Complex Contagion and the Weakness of Long Ties

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Abstract

The strength of weak ties is that they tend to be *long* – they connect socially distant locations. Recent research on "small worlds" shows that remarkably few long ties are needed to give large and highly clustered populations the "degrees of separation" of a random network, in which information can rapidly diffuse. We test whether this effect of long ties generalizes from *simple* to *complex* contagions – those in which the credibility of information or the willingness to adopt an innovation requires independent confirmation from multiple sources. Using Watts and Strogatz's original small world model, we demonstrate that long ties may not only fail to speed up complex contagions, they can even preclude diffusion entirely. Results suggest that the spread of collective actions, social movements, and risky innovations benefit not from ties that are long but from bridges that are wide enough to transmit strong social reinforcement. Balance theory shows how wide bridges might also form in evolving networks, but this turns out to have surprisingly little effect on the propagation of complex contagions. We find that *hybrid* contagions, which have high thresholds for some nodes and low-thresholds for others, can propagate on perturbed networks if the latter are sufficiently numerous. However, for purely complex contagions, propagation depends decisively on wide bridges, a characteristic feature of spatial networks. This may account in part for the widely observed tendency for social movements to diffuse spatially.

"All politics is local."

– Rep. Tip O'Neil, former Speaker of the U.S. House of Representatives

Pundits and activists have recently discovered the Internet as a medium with unprecedented opportunities for mass mobilization (Tilly 2004). The Internet is a scale free network (Barabasi and Jeong 2000) whose highly skewed degree distribution is ideal for very rapid diffusion of information.

Despite this growing excitement, leading scholars of social movements have expressed doubts about using the Internet as a medium for mobilizing collective action.[1] Beginning with McAdam's *Freedom Summer,* numerous case studies have shown that "spreading the word" alone is not sufficient to recruit new members to a social movement. Recruitment also requires having friends in the movement (McAdam and Paulsen 1993; McAdam and Rucht 1993)[2] who do more than *inform*, they also *persuade*.

The debate over the effectiveness of the Internet for social movement mobilization illustrates the important distinction between the *acquisition* of information and the decision to *act* on the information. Information, like disease, can be acquired passively from many sources of casual social contact. In contrast, the decision to act on the information (participate in a social movement or adopt an innovation), is an active choice, and in making that choice, people are influenced not only by the content of the information (such as the effectiveness of an innovation or social movement) but they are

1 For a parallel argument from the perspective of persuasive communication, see Wellman and Caroline (2002).

2 McAdam's (1988) study of recruitment to Freedom Summer also found that people were much more likely to join if they had multiple friends who were already members.

also influenced by the observation of prior adopters, especially those that they know. Examples are easy to find, whether the innovation is a belief, ideology, norm, technology, organizational form, fad, or fashion.[3] People may hear about a movement to "think globally, act locally," but it is when they see people they know getting involved that they become most susceptible to recruitment. Similarly, many people may hear about a new fashion, but it is not until they see their friends display it that they are persuaded to go along (Crane 1999). From hybrid corn (Ryan and Gross 1943) to medical innovations (Coleman, Katz and Menzel 1966), the pattern is well-documented. The decisive event is not hearing about an innovation, but observing enough people participating to be convinced that the innovation should be adopted (Coleman 1990; Simmel 1950; Rogers 1995).

The important distinction here is not between friends and strangers. The influence of friends is hardly surprising, given the strength of the ties that are sustained over time through commitment, trust, and emotional attachment. However a distinction which is less appreciated, but perhaps even more important, is between the structural features of social networks that promote the dissemination of information about an innovation or social movement and the network features that promote the spread of density-dependent decisions to act on the information. Our principal thesis is that *network structures that are highly efficient for the rapid dissemination of information are often not conducive to*

3 Decision-theoretic research on adoption of innovation includes studies of HIV prevention (Kelly et al. 1997; Miller et al. 1998), birth control (Rogers and Kincaid 1981), religion (Chaves and Montgomery 1996), and collective action (Chwe 1999; Kim and Bearman 1997; Marwell and Oliver 1993; Gould 1995; Macy 1991).

the diffusion of collective action based on the information. In particular, we challenge the accepted wisdom that social diffusion benefits from ties that are long.

Information, Disease, and the Strength of Weak Ties

Friendships are strong ties. However, diffusion over social and information networks displays a striking regularity that Granovetter (1973) called "the strength of weak ties." As Granovetter put it (1973: 1366), "whatever is to be diffused can reach a larger number of people, and traverse a greater social distance, when passed through weak ties rather than strong."

