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Linking Network Structure and Diffusion through Stochastic Dominance

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Abstract

Recent research identifies stochastic dominance as critical for understanding the relationship between network structure and diffusion. This paper introduces the concept of stochastic dominance, explains the theory linking stochastic dominance and diffusion, and applies this theory to a number of diffusion studies in the literature. The paper illustrates how the theory connects observations from different disciplines, and details when and how those observations can be generalized to broader classes of networks.

Key words: social networks, diffusion, stochastic dominance

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1. Introduction

Network structure affects the speed and extent to which information, disease, behavior, and innovations diffuse (Abrahamson and Rosenkopf, 1997; Newman, 2002; Sander et al., 2002; Cowan and Jonard, 2004; Young, 2006; Ohtsuki et al., 2006; Centola and Macy, 2007). Often there is an abrupt transition from those networks in which the diffusion process dies out completely to those in which it envelops the network. Our understanding of the relationship between network structure and diffusion is built on observations from different disciplines, which employ different techniques and consider different families of networks leaving an array of similar results with no formal connection.

Broadly speaking, there seems to be a greater tendency towards diffusion in networks that are “more random.” Underlying this observation are a range of studies employing the Watts-Strogatz family of networks (Watts and Strogatz, 1998), which interpolate between random and regular networks. However, without a general theory we cannot extrapolate from these studies to conclude that “increasing randomness” promotes diffusion in all networks.

Recently, several scholars have demonstrated how stochastic dominance can be used to order networks according to their proclivity to sustain diffusion (Jackson and Yariv, 2005, 2007; Jackson and Rogers, 2007; López-Pintado, 2008; Galeotti et al., 2009). These results explain the propensity for diffusion in the more random Watts-Strogatz networks and provide a means for understanding diffusion in more general networks. The theory reveals that increasing randomness does not always increase diffusion; instead, the relationship is conditional on the form of local reinforcement in the diffusion process.

Use of the concept of stochastic dominance (Rothschild and Stiglitz, 1970) has largely been confined to the theoretical economics and finance literatures, and as such may be unfamiliar to scholars in many of the growing number of fields that study networks. Additionally, proofs of the main results relating stochastic dominance and diffusion rely on a mean-field approach borrowed from statistical mechanics which may be unfamiliar to many social scientists. The aim of this article is threefold. First, we wish to introduce the concepts of stochastic dominance to a broader audience of scholars interested in network analysis. Second, we seek to explain and provide intuition for the recent results relating stochastic dominance and diffusion without requiring the technical expertise to parse the mean-field arguments. And third, we indicate how the stochastic dominance results connect independent observations from a variety of network diffusion studies as well as when and

how these observations can be generalized.

2. Stochastic Dominance

To impose order on the bewildering number of possible networks, theorists have constructed a long list of methods for measuring and categorizing them. Networks can be bipartite, star-shaped, scale-free, regular, Eulerian, hamiltonian, small-world, connected, planar, or sparse. Each network has a girth, diameter, cyclomatic number, chromatic number, centralization, density, average path length, average degree, clustering coefficient, and fraction of transitive triples.¹ Among all of these features, the degree distribution of a network plays a key role in diffusion.

The *degree* of a node in a network is simply the number of edges connected to that node. The *degree distribution* of the network is the probability distribution P defined by setting $P(d)$ equal to the fraction of degree d nodes in the network. We will be interested in ordering degree distributions in two ways. A distribution P *first order stochastically dominates* (FOSD) a distribution P' if

$$\sum_{d=0}^D P(d) \leq \sum_{d=0}^D P'(d) \tag{1}$$

for all D . The dominance is strict if the inequality is strict for at least some D . Equivalently, P FOSD P' if for every nondecreasing function $u : \mathbb{R} \rightarrow \mathbb{R}$,

$$\sum_{d=0}^{D_{max}} u(d)P'(d) \leq \sum_{d=0}^{D_{max}} u(d)P(d), \tag{2}$$

where D_{max} is the maximum degree of any node in the network. The notion of stochastic dominance is most familiar in the context of the valuation of risky assets. If P and P' are two lotteries then P FOSD P' if the expected payoff from P is greater than the expected payoff from P' for any nondecreasing utility function u .

