta d + x)}{1 + \lambda(\theta d + x)}. (2)$$

Substituting (2) into the definition of θ in (1), we obtain

$$\theta = \frac{1}{m} \sum_{d} \frac{\lambda(\theta d^2 + xd)P(d)}{1 + \lambda(\theta d + x)}.$$
 (3)

For any given degree distribution, there may exist multiple steady-states. For instance, if x=0 then $\theta=\rho=0$ is always a steady-state infection level, but there can also exist a higher steady-state level. In particular, as we discuss below, there is either a unique steady-state infection level, or else there are exactly two steady-state infection levels: one at 0 and another positive one. Let $\overline{\theta}$, $\overline{\rho}(d)$ and $\overline{\rho}$ denote the largest steady-state average neighbor infection level, infection level as a function of degree, and overall average infection level.

For the remainder of the analysis, all statements refer to mean-field approximations.

2.3 Existence of Non-Zero Steady-State Infection Rates

Let

$$H_P(\theta) = \sum_{d} \frac{P(d)\lambda(d^2\theta + xd)}{m(1 + \lambda(d\theta + x))}.$$

Fixed points of H_P correspond to steady-state (neighbor) infection levels for a distribution P; and given θ , the corresponding $\rho(d)$'s and ρ are uniquely determined.

Note that H_P is strictly increasing and strictly concave in θ and has range in [0,1]. Note also that $H_P(0) = 0$ if and only if x = 0. Thus, it follows that if x > 0, then there is a unique steady state θ which is greater than 0; and if x = 0, then there is another steady state above $\theta = 0$ if and only if $H'_P(0) > 1$. Let us check when this holds.

$$H'_P(\theta) = \sum_{d} \frac{P(d)\lambda d^2}{m(1 + \lambda(d\theta + x))^2}.$$

Thus, when x = 0

$$H_P'(0) = \frac{\lambda E_P[d^2]}{E_P[d]}.$$

Therefore, in the case where x=0 there is a steady-state at 0, and there is also a second steady-state $\bar{\theta} > 0$ (and a corresponding largest steady-state average infection rate $\bar{\rho} > 0$) if and only if $\lambda > \frac{E_P[d]}{E_P[d^2]}$. To summarize:

PROPOSITION 1 Consider the mean-field approximated steady-state infection rate for a given degree distribution P.

- If x > 0, then there is a single steady-state infection rate and $\overline{\theta} > 0$.
- If x = 0 and $\lambda \leq \frac{E_P[d]}{E_P[d^2]}$, then there is a single steady-state infection rate and $\overline{\theta} = 0$.
- If x = 0 and $\lambda > \frac{E_P[d]}{E_P[d^2]}$, then there are two steady-state infection rates: 0 and another where $\overline{\theta} > 0$.

This defines a so-called phase transition (in the case where x=0), where infection can spread in a society if and only if $\lambda > \frac{E_P[d]}{E_P[d^2]}$. In the case of a scale-free network, where the degree distribution follows a power law and $E_P[d^2]$ is infinite, this means that infection can spread regardless of the infection rate (see Pastor-Satorras and Vespignani [9]). In the case of a Poisson random network this requires $\lambda > \frac{1}{1+m}$, where m is the average degree.

2.4 Stochastic Dominance, Changes in Networks, and Relative Infection Rates

We now examine how the largest steady state infection level changes as the network is changed, as captured through stochastic dominance shifts of the degree distribution.

A distribution P' strictly first order stochastically dominates another distribution P if $\sum_{d=0}^{Y} P'(d) \leq \sum_{d=0}^{Y} P(d)$ for all Y, with strict inequality for some Y. That is, nodes' degrees are systematically higher under P' than under P. If P' strictly first order stochastically dominates P, then the expectation of a strictly increasing function will be higher under P' than under P.

A distribution P' is a strict mean-preserving spread of another distribution P if it has the same mean and $\sum_{Y=0}^{Z} [\sum_{d=0}^{Y} P'(d) - P(d)] \ge 0$ for all Z, with strict inequality for some Z. If P' is a strict mean-preserving spread of another distribution P, then it leads to a higher expectation of any strictly convex function.