"Strong" and "weak" have a double meaning in Granovetter's usage. One meaning is relational (at the dyadic level), the other is structural (at the population level). The relational meaning refers to the strength of the influence that is conveyed through the tie. Weak ties connect acquaintances who are relatively less invested in the relationship and less readily influenced by one another. Strong ties connect close friends or kin whose interactions are frequent, affectively charged, and highly salient to each other. Strong ties increase the trust we place in close informants, the exposure we incur from contagious intimates, and the influence of close friends. As Rogers (1995: 340) notes, "Certainly, the influence potential of network ties with an individual's intimate friends is stronger than the opportunity for influence with an individual's 'weak ties'."

Granovetter introduces a second, *structural*, meaning. The structural strength of a tie refers to the ability of a tie to facilitate propagation by linking otherwise distant nodes in a social network. Granovetter's insight is that ties that are weak in the relational sense – that the relations are less salient – are often strong in the

structural sense – that they provide shortcuts across the social topology. The strength of weak ties is that casual relations are more likely to be formed between socially distant actors with few network "neighbors" in common. Long ties greatly increase the rate at which information propagates, despite the weakness of the tie as a conduit.

Conversely, strong social relations also have a structural weakness – transitivity. If Adam and Betty are close friends, and Betty and Charlie are close friends, then it is also likely that Adam and Charlie are close friends. Information in closed triads tends to be redundant, which inhibits diffusion. Adam, Betty, and Charlie may strongly influence one another, but if they all know the same things, their network will not help them learn about opportunities, developments, or new ideas that lie outside their closed circle. That is the weakness of their strong ties.

Small Worlds

Granovetter's insight has become one of the most cited and influential contributions of sociology to the advancement of knowledge across many disciplines, from epidemiology to computer science. However, the full impact was not realized until recently, when Watts and Strogatz (1998) made an equally startling discovery. Not only do weak ties facilitate diffusion when they provide "shortcuts" between remote clusters, but it takes only a small fraction of these long ties to give even highly clustered networks the "degrees of separation" characteristic of a random network. This means that information and disease can spread very rapidly even in a "small world" composed mostly of tightly clustered provincial communities with strong ingroup ties, so long as a few of the ties are long. It takes only a few contagious people traveling between remote villages to make the entire population highly

vulnerable to catastrophic epidemics. It takes only one villager with a cousin in the city to bring news of job openings at a factory. Simply put, an added strength of weak but long ties is that it takes remarkably few of them to give even highly clustered networks a very low characteristic path length (the typical distance between any two nodes in the network).

This principle is so clear and familiar that restating it may seem to beg banality. Nevertheless, we believe that the "weak ties" intuition can be misleading. The "strength of weak ties" applies to the spread of information and disease but not to many types of social diffusion which depend on influence from prior adopters, such as participation in collective action, the use of costly innovations, or compliance with emergent norms. For these contagions, we contend that long ties are not strong in either of Granovetter's meanings, relational or structural.

The implication of the relational meaning is immediately apparent. A low level of trust and familiarity between socially distant persons inhibits persuasion and imitation. What is not at all obvious is our contention that long ties have a structural weakness – they can also *inhibit* the diffusion of many social contagions.

Simple and Complex Contagions

Long ties clearly do not inhibit the spread of disease or information, even if contacts are less frequent or relations less trusted. However, many social contagions are not like disease or information. Information and disease are *simple contagions*, which means they can be spread by contact with a single source. Hence, a single tie is sufficient to allow an epidemic to leap over large social distances to a remote region

of the network. For simple contagions, the longer the ties – that is, the greater the social distance that they traverse – the faster the propagation.

Using analytical and computational models, we show that this principle does not generalize to complex contagions. A contagion is complex if its transmission requires an individual to have contact with two or more sources of activation. *The transmission of disease may take multiple exposures to an infected neighbor, but it does not require exposure to multiple infected neighbors.*

The distinction between *multiple exposures* and *exposure to multiple sources* is subtle and easily overlooked, but it turns out to be decisively important for understanding the weakness of long ties. It may take multiple exposures to pass on a contagion whose probability of transmission in a given contact is less than one. If the probability of transmission is P, the probability of contracting the disease after E exposures is $1-(1-P)^E$. Even for very small probabilities, for any $P > 0$ it remains possible to contract the contagion from a single encounter. Further, each contact with the same infected individual counts as an additional exposure.

By contrast, for complex contagions to spread, multiple sources of activation are required since contact with a single active individual is not enough to trigger adoption. There are abundant examples of complex contagions. The credibility of an urban legend (Heath, Bell and Sternberg 2001), the costs of using new technologies (Coleman 1966), educational attainment (Berg 1970), the willingness to participate in a migration (MacDonald and MacDonald 1974), and incentives to exit formal gatherings (Granovetter 1978; Schelling 1978) all depend on how many of one's contacts have already become participants.