We say that a network Γ first order stochastically dominates a network Γ' if the degree distribution for Γ FOSD the degree distribution of Γ' . In general, we think of a network that first order stochastically dominates another as having more edges. In particular, setting $u(d) = d$ in equation (2) implies that if Γ FOSD Γ' , then the average degree of Γ is greater than the average degree of Γ' .

¹See the books by Diestel (2000) and Jackson (2008) for definitions.

We will also be interested in ordering networks by second order stochastic dominance. A distribution P *second order stochastically dominates* (SOSD) a distribution P' if

$$\sum_{C=0}^D \left(\sum_{d=0}^C P(d) \right) \leq \sum_{C=0}^D \left(\sum_{d=0}^C P'(d) \right) \quad (3)$$

for all D . The dominance is strict if the inequality is strict for at least some D . Equivalently, P SOSD P' if for every nondecreasing concave function $u : \mathbb{R} \rightarrow \mathbb{R}$,

$$\sum_{d=0}^{D_{max}} u(d)P'(d) \leq \sum_{d=0}^{D_{max}} u(d)P(d). \quad (4)$$

FOSD implies SOSD, but not vice versa. If P and P' have the same mean then P SOSD P' is equivalent to P' is a *mean preserving spread* of P . As with first order stochastic dominance, second order stochastic dominance has an interpretation in terms of risk: if a lottery P SOSD a lottery P' then any risk averse individual prefers P to P' .

We say that a network Γ second order stochastically dominates a network Γ' if the degree distribution for Γ SOSD the degree distribution of Γ' , and Γ' is a mean preserving spread of Γ if the same relationship holds for their degree distributions. We can think of a network that is a mean preserving spread of another as having the same average degree but greater variation in the degree.

Figure 1 depicts two networks Γ and Γ' , and Figure 2 plots their degree distributions.² Both networks have the same number of nodes and edges, and thus the same average degree, but Γ' is a mean preserving spread of Γ . Second order stochastic dominance can be used to order commonly studied network families: assuming the same average degree, a scale-free (or power law) network is a mean preserving spread of an exponential network, which is a mean preserving spread of a Poisson network, which is a mean preserving spread of a regular network.

3. Diffusion

Many studies address the impact of network structure on diffusion (Abrahamson and Rosenkopf, 1997; Watts, 1999, 2002; Chwe, 2000; Morris, 2000; Pastor-Satorras and Vespignani, 2001a,b; Newman, 2002; Sander et al., 2002; Young, 2006; Cowan and Jonard, 2004; Centola et al., 2005); however, the critical role of stochastic dominance was identified relatively recently (Jackson and Yariv, 2005,

²Figure 1 and all network computations were made in R using the statnet package (Handcock et al., 2003).

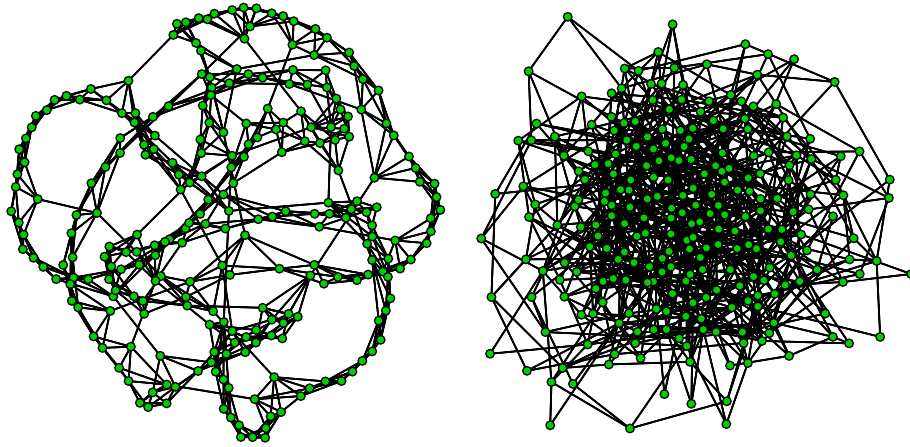


Figure 1: Two networks, Γ (left) and Γ' (right) with the same number of nodes and edges, but different degree distributions. Γ' is a mean preserving spread of Γ . Both networks are Watts-Strogatz networks with $N = 250$ and $k = 3$ (see the section *Watts-Strogatz Small-worlds* below). For Γ $p = .05$, and for Γ' $p = .9$.