THEOREM 1 Consider two distributions P' and P, with corresponding largest steady-state average neighbor infection rates $\overline{\theta}'$ and $\overline{\theta}$, and largest steady-state overall average infection rates $\overline{\rho}'$ and $\overline{\rho}$; and suppose that $\overline{\theta} > 0$.

- (I) If P' and $P'(d)d/E_{P'}[d]$ strictly first order stochastic dominate P and $P(d)d/E_P[d]$, respectively, then $\overline{\theta}' > \overline{\theta}$ and $\overline{\rho}' > \overline{\rho}$.
- (II) If P' is a strict mean-preserving spread of P, then $\overline{\theta}' > \overline{\theta}$.

Theorem 1 shows that shifts in the distribution in terms of either a strict first order stochastic dominance shift or a strict mean-preserving spread lead to increases in the largest (nonzero) steady-state neighbor infection rates, and sometimes average infection rates. The result only applies when there is a nonzero steady state. As we saw in the previous section, there can be situations where the only steady-state infection rates are 0.

It is important to remark that while part (I) orders both the neighbor infection rate and the overall average infection rate, part (II) only makes a claim about the neighbor infection rate. This is because the overall infection rate can go either up or down when one considers a mean-preserving spread of a distribution. In order to conclude that the overall average infection rate goes up (or down), we need to focus on specific families of distributions.

This issue is illustrated in the following example.

Example 1

Let $\lambda=1$ and x=0. Let P be a distribution with weight 1 on degree d=3 and P' be a distribution with even weight on degrees d=3 and d=4, while P'' is a distribution with even weight on degrees d=2 and d=4. So, P' strictly first order stochastic dominates P and P'' is a strict mean-preserving spread of P. We can solve (3) to find the nonzero steady state values of θ for each distribution. For instance, for P this simply becomes

$$\theta = \frac{9\theta}{3(1+3\theta)},$$

which has solutions 0 and 2/3. So, $\overline{\theta} = \overline{\rho} = \overline{\rho}(3) = 2/3$.

We can do the same calculations for P'. Here we find that $\overline{\theta}' = .716$ while $\overline{\rho}' = .712$. Furthermore, $\rho(4) = .741$ while $\rho(3) = .682$. So each of the steady state values has increased.

However, performing the same calculation for P'' we find that $\overline{\theta}'' = .679$ while $\overline{\rho}'' = .654$. Here $\rho(4) = .731$, while $\rho(2) = .576$. If we look at any node and trace one of its links, the neighbor we find is twice as likely to be of degree 4 than 2 (as degree 4 nodes have twice as many links in the network). This accounts for the higher chance that a given neighbor is infected. Yet, the overall infection rate in the society has still gone down, which is due to the fact that the infection rate among nodes with degree 2 is relatively low.

2.5 An Application to a Class of Degree Distributions

If we impose more structure on the class of degree distributions admitted, then we can order the overall average infection rates when we examine meanpreserving spreads.

Here we examine a family of degree distributions from a model of Jackson and Rogers [4], which fits well across a wide variety of social networks. This class of distributions is general enough to include both scale-free networks and networks formed uniformly at random as extreme cases. The degree distribution has a cumulative distribution function of ⁵

$$F(d) = 1 - \left(\frac{rm}{d + rm}\right)^{1+r},\tag{4}$$

⁵Jackson and Rogers [4] also allow for a minimum degree in the distribution. Here, to keep things uncluttered, we only examine the case where it is 0.

where m is average degree and r is a parameter taking values strictly between 0 and infinity. The distribution approaches a scale-free distribution or exponential distribution,⁶ respectively, as r tends to these extremes.⁷

First, let us examine the threshold infection transmission rate λ below which the steady-state average infection rate in the population is zero. Here, $E_F[d^2] = 2rm^2/(r-1)$ when r > 1 and it is infinite when $r \leq 1$.⁸ Then applying Proposition 1 we obtain the following.