Mechanisms of Complex Contagion

There are at least four mechanisms that might explain why complex contagions require exposure to multiple sources of activation: strategic complementarity, credibility, legitimacy, and emotional exchange.

1. *Strategic complementarity.* Simply knowing about an innovation is rarely sufficient for adoption (Gladwell 2000). Many innovations are costly, especially for early adopters but less so for those who wait. The same holds for participation in collective action. Studies of strikes (Klandermans 1988), revolutions (Gould 1996), and protests (Marwell and Oliver 1993) emphasize the positive externalities of each participant's contribution. The costs and benefits for investing in public goods often depend on the number of prior contributors – the "critical mass" that makes additional efforts worthwhile.
2. *Credibility.* Innovations often lack credibility until adopted by neighbors.[4] For example, Coleman et al. (1966, 1983) found that doctors were reluctant to adopt medical innovations until they saw their colleagues using it. Markus (1987) found the same pattern for adoption of media technology. Similarly, the spread of rumors (Granovetter 1978), urban legends (Heath, Bell and Sternberg 2001), and folk knowledge generally depends upon multiple confirmations of the story before there is sufficient credibility to report it to others. Hearing the same story from different

4 This applies as well to the spread of information. Hearing about a job the first time is usually sufficient, but gossip may not be believed until confirmed by independent sources. Hence, the distinction between simple and complex contagions is not perfectly correlated with the distinction between information and innovation. Not all information is passively acquired and not all innovations are influenced by prior adopters.

people makes it seem less likely that surprising information is nothing more than the fanciful invention of the informant.

3. *Legitimacy.* Knowing that a movement exists or that a collective action will take place is rarely sufficient to induce bystanders to join in. Having several close friends participate in an event often greatly increases an individual's likelihood of also joining (Finkel Muller and Opp 1989; Opp and Gern 1993), especially for high-risk social movements (McAdam and Paulsen 1993). Decisions about what clothing to wear, what hair style to adopt, or what body part to pierce are also highly dependent on legitimation (Grindereng 1967). Innovators risk being shunned as deviants until there is a critical mass of early adopters (Crane 1999; Watts 2002), and non-adopters are likely to challenge the legitimacy of the innovation.
4. *Emotional contagion.* Most theoretical models of collective behavior – from action theory (Smelser 1963) to threshold models (Granovetter 1973) to cybernetics (McPhail 1991) – share the basic assumption that there are expressive and symbolic impulses in human behavior that can be communicated and amplified in spatially and socially concentrated gatherings (Collins 1993). The dynamics of cumulative interaction in emotional contagions has been demonstrated in events ranging from acts of cruelty (Collins 1974) to the formation of philosophical circles (Collins 1998).[5]

5 For a series of empirical studies, see the special issue of *Mobilization* on "Emotions and Contentious Politics" (Aminzade and McAdam, eds., http://www.mobilization.sdsu.edu/volumes/Volume07.html#).

Complex Contagion: Contested and Uncontested

These four mechanisms explain why many social contagions require exposure to multiple sources of activation. However, some of the examples used to illustrate these complex contagions also resemble the spread of information and disease, in that the contagions are *uncontested*. We define a contagion as uncontested if activation depends solely on the number of neighbors who are activated, without regard to the number who are *not* activated. For example, neighbors give us their germs but they do not give us their immunities, and uninformed neighbors do not prevent access to those who are. Neighbors also tell jokes that are much funnier when heard for the first time, and thus more likely to be spread if others are assumed not to also know it. It is the same with many complex contagions, such as the spread of urban legends and fashionable technologies (e.g., iPods). For uncontested contagions, it is the absolute number of others whose state is *different* from one's own that triggers the decision to acquire that state, regardless of the number whose state is the *same* as one's own. Thus, unlike the spread of information or disease, complex contagions require exposure to multiple sources, but as with these simple contagions, if complex contagions are uncontested, non-adopters do not discourage their neighbors from adopting. In contrast, *contested* contagions depend not only on having more than one activated neighbor, they also depend on the number who are *not* activated. Neighbors who have joined a demonstration or adopted a controversial innovation increase the pressure to follow suit, while skeptics, cynics, and opponents on the sidelines decrease it. We define a contagion as "contested" if non-activated neighbors exert countervailing influence. Hence, with contested contagions, the threshold of activation, τ, is based on the proportion of neighbors who are activated, not the number (0

$\leq \tau_i \leq 1$). A node i with threshold $\tau_i = 0.5$ can only be activated if half of i's neighbors are activated. For uncontested contagions, the corresponding range is given by $0 \leq \tau_i \leq z$, where z is the number of i's neighbors. (Note that τ_i indicates that thresholds vary across nodes, while the unindexed τ will be used to indicate that all nodes have identical thresholds.)