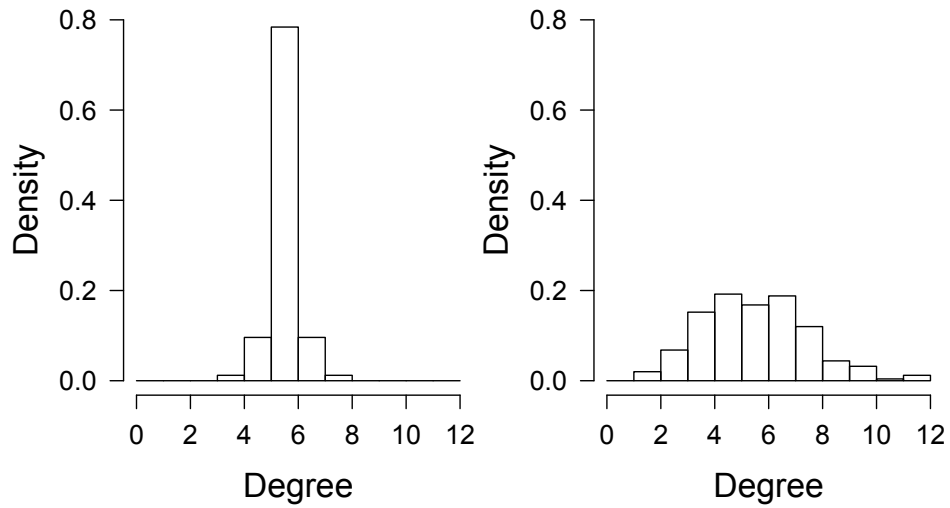


Figure 2: The degree distributions for the networks Γ (left) and Γ' (right) shown in Figure 1.

2007; Jackson and Rogers, 2007; López-Pintado, 2008; Galeotti et al., 2009; Lamberson and Page, 2009). In nearly all network diffusion studies, individual nodes of a network are more likely to transition from a status quo state to a new state of interest when more of their neighboring nodes have made that transition; there is some type of local reinforcement. For example, a person is more likely to become infected with a disease when more of their contacts have the disease, or a person is more likely to adopt a new technology when more of their friends and family have adopted the technology. The form of this local reinforcement may differ across models or appear as a parameter of the model.

To be more precise we will consider a variation of the basic susceptible-infected-susceptible (SIS) model of infection (Bailey, 1975). In this model every individual is in one of two states, susceptible or infected. Susceptible agents that come into contact with infected agents run the risk of becoming infected. An individual can recover from the infection, but once she does she is immediately susceptible to becoming infected again. There is no mortality in the model.

In an SIS model with no network structure, if each contact between an infected individual and a susceptible individual leads to a new infection with probability β , and the probability that an infected individual recovers in each time step is γ , then the disease will spread from an initial infection if

$$\frac{\beta}{\gamma} > \frac{1}{N}, \tag{5}$$

where N is the total population size.

To add network structure to the model, we adjust the probability of infection to reflect the agents' number of neighbors and number of infected neighbors. The probability that a susceptible agent of degree d with x infected neighbors becomes infected in each small time step is $\beta \cdot f(d, x)$. Following López-Pintado (2008), we call the function f the *diffusion function* and the ratio $\lambda = \frac{\beta}{\gamma}$ the *effective spreading rate*. As in the SIS model without network structure, there is a *diffusion threshold* λ^* such that if $\lambda > \lambda^*$ then the infection will spread from an initial infection to a non-zero steady state; if $\lambda \leq \lambda^*$ then the infection will die out. Figure 3 illustrates the diffusion threshold for the two networks shown in Figure 1. The figure plots the average percent infected nodes of each network for different values of the effective spreading rate. The diffusion threshold for Γ' is lower than the diffusion threshold for Γ , and so diffusion occurs more easily in Γ' .