COROLLARY 1 Consider a network with a degree distribution described by (4), and suppose that x = 0. The minimal λ in order to have a positive steady-state infection rate is 0 if $r \le 1$ and (r-1)/(2rm) when r > 1.

The threshold λ to have positive infection is (weakly) increasing in r, so that higher effective spreading rates are required to achieve a positive infection rate in the steady-state for networks with degree distributions generated from higher r. The cutoff is also decreasing in m, fixing r. This has a straightforward intuition as increasing average degree increases connectivity in the sense of first order stochastic dominance (Theorem 2), so that the cutoff rate is lowered and the steady state infection rate is increased, holding all else constant.

Now let us explore the largest steady-state average infection levels.

A nice property of the family of degree distributions in (4) is that they can be ordered in the sense of stochastic dominance as r and m are varied.

THEOREM 2 [Jackson and Rogers [4]] Consider a distribution F with parameters (m,r) and a distribution F' with parameters (m',r'), where both distributions satisfy (4) relative to their corresponding parameters.

- (I) If r = r' > 0 and m' > m, then F' strictly first order stochastic dominates F.
- (II) If m = m' > 0 and r' < r, then F' is a strict mean preserving spread of F.

⁶The exponential distribution, $F(d) = 1 - \exp(-d/m)$, corresponds to the degree distribution of a growing network where new nodes pick existing nodes uniformly at random to form links with. It is a growing adaptation of a Poisson random network.

⁷The distribution in (4) derives from an approximation to a growing network system in which degree is viewed as a continuously-valued quantity.

 $^{^8}E_P[d^2] = \int_0^\infty d^2(1+r)(rm)^{1+r}(d+rm)^{-2-r}dd$. Integrating twice by parts yields the expression.

The fact that the degree distributions obey stochastic dominance relationships with respect to the network formation parameters allows one to apply the results from the SIS model to this particular class of distributions. As a direct consequence of Theorems 1 and 2 (noting that the proof of Theorem 2 from Jackson and Rogers [4] is easily extended to show stochastic dominance of the cumulative distribution function corresponding to f(d)d/m as m is increased by showing that the cdf is decreasing in m for all d > 0), we have the following.

COROLLARY 2 Consider a distribution F with parameters (m,r) and a distribution F' with parameters (m',r'), where both distributions satisfy (4) relative to their corresponding parameters. Let the corresponding largest steady-state average neighbor infection rates be $\overline{\theta}'$ and $\overline{\theta}$, and the largest steady-state overall average infection rates be $\overline{\rho}'$ and $\overline{\rho}$, respectively; and suppose that $\overline{\theta} > 0$.

(I) If
$$r = r' > 0$$
 and $m' > m$, then $\overline{\theta}' > \overline{\theta}$ and $\overline{\rho}' > \overline{\rho}$.

(II) If
$$m = m' > 0$$
 and $r' < r$, then then $\overline{\theta}' > \overline{\theta}$.

This still leaves open the question of the relative ranking of $\overline{\rho}'$ and $\overline{\rho}$ when F' is a strict mean-preserving spread of F. We now examine that question.

PROPOSITION 2 Consider a distribution F with parameters (m,r) and a distribution F' with parameters (m',r'), where both distributions satisfy (4) relative to their corresponding parameters and have non-zero maximal steady-state infection levels. If m = m' > 0 and r' < r, then there exist $\underline{\lambda}$ and $\overline{\lambda}$ such that:

- If $\frac{\nu}{\delta} < \underline{\lambda}$ and x is low enough, then the steady-state average infection rate under a mean-field approximation is higher under r' than under r.
- If $\frac{\nu}{\delta} > \overline{\lambda}$, then the steady-state average infection rate under a mean-field approximation is lower under r' than under r.

Thus, we can order the average infection rates, but which way they are ordered reverses depending on whether the infection transmission rate λ is high or low.