Examples of contested contagions abound, including threshold effects in collective action with partial jointness of supply (Marwell and Oliver 1993), the legitimacy of emergent norms (Oberschall and Kim 1996), the efficacy of participation in social movements (Macy 1991), the credibility of social influence (Friedkin 2000; Latané Nowak and Luiu 1994), the pressure to conform (Asch 1956), the seduction of a riot (Granovetter 1978), the anxiety of witch hunts (Centola, Willer and Macy 2005), and the preference for voting (Katz and Lazarsfeld 1954). In all these examples, activation depends not only on the number who are active but also on the number who are not.

By definition, all simple contagions are uncontested; if unactivated neighbors exert counterpressure, then as the number of neighbors increases, so too does the number of activated neighbors needed to trigger adoption. Figure 1 diagrams the distinctions between simple and complex contagions, and between complex contagions that are uncontested and contested.

[Figure 1 about here]

Effects of Group Size

The distinction we propose between simple, uncontested, and contested contagions is intended as more than a descriptive typology. The distinction is also important theoretically. The possibility that all neighbors exert influence, not just those that are

activated, has an important implication for the effect of neighborhood size. Suppose a population is entirely unactivated except for a small number of randomly activated members. With uncontested contagions like disease or information, the more people one comes in contact with, the greater the chance of encountering an informed or infected individual. Thus, *the larger the number of neighbors, the greater the chance of becoming activated.* That is the case for both simple and complex contagions, so long as the latter are uncontested (that is, unactivated neighbors do not exert pressure against adoption).

With contested contagions, it is quite the opposite. *The more neighbors an actor has, the lower the susceptibility to activation* (Watts 2002). Suppose everyone has four neighbors and a threshold of 0.5, which means that two activated neighbors are required to offset the counterpressure from two unactivated neighbors. If two activated members happen to be in the same neighborhood, the contagion will spread to a new neighborhood. Now suppose everyone has 24 neighbors. This increases the probability of contact with activated neighbors, but it also increases exposure to counterpressures from neighbors who remain unactivated. Thus, the number of activated neighbors needed for the contagion to spread increases from two to twelve. Twelve activated neighbors are needed because the influence from the converted neighbors is offset by the countervailing influence of the other twelve, something that would not happen in the spread of disease or information.

This analysis also has implications for the effects of group size on participation in collective action. For public goods with pure jointness of supply (such as public broadcasting), free-riders do not reduce the incentive to contribute, since the cost to supply one person is the same as the cost to supply everyone (Marwell and Oliver 1993).

The larger the community that enjoys public broadcasting, the greater the chances of finding people who are willing to contribute unconditionally, no matter how many others might enjoy the public goods for free.

However, not all public goods have pure jointness. For example, the effort required to remove litter from a public beach increases with the number of people who use it. A lone volunteer might be willing to clean up the beach if only ten people used it, but if a thousand people used it, that same person might only volunteer if others did so as well. Participation of the volunteer requires additional volunteers as the number of non-volunteers using the beach increases. Where contributions are encouraged by volunteers and discouraged by free-riders, collective action is likely to spread as a contested contagion, in which the incentive to contribute increases with the proportion of contributors, not the absolute number.

An Analytical Model of Complex Contagions on a Ring Lattice

Although the effects of group size are an interesting implication of complex contagions, our central purpose is to call attention to the implications for the effects of network structure, and in particular, to challenge the generalization of the "small world" principle from the spread of information and disease to many other types of social diffusion.

The classic formalization of the "small world" intuition comes from Watts and Strogatz (1998). They demonstrate that the rate of propagation on a clustered network can be dramatically increased by randomly rewiring a few local network ties, making them into bridge ties that reduce the distance between arbitrarily chosen nodes in the network.

Most theoretical work on small world networks assumes simple contagions like information and disease that have thresholds of activation at the theoretical lower limit for propagation through social contact. There are good reasons to make this assumption. Simple contagions can be studied on random networks, which are highly amenable to analytic treatment (Erdos and Renyi 1959). In contrast, complex contagions cannot propagate on random networks (Morris 2000). Further, mathematical approximations can be made for simple contagions (Watts 2002), which cannot be used for those that require multiple sources of activation. The assumption that the global properties of complex contagions can be extrapolated from the properties of simple contagions is thus highly convenient.