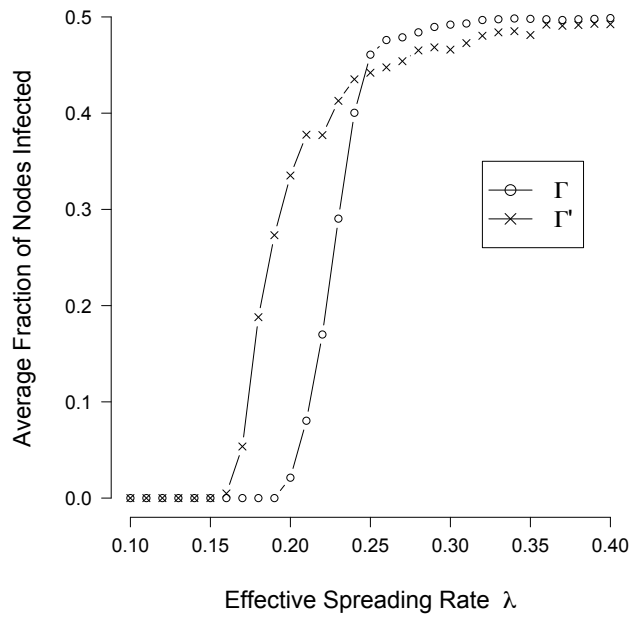


Figure 3: The average fraction of infected nodes for the two networks, Γ and Γ' , shown in Figure 1 for different values of the effective spreading rate $\lambda = \frac{\beta}{\gamma}$ from 10 realizations of the SIS diffusion model with $f(d, x) = x$.

The main result relating stochastic dominance and diffusion is as follows.³

Theorem 1 (López-Pintado 2008). *The diffusion threshold for a network Γ will be lower than that for a network Γ' (and thus an infection is more likely to spread through Γ than Γ') if*

- Γ FOSD Γ' , or
- Γ is a MPS of Γ' and $d^2 f(d, 1)$ is convex for all $d \geq 1$, or
- Γ' is a MPS of Γ and $d^2 f(d, 1)$ is concave for all $d \geq 1$.

The theorem says that diffusion is more likely to occur in networks with more connections or those with more variability in the number of connections if $d^2 f(d, 1)$ is convex or less variability in connections if $d^2 f(d, 1)$ is concave.

To give some intuition behind the theorem, notice that increasing the degree of an agent in the network increases not only the probability that she is infected, but also the probability that she infects someone else. This leads to the d^2 term. Thus, the effect of one degree d agent with one infected neighbor on the total infected population varies like $\beta d^2 f(d, 1)$. Since we are interested in the diffusion rate in Γ relative to the rate in Γ' , and the β term appears in both, we can ignore it and consider only $d^2 f(d, 1)$. Now, we care about the expectation of this measure of infectivity over all agents in the network, so we integrate this against the degree distribution to obtain

$$\sum_{d=1}^{D_{max}} d^2 f(d, 1) P(d). \quad (6)$$

If Γ FOSD Γ' (as in the first bullet of the theorem), since $d^2 f(d, 1)$ is increasing in d , by equation (2) if P FOSD P' , equation (6) will be smaller if we replace P by P' i.e. the infection spreads more in Γ than Γ' . By equation (4), if $d^2 f(d, 1)$ is concave and Γ' is a mean preserving spread of

³We wish to emphasize that this result relies on a mean-field approach, which requires several implicit assumptions regarding the diffusion dynamics. Here, we focus on the qualitative implications of the theorem and the broad similarities between the many diffusion models to which it applies in one form or another, so we will not digress into these technical conditions. For a precise statement of the second and third bullets of this theorem and the assumptions it requires see the article by López-Pintado (2008). The first bullet does not appear in the article by López-Pintado (2008), but the proof is straightforward from the results there. For similar results under different models of diffusion see the articles by Jackson and Yariv (2005, 2007), Jackson and Rogers (2007), Lamberson and Page (2008) and Galeotti et al. (2009).

Γ (as in the third bullet of the theorem), then equation (6) is smaller if we replace P with P' i.e. the infection spreads more in Γ than Γ' . The logic for the second bullet is similar, noting that if $d^2 f(d, 1)$ is convex then $-d^2 f(d, 1)$ is concave.