We remark that this is in contrast to what is suggested by much of the previous literature which has focussed on the threshold λ 's for which one has a positive infection rate, rather than on the maximal infection rate. For high

Infection and Network Structure

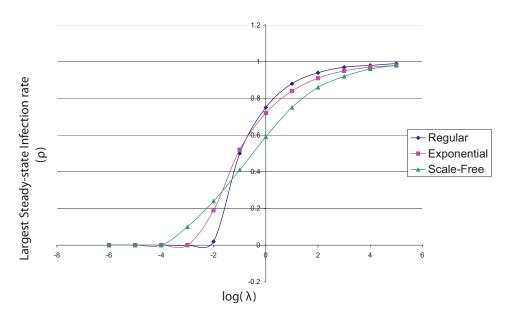


Figure 1: Steady-state infection rate, ρ , as a function of λ for three degree distributions.

 λ 's it is the more regular networks that lead to higher infection rates, rather than the more heterogeneous networks such as those following a power law.

The relationship is captured in Figure 1, which illustrates the largest steady-state infection rate for three different network structures as a function of λ . The three network structures all have the same mean degree of 4, and are a regular network, an exponential random network (corresponding to $r = \infty$), and a scale-free network (corresponding to r = 0). The scale-free distribution is a strict mean-preserving spread of the exponential which is a strict mean-preserving spread of the regular network.

The intuition behind this result can be expressed as follows. The change in infection rate due to a change in the degree distribution comes from countervailing sources, as more extreme distributions have relatively more very high degree nodes and very low degree nodes. Very high degree nodes have high infection rates and serve as conduits for infection, thus putting upward pressure on average infection. Very low degree nodes have fewer neighbors

⁹The figures are based on degree distributions that are adjusted to have maximal degree of 20, and then the ρ is calculated by iterating to find fixed points θ for a grid of λ 's.

to become infected by and thus have relatively low infection rates. Which of these two forces is the more important one depends on the ratio $\lambda = \frac{\nu}{\delta}$, i.e., the effective spreading rate. For low λ , the first effect is the more important one, as nodes recover relatively rapidly, and so there must be nodes with many neighbors in order keep the infection from dying out. In contrast, when λ is high, then nodes become infected more quickly than they recover. Here the more important effect is the second one, as most nodes tend to have high infection rates, and so how many neighbors a given node has is more important than how well those neighbors are connected.

3 Appendix

Let

$$H_P(\theta) = \sum_{d} \frac{P(d)\lambda(d^2\theta + xd)}{m(1 + \lambda(d\theta + x))}.$$

Fixed points of H_P correspond to steady-state neighbor infection levels for a distribution P.

Proof of Theorem 1: First, note that

$$H_{P'}(1) = \sum \frac{P'(d)\lambda(d^2 + x)}{m(1 + \lambda(d + x))} < \sum \frac{P'(d)(d^2 + x)}{m(d + x)} \le \sum \frac{P(d)d^2}{md} = 1.$$

Thus, since $\overline{\theta}'$ is the largest θ such that $\theta = H_{P'}(\theta)$, and we know that $1 > H_{P'}(1)$, it follows that $\theta > H_{P'}(\theta)$ for all $\theta \in (\overline{\theta}', 1]$.

So, suppose to the contrary of the theorem under either (I) or (II), that $\overline{\theta}' \leq \overline{\theta}$. Then it follows that from the above that

$$\overline{\theta} \ge H_{P'}(\overline{\theta}). \tag{5}$$

Since $\lambda(d^2\theta + x)/(1 + \lambda(d\theta + x))$ is strictly increasing and strictly convex in d when $\theta > 0$, it follows from either strict first order stochastic dominance of P'(d)d/m' over P(d)d/m, or that P' is a mean-preserving spread of P, that $H_{P'}(\theta) > H_P(\theta)$ for any $\theta > 0$. Thus, since $\overline{\theta} > 0$, it follows from (5) that

$$\overline{\theta} \ge H_{P'}(\overline{\theta}) > H_P(\overline{\theta}),$$

contradicting the fact that $\overline{\theta} = H_P(\overline{\theta})$.

Thus, under either (I) or (II), $\overline{\theta}' > \overline{\theta}$.

Let us now argue that in case (I) $\overline{\rho}' > \overline{\rho}$.