Unfortunately, it can also be highly misleading. When activation requires confirmation or reinforcement from multiple sources, the effect of bridges[6] depends not only on their *length* (the path distance that is spanned) but also on their *width* (the multiplicity of short paths between otherwise non-adjacent nodes). The importance of bridge width has been overlooked in previous research because simple propagation requires only a single link between source and target. However, as McAdam and Paulsen point out, "[t]he fact that we are embedded in many relationships means than any major decision we are contemplating will likely be

6 Strictly speaking, a "bridge" is an edge whose removal disconnects a graph, while a "local bridge" of length l is a single tie between nodes whose distance would otherwise be at least l. We use the term "bridge" exclusively in the latter sense and therefore drop the modifier "local." However, we also disallow the assumption that a bridge consists of a single tie. For simple contagions, a single tie (or edge) is sufficient to form an effective bridge. For minimally complex contagions, an effective bridge consists of three ties, and the number increases with the threshold of the contagion. Thus, we define a bridge as a set of short paths ($l \leq 3$) that reduce the distance for a contagion to propagate between non-adjacent nodes, and the width w_{ks} of the bridge from s to k is defined as the number of these short paths.

mediated by a significant subset of those relationships" (McAdam and Paulsen, 1993: 646). Research on complex contagions, such as the spread of participation in collective action and social movements, points to the need to consider what happens when thresholds are higher and activation requires exposure to more than one activated source.

Figure 2 illustrates the importance of bridge width for complex contagions, using the same ring lattice with degree $z = 4$ that Watts and Strogatz (1998) used to demonstrate the small worlds effect. A ring lattice is a one-dimensional spatial network that allows the simplest analytical model of the effects of adding a very small number of long ties to an ordered graph. Following Watts and Strogatz, network density is held constant by removing existing ties from nodes that acquire random ties (indicated by dashed lines in Figure 2). We also follow previous authors (Watts and Strogatz 1998, Watts 1999, Newman 2000) in assuming that every node has equal influence and every tie has equal weight. These assumptions are necessary to identify the structural effects of long ties (*vis-à-vis* the small world effect) without the confounding effects of influence heterogeneity across dyads.[7]

The upper panel of Figure 2 illustrates the simple contagion assumed by Watts and Strogatz, in which all nodes have threshold $\tau = 1$, such that unactivated nodes (indicated by the clear circles) can be activated by contact with any member of a seed

7 The effect of long ties on the spread of complex contagions is sufficiently complicated and important that we have resisted the strong temptation to pursue what is clearly a fascinating question: how the small world effect may depend on the distribution of status, power, influence, and attraction. We eagerly anticipate the opportunity to build on the findings we report here as we relax these and other simplifying assumptions in the small world studies by Watts and Strogatz that provide the starting point for our research on the effects of allowing activation thresholds to rise above the theoretical lower limit for contagion.

neighborhood (indicated by the solid nodes). The seeds correspond to new arrivals who have already acquired the contagion, such as a disease. The lower panel is identical, except that $\tau = 2$, the minimum number of activated neighbors required to propagate a complex contagion.

[Figure 2 about here]

The small worlds effect is evident in the upper panel, where *s* is the focal node of a seed neighborhood *S* in which all *z* neighbors of *s* are activated (solid nodes). Node *i* is the focal node of an unactivated neighborhood containing *j* and *k* (clear nodes). The ovals demarcate neighborhoods of the focal nodes and show the overlap between neighborhoods with $z = 4$. With thresholds that correspond to a simple contagion ($\tau = 1$), adding a single random tie from *s* to *i* creates a shortcut across the ring that reduces the time required for a cascade to reach all the nodes. This effect is now so well documented for the ring lattice (Watts and Strogatz 1998; Watts 1999; Newman and Watts 1999; Newman 2000) that we do not elaborate further.

Instead, we focus attention on the robustness of this result as thresholds increase above the very low levels assumed in previous studies. The lower panel of Figure 2 shows how an increase in thresholds from $\tau = 1$ to $\tau = 2$ triples the width of the bridge required to carry the contagion, from one tie to three. The two random ties from *S* to *i* are sufficient to activate *i*, and the single tie from *S* to *j* is sufficient to activate *j*, given *j*'s tie to *i*. Once activated, *i* and *j* are sufficient to activate *k*, and so on. The width w_{ks} of the bridge from *s* to *k* is defined as the number of short paths ($l \leq 3$) between non-adjacent *k* and *s,* via *i* and *j*. In this example, $w_{ks} = 3$. For $\tau = 2$, $w = 3$ is the minimum width of an *effective* bridge, one that is wide enough to propagate a complex contagion.