A useful corollary to Theorem 1 is:

Corollary 1 (López-Pintado 2008). *If Γ is a mean preserving spread of Γ' and f depends only on x (not on d), then the diffusion threshold for Γ is lower than that for Γ' .*

Thus, if the likelihood that any agent becomes infected depends only on the number of infected contacts she has, independent of her total number of contacts, increasing the variation of the degree distribution makes diffusion easier. This follows immediately from Theorem 1 by setting f equal to a positive constant c in the second bullet and observing that $d^2 \cdot c$ is convex.

Figure 3 illustrates an example of Corollary 1. The diffusion function $f(d, x) = x$ depends only on x , so Corollary 1 applies. Since Γ' is a mean preserving spread of Γ , Corollary 1 implies that the diffusion threshold for Γ' is lower than that for Γ , as the computations depicted in Figure 3 confirm.

4. Network Structure and Diffusion

In this section we describe several models and results on network diffusion and demonstrate how they can be understood through the lens of stochastic dominance and in particular as versions of Theorem 1 or Corollary 1.⁴

4.1. Watts-Strogatz Small-worlds

Some of the most commonly modeled networks are the Watts-Strogatz small-worlds (Watts and Strogatz, 1998). This family of networks is parameterized by a single variable p ranging from zero to one. To construct the network corresponding to a given value of p , begin with a ring lattice in which each of N nodes is connected to its k closest neighbors. For each node n of the network, consider each edge connected to that node and with probability p disconnect the opposite end of that edge and reconnect it to another node chosen uniformly at random from all of the nodes not

⁴Theorem 1 does not prove all of the results from the papers discussed, since in many cases minor modifications must be made in order to fit the relevant model to the conditions of the theorem or vice versa.

already connected to n . For $p = 0$, the corresponding network is the original regular ring lattice and for $p = 1$ the network is random. For intermediate levels of p the resulting network exhibits two characteristics of many empirical networks, low average path length (the so-called “small-world” phenomenon) and high clustering. The corresponding degree distributions range from a delta distribution when $p = 0$ to a Poisson distribution when $p = 1$. The networks depicted in Figure 1 are Watts-Strogatz networks with $N = 250$ and $k = 3$; Γ (left) has $p = .05$, and Γ' (right) has $p = 1$. Figure 2 plots their degree distributions.

Since for every p the number of nodes and the number of edges remains the same, the average degree is constant, and thus all of the networks in the family are equal under the first order stochastic dominance relation. However, if $p > p'$ then the Watts-Strogatz network with parameter p is a mean preserving spread of the Watts-Strogatz network with parameter p' . In light of this ordering and Theorem 1, many properties of networks that vary monotonically with p in the Watts-Strogatz family may hold more generally for arbitrary networks under the mean preserving spread relation.

For example, in the seminal paper by Watts and Strogatz (1998) in which the small-world family is introduced, the authors consider a standard diffusion model as an illustration of the significance of their construction for dynamic processes. Initially all of the population is healthy and at time zero one infected individual is introduced. Individuals recover at a fixed rate and during each unit of time infect each of their neighbors with probability r . Watts and Strogatz observe through simulation that the critical infectiousness, above which an epidemic sweeps the network and below which the disease vanishes, decreases with p . We can see this as a consequence of stochastic dominance. Since in this case the likelihood that an agent becomes infected depends only on the number of her neighbors that are infected, independent of her total number of neighbors, Corollary 1 implies that the Watts-Strogatz networks with a higher p will have a lower diffusion threshold and thus be more prone to diffusion of the infection.

4.2. Concurrency and Disease Spread

Kretzschmar and Morris (1996) investigate the effect of concurrent partnerships on the spread of sexually transmitted diseases. In their model the network of connections represents sexual partnerships and is constantly in flux as new partnerships are formed and old partnerships are dissolved. However, while specific partnerships change, the degree distribution remains relatively unchanged (coincidentally this more closely fits the assumptions of the mean-field approximation than a static

network). When an infected individual is in a sexual relationship with a susceptible individual the disease is transmitted with a fixed probability.

Kretzschmar and Morris consider a population level measure of concurrency (the number of relationships that an individual carries on simultaneously) which they call the index of concurrency and denote κ_3 . Since any two edges that connect to the same node in the sexual contact graph at a given time correspond to concurrent relationships, concurrency is related to the degree distribution of the graph. In particular, their measure is approximately

$$\kappa_3 = \frac{\sigma^2}{\mu} + \mu - 1, \quad (7)$$

where μ and σ are the mean and standard deviation of the degree distribution respectively. They examine the effect of varying the level of concurrency on the extent that a simulated disease spreads from a single infection and find that the number of agents infected in a fixed time grows exponentially with κ_3 .