¿From (2), $\rho(d) = \frac{\lambda(\theta d + x)}{1 + \lambda(\theta d + x)}$. Since this is a strictly increasing function of θ , $\overline{\rho}'(d) > \overline{\rho}(d)$ for any d > 0. This implies that

$$\overline{\rho}' = \sum_{d} \overline{\rho}'(d) P'(d) > \sum_{d} \overline{\rho}(d) P'(d). \tag{6}$$

Strict first order stochastic dominance and the fact that $\rho(d) = \frac{\lambda(\theta d + x)}{1 + \lambda(\theta d + x)}$ is strictly increasing in d implies that

$$\sum_{d} \overline{\rho}(d)P'(d) > \sum_{d} \overline{\rho}(d)P(d) = \overline{\rho}. \tag{7}$$

The result follows from (6) and (7).

Proof of Proposition 2: Multiplying both sides of (2) by $1 + \lambda(\theta d + x)$ and integrating with respect to F(d), we obtain

$$\rho = \frac{\lambda \theta m (1 - \theta) + \lambda x}{1 + \lambda x}.$$
 (8)

We now use Theorem 1, which states that if F' is a strict mean-preserving spread of F, then the corresponding $\overline{\theta}' > \overline{\theta}$.

¿From (8), we know that ρ is increasing in θ when θ is below 1/2; but decreasing when it is above 1/2.

¿From (3), we know that θ is near zero for low λ and low x, and near one for large enough λ , for any given P. Given r and r', we can then find bounds on λ and x below which both $\overline{\theta}$ and $\overline{\theta}'$ are below 1/2, and a corresponding bound on λ above which both $\overline{\theta}$ and $\overline{\theta}'$ are above 1/2. The proposition then follows from the fact that $\overline{\theta}' > \overline{\theta}$, and the increasing or decreasing nature of (8), depending on whether the corresponding θ is above or below 1/2.

References

- [1] Bailey, N.T.J. (1975) The Mathematical Theory of Infectious Diseases, Griffin: London.
- [2] Galeotti, A. and F. Vega-Redondo (2006) "Strategic Analysis in Complex Networks with Local Externalities," mimeo: University of Essex
- [3] Galeotti, S. M.O. Vega-A., Goyal, Jackson, and L. Yariv (2005)"Network Games," Redondo. http://www.stanford.edu/~jacksonm/netgames.pdf

- M.O., and B.W. Rogers (2006) "Meeting Strangers Friends of Friends: How Random Are Social Netand works?" forthcoming in the American Economic Review, http://www.stanford.edu/~jacksonm/netpower.pdf.
- [5] Jackson, M.O. and L. Yariv (2005) "Diffusion on Social Networks," Économie Publique, Numéro 16, pp 3-16, 2005/1.
- [6] Jackson, M.O. and L. Yariv (2006) "The Diffusion of Behavior and Equilibrium Structure on Social Networks," forthcoming: American Economic Review (papers and proceedings), preprint: http://www.stanford.edu/~jacksonm/netbehavior.pdf
- [7] Lopez-Pintado, D. (2006) "Diffusion in Complex Social Networks," working paper: Columbia University.
- [8] Newman, M.E.J. (2002) "Spread of Epidemic Diseases on Networks," *Physical Review E*, 66, 016128.
- [9] Pastor-Satorras, R. and A. Vespignani (2001) "Epidemic Spreading in Scale-Free Networks," *Physical Review Letters*, 86:14, 3200-3203.
- [10] Pastor-Satorras, R. and A. Vespignani (2001) "Epidemic dynamics and endemic states in complex networks," *Phys. Rev. E* **63**, 066117.
- [11] Pastor-Satorras, R. and A. Vespignani (2002) "Immunization of complex networks" *Phys. Rev. E* **65**, 036104.
- [12] Rothschild, M. and J. Stiglitz (1970) "Increasing Risk: I. A Definition," *Journal of Economic Theory* 2: 225-243.
- [13] Sander, L.M., C.P. Warren, I.M. Sokolov, C. Simon, J. Koopman (2002) "Percolation on heterogeneous networks as a model for epidemics," *Mathematical Biosciences*, 180, 293–305.