It might appear that an increase from one to three in the number of random ties needed to bridge across the ring is rather trivial. It is not. For simple contagions, any random tie can form an effective bridge across the ring. For complex contagions, a random tie is useless unless it is part of a bridge with sufficient width, given the threshold levels. The higher the thresholds, the more ties that are required for an effective bridge. Even in the limiting case for complex contagion, where $\tau = 2$, the probability that three random ties will connect two unactivated neighbors with two or more activated neighbors can be very small, depending on the size and density of the ring and the proportion of activated nodes. Thus, it is likely that many more than three random ties may be needed to effectively bridge the ring.

Even then, it might still appear that the problem is trivial. If random ties that are not part of an effective bridge have no effect on propagation, we can just keep adding random ties until an effective bridge is formed across the ring.

This turns out to be the decisive problem. *Adding random ties disrupts the local propagation of complex contagions.* For simple contagions, adding a random tie has no effect on local propagation. Note the deleted tie to *i* in the upper panel of Figure 2. This has no effect on the spread of the contagion in either direction from *i*.

In contrast, the local propagation of complex contagions depends on wide bridges, in precisely the same way that wide bridges are needed to span across the ring. The deleted tie to *i* in the lower panel of Figure 2 means that *i*'s neighbor to the left cannot be activated, blocking the contagion from spreading further in that direction. More generally, if network density is held constant by deleting existing ties from nodes that acquire random ties, every random tie that is not part of an effective bridge reduces the

width of a bridge between non-adjacent nodes. Alternatively, if no existing ties are deleted and density is allowed to increase as ties are randomly added, a contested contagion may still be blocked due to the increased exposure to unactivated neighbors.

We prove this using a series of theorems that derive the implications of a minimal increase in activation thresholds, from one activated source to two. Although many complex contagions have much higher thresholds, we make the conservative assumption that thresholds differ minimally from the simple contagions assumed in previous research on small worlds. We develop the proofs for the ring lattice used by Watts and Strogatz, with constant density and constant thresholds, and then extend the argument to less tractable networks using computational models.

The proofs depend on two assumptions, whether or not contagions are contested, and whether or not density is held constant as ties are randomly added. The proofs therefore address all four combinations of these binary conditions. For contested contagions, thresholds are the fraction of a node *i*'s neighbors that need to be activated in order for *i* to also become activated, with a range of $2/z \leq \tau \leq 1$. For uncontested contagions, thresholds are the corresponding absolute number, hence τ can range from $2 \leq \tau \leq z$.

The argument involves two main theorems. Theorem 1 establishes the need for a very large number of random ties in order to create even a single effective bridge across the ring. Theorem 2 is the more important of the two. It proves that adding random ties erodes the ability of a ring lattice to propagate complex contagions. Both theorems assume uncontested contagions that are minimally complex (two, rather than one,

neighbor must be activated[8]). Both also assume density is held constant by removing existing ties as random ties are added. Each theorem has three corollaries that extend the argument to the other three combinations of scope conditions (contested contagions and increasing density). The proofs are appended.

Theorem 1. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2$, holding network density constant and adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for an uncontested contagion approaches 0 for $N >> z$.

Corollary 1.1. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2/z$, holding network density constant while adding the minimum number of random ties to bridge across the ring, the probability of creating an effective bridge for a contested contagion approaches 0 for $N >> z$.

Corollary 1.2. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2/z$, allowing network density to increase while adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for a contested contagion approaches 0 for $N >> z$.

8 However, more than two neighbors must be activated if the number of unactivated neighbors were to increase through the addition of random ties, given the assumption that unactivated neighbors exert countervailing influence. For the case where contagions are uncontested, only two activated neighbors are needed, no matter how many random ties are created to unactivated neighbors.

Corollary 1.3. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2$, allowing network density to increase while adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for an uncontested contagion approaches 0 for $N >> z$.

Theorem 2. For a ring lattice identical to that for Theorem 1 (including constant density and uncontested contagion), if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Corollary 2.1. For a ring lattice identical to that for Theorem 1 except that network density increases as random ties are added, and if random ties fail to create an effective bridge, each additional random tie has no effect on the number of nodes that a cascade can be expected to reach.