The relationship between varying levels of κ_3 and stochastic dominance is ambiguous because κ_3 is not monotonically related to μ ; however, for the family of networks that Kretzschmar and Morris examine, μ remains fixed. Thus, increases in κ_3 can be accounted for by increases in σ and therefore correspond to mean preserving spreads. Because the probability that an agent becomes infected depends only on the number of her infected partners, Corollary 1 applies, so the increased diffusion with increased concurrency is explained by the stochastic dominance relation.

4.3. Increasing Returns and Winner-take-all Markets

Besides the spread of disease, diffusion models are often used to represent the adoption of products or innovations. In many situations an agent might prefer a product that has been purchased more by other consumers. When this occurs, the market is said to exhibit *increasing returns* (Arthur, 1994). For example, a consumer can expect that more software will be developed for a more popular hardware platform, thus making that hardware platform more desirable. In this case, the increasing returns are global; it is the overall level of adoption in a population that affects the availability of hardware. In other cases, the increasing returns may be local. For example, a professor might prefer to use a computer with the same operating system as her coauthors so that she can more easily share files with them. In this case it is only the choices of the individuals that are “near” the agent in some social sense that affect the agent’s purchasing decision, so we

call the returns *local*.⁵ Arthur (1989) shows that when consumers choose among a set of products based on global increasing returns eventually one of the products will come to dominate the market completely. This outcome is popularly referred to as the *winner-take-all* outcome (Frank and Cook, 1996).

When consumers choose based on local increasing returns, multiple products can split the market (Janssen and Jager, 2003; Lee et al., 2006; Lamberson, 2008). We observe this “local bias” in reality when, for example, most professors in one field use Macintosh computers while most in another use PCs. The tendency of the market to converge to a winner-take-all outcome or a shared market depends on the structure of the social network. For example, consider the model developed by Lee et al. (2006). A new technology is introduced in two variants, A and B . Consumers choose whether to adopt the new technology at all, and when they do, whether to adopt variant A or variant B . Both global and local increasing returns (Lee et al. refer to them as indirect and direct network effects) drive consumer adoptions. Specifically, an agent i 's utility from choosing variant A is

$$U_i(A) = a_i + \alpha x_{i,A} + \beta \pi_A, \quad (8)$$

where a_i is an agent specific preference for variant A , $x_{i,A}$ is the number of i 's neighbors using variant A and π_A is the proportion of all adopters in the population choosing variant A . The α and β terms are weights to adjust the relative strength of the global and local returns. The analogous utility for variant B is

$$U_i(B) = b_i + \alpha x_{i,B} + \beta \pi_B. \quad (9)$$

A consumer adopts the new technology when her utility from one of the variants is greater than 0 (one can think of a_i and b_i as costs of adoption, so a consumer adopts when her utility overcomes these costs), and then she chooses whichever variant offers her the greatest utility. Agents are allowed to periodically switch variants if they find that their preference ordering has reversed. Lee et al. simulate the purchases of a population of agents on a Watts-Strogatz family of networks and examine the effect of the network parameter p on the probability that the market converges to a winner-take-all outcome.

For the moment, consider only the diffusion of a single variant, say A , and ignore the global increasing returns $\beta \pi_A$. In this case, the probability of an agent adopting depends only on the

⁵Increasing returns can also be local in the product space as opposed to the social space (Lamberson, 2008).

number of her neighbors that adopt, so applying Corollary 1, we would expect diffusion to occur more easily for higher values of p . The same argument can be applied to variant B , which causes the market to be more unstable when p is higher. The effect is exacerbated by the global increasing returns making a winner-take-all outcome more likely in networks with a higher value of p .⁶

This is exactly the result that Lee et al. find by simulation. With their parameter choices, moving from networks with values of $p < .2$ to those with $p > .5$ changes the frequency of a winner take all outcome from close to zero to nearly one. The theory implies that these results would also hold for other types of networks ordered by second order stochastic dominance. Moreover, we would expect the same argument to apply to networks ordered by first order stochastic dominance, which Lee et al.'s simulations alone cannot suggest since all of the networks in the Watts-Strogatz family they employ have the same average degree.