Corollary 2.2. For a ring lattice identical to that for Theorem 1 except that the contagion is contested, if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Corollary 2.3 For a ring lattice identical to that for Theorem 1 except that network density increases as random ties are added and the contagion is contested, if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Beyond the Ring Lattice: A Computational Model

For the ring lattice, the analytical results can be condensed to two main implications of the effect of random ties on the spread of complex contagions. Assuming that the number of random ties added to a ring lattice is the minimum for an effective bridge:

1. The probability of creating an effective bridge between activated and unactivated nodes drops from 1.0 for simple contagions to near zero for contagions that are minimally complex, whether contagions are contested or uncontested, and whether or not density is held constant or allowed to increase as random ties are added. Creating an effective bridge across a ring lattice requires only a single tie for simple propagation. As thresholds increase, and effective bridges become wider, the expected number of ties that must be randomized in order to obtain the required configuration increases exponentially
2. The number of nodes that a cascade can be expected to reach drops from *N* for simple contagions to a small fraction for contagions that are complex, except in the special case where contagions are uncontested and the addition of random ties leads to increased density rather than deleted ties. Otherwise, each random tie that is added reduces the capacity of the ring to support local propagation.

These results for a one-dimensional lattice do not necessarily generalize to higher dimensional structures, which provide detours around local ties that have been deleted. However, these structures lack the analytical simplicity of the ring lattice. For networks with more complicated geometries, we used computational models to extend the analytical results for the ring lattice. We began by replicating the small worlds experiments on the spread of simple contagions, using a two-dimensional lattice with

Moore neighborhoods ($z = 8$),[9] instead of the ring-lattice used in earlier studies (Watts and Strogatz 1998; Watts 1999; Newman and Watts 1999). We then repeated the experiment, with only one change. We increased activation thresholds above the theoretical minimum for propagation through social contact. Propagation of complex contagions becomes ever more difficult as thresholds increase above the minimum for complex contagions ($\tau_i = 2$), requiring even wider bridges. Thus, a very conservative test of the effect of network perturbation on complex contagions is to assume thresholds that are at the minimum for complex contagion.

As in previous studies (Newman 2000), we assumed that thresholds are deterministic (the probability of activation goes from zero to one as the threshold is crossed), and that once a node is activated, it does not revert to its former state.[10] The parameter p ($0 \leq p \leq 1$) governs the proportion of ties that are rewired,[11] where $p = 0$ corresponds to a regular lattice and $p = 1$ corresponds to a random network. Between 0 and 1, there is a critical region for p in which there is high local clustering with low characteristic path length, corresponding to a small world network.

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9 Moore neighborhoods give each node eight neighbors on a two-dimensional grid, four on the rows and columns and four on the diagonals. Degree z can then be increased from 8 to 24 to 48 (and so on) by increasing the neighborhood radius r, where $z = 4(r^2+1)$. Qualitatively similar results are found for $r = 1$ and $r > 1$.

10 Stochastic thresholds do not change the results qualitatively so long as positive probabilities are constrained to thresholds within the range $1/z < \tau \leq 1$, which is a scope condition for a theory of complex contagion. We leave the effects of reversible states to future research.

11 Newman and Watts (1999) show that adding ties to a regular lattice is more robust than the rewiring method (Watts & Strogatz 1998; and Watts 1999) because it is eliminates the possibility of multiple components forming at high values of p. For the computational experiments below, we use the rewiring technique proposed by Maslov and Sneppen (2002), which allows each node to keep a constant degree for all values of p.

Figure 3 illustrates the effects on the propagation dynamics of simple contagions of randomizing ties in a two-dimensional regular lattice with Moore neighborhoods. The abscissa represents the number of timesteps, while the ordinate shows the number of activated nodes. The dashed line shows the growth of a simple contagion on a regular lattice with no perturbation ($p = 0$), and the solid line indicates the growth of a simple contagion on a small world network in which $p = 0.1$. The growth of the activated population is much faster in the small world network, as would be expected from previous results (Newman 2000).

[Figure 3 about here]

The ordinate on the inset shows the rate of propagation as the timesteps t required for the contagion to saturate the network (99% of the nodes), and the abscissa represents the order parameter p. As p increases, t decreases until it reaches the lower bound given by a random network at $p \approx 0.1$. Figure 3 confirms a key insight of the small world model – that propagation rates approach those of random networks while the network still has abundant local structure (Watts and Strogatz, 1998).

We then replicated the small world experiment using the same model, only this time we raised thresholds just above those of the simple contagions used to demonstrate the small worlds effect. As before, we seeded the model with the minimum number of activated nodes to allow a contagion to spread. With simple contagions, only one seed is needed. With higher thresholds, more seeds are required. We therefore selected a single node as the focal node and then activated the neighbors of that node.