⁶While qualitatively Lee et al.'s results fit the predictions of Theorem 1 and Corollary 1, the threshold model that they consider better matches the model and similar results on stochastic dominance and diffusion of Jackson and Yariv (2005, 2007) than those of the SIS model discussed here and in López-Pintado (2008).

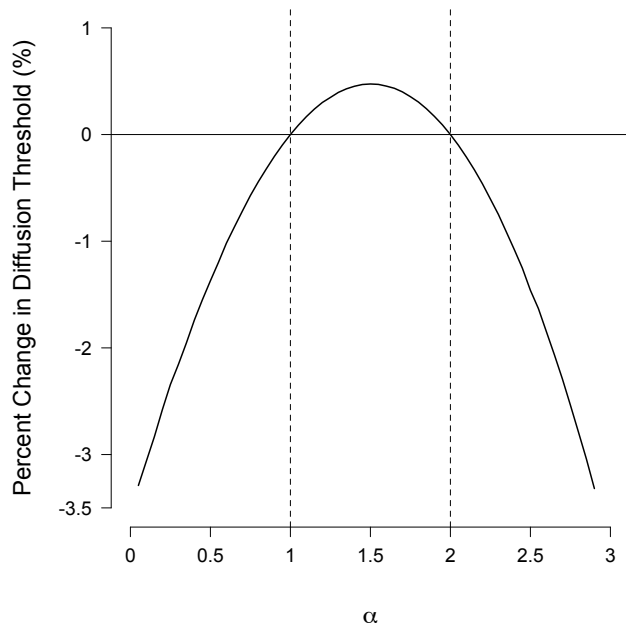


Figure 4: The difference in the critical threshold between a Watts-Strogatz network with $p = 0$ ($N = 1000$, $k = 10$) and with $p = 1$ as the exponent α in the diffusion function $f(d, x) = xd^{-\alpha}$ is varied. When α is between one and two, increasing the randomness of the network raises the diffusion threshold and thus makes diffusion less likely. For all other values of α , increasing randomness lowers the diffusion threshold.

5. Reversing the Inequality

In all of the examples we have discussed and most of the examples we have encountered in the literature, moving from one network to a mean preserving spread of that network increases the likelihood of diffusion. Based on these observations alone, one might conclude that a mean preserving spread of the degree distribution always leads to greater diffusion, but with the theory of stochastic dominance in hand we can see that the effect is conditional on the form of the diffusion function.

Corollary 1 partially explains the observations. In order to find a case where a mean preserving spread of the degree distribution decreases diffusion, the diffusion function must depend not only on the number of infected contacts that an individual has, but also on the degree of the individual. Moreover, suppose that the diffusion function is of the form $f(d, x) = xd^{-\alpha}$. A mean preserving spread results in a decrease in diffusion when $d^2 f(d, 1) = d^{2-\alpha}$ is concave for all $d \geq 1$, which only holds for values of α between one and two. This is illustrated in Figure 4, which plots the percent change in the critical threshold between a Watts-Strogatz network with $p = 0$ ($N = 1000, k = 10$) and with $p = 1$ as the exponent α in the diffusion function is varied.⁷ For values of α between one and two, moving from a regular lattice to a random network increases the diffusion threshold (by .47% at the most). For any other value of α , the diffusion threshold is lower in a random network than in a regular one, and there is no limit on the magnitude of this effect as α is increased. Of course the diffusion function may not be of the form $f(d, x) = xd^{-\alpha}$, but in a sense there are many fewer diffusion functions under which a mean preserving spread decreases diffusion than there are under which the opposite relationship holds.

6. Conclusion

Stochastic dominance helps to explain, connect, and generalize the many observations relating network structure to diffusion. Ongoing research continues to expand our understanding of these relationships and their implications for different and more general diffusion mechanisms (Galeotti et al., 2009) as well as policies for exploiting these relationships (Galeotti and Goyal, 2008).

⁷The critical diffusion threshold was calculated for fifty networks of each type using the formula from López-Pintado (2008).

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