Figure 4 shows the effect of random rewiring ($p = 0.1$, constant density) on the propagation of contested (dotted line) and uncontested (solid line) contagions. Figure 4

also reports the effect of adding random ties ($p = 0.1$, increasing density) on the propagation of contested (dark dotted line) and uncontested (dark line) contagions. As above, the growth of the contagion is indicated by the number of active nodes per timestep *t*. Compared to the regular lattice (solid line with circles), contested and uncontested contagions in the constant density model initially spread more slowly on a small world network than on a regular lattice. However, their accelerating growth curves cause these contagions to spread in slightly fewer overall timesteps in the randomized network than in the regular network.[12]

The inset in Figure 4 shows the average propagation rate of complex contagions as the network is perturbed. As before, the rate of propagation is indicated by the average number of timesteps *t* required for the contagion to saturate the network. For both contagions, as *p* increases with constant density, *t* first decreases slightly and then reverses direction, increasing dramatically. For contested contagions with increasing density, both the growth of the activated population and the overall rate of propagation is slower in a small world network than in a regular network. By contrast, increasing the density of random ties only facilitates the propagation of uncontested contagions, consistent with Corollary 2.1. That is because adding ties to unactivated nodes reduces the *proportion* of ties to activated nodes, but has no effect on the *number*.[13]

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12 When we increase the variance in the distribution of τ (while holding the mean constant at $\tau = 0.25$), we find that the number timesteps required to saturate the network increases more dramatically as *p* increases from 0 to 1. This effect becomes even more pronounced as the mean of the distribution is increased above .25, at which point we no longer observe the slight increase in propagation rates around $p = 0.1$ (the level of randomization corresponding to a small worlds network).

13 We therefore simplify subsequent figures by reporting only the effects of rewiring ties, holding density constant.

The steep increase in the number of timesteps required to complete successful cascades when $p \approx 0.1$ is a sign of criticality for the propagation of complex contagions. This is confirmed in Figure 5, which measures the frequency of successful cascades as p increases from 0 to 1, for contested (dotted line) and uncontested (solid) contagions. In both cases, the effect of network perturbation is highly non-linear. We observe not a steady decline but a dramatic shift from almost complete success on each trial, to zero success.

[Figures 4 and 5 about here]

This abrupt change in global dynamics is indicative of a first-order phase transition in cascade behavior. A first order phase transition, such as the transition of water to steam, indicates a radical change in a system's basic properties. In the case of boiling water, the shift in density at the phase transition is sudden and large, requiring complex analytic techniques to model the process (Landau and Lifshitz 1994). For complex contagions, the change is just as striking. This result identifies a critical point for ordered social networks, below which an increase in the number of random ties has almost no effect on the network's ability to propagate complex contagions. However, once the fraction of random ties exceeds this critical point, these contagions can no longer propagate at all. In short, small changes to the network structure, which are imperceptible to individual actors (Watts and Strogatz 1998), can precipitate a radical shift in the collective dynamics of complex contagions.

Figures 4 and 5 assume thresholds at the lower limit for complex contagions. The effects of network perturbation are even more dramatic as thresholds increase further, as shown in Figure 6, for conditions identical to those in Figures 4 and 5 except that $\tau = 3$.

Higher thresholds preclude the initial increase in propagation rates for low but increasing values of p (evident in Figure 4). The effect of p is now monotonic, and the phase transition occurs much earlier, at around $p = 0.03$ instead of $p = 0.1$ (with $\tau = 2$).

[Figure 6 about here]

To sum up, this first experiment demonstrates that long ties between remote social groups accelerate the spread of simple contagions, but can have the opposite effect on complex contagions, even with thresholds that are only slightly higher than those assumed in previous research on cascades in small worlds networks. Simple contagions can spread through a single tie while complex contagions cannot – they require wider bridges. The higher the thresholds, the wider the bridges need to be, and the more random ties that are required to create effective bridges. As more random ties are added, a phase transition eventually transforms the network from one that can sustain complex contagions to one that cannot.

Yet this finding also poses a new puzzle: *How is it possible that complex contagions are able to spread through real social networks*?

An intuitively plausible answer comes from the theory of structural balance (Cartwright & Harrary 1956). What if perturbing local ties causes the network to evolve non-randomly? Structural balance theory implies a tendency for social relations to be transitive, that is, if a node is a member of two dyads, it is more likely that the two dyads will form a closed triad. Thus, if a random tie forms between otherwise distant nodes i and r, the probability increases that another long tie will form between i and a neighbor of r. This second tie increases the width of the bridge between the neighborhoods of i and r.

To test the structural balance hypothesis, we developed an extension of the Newman and Watts (1999) small world model, in which random bridge ties are added to a two-dimensional lattice network ($z = 8$) instead of rewired from exiting ties. We adapted their model, using the parameter $G >> 1$ to limit the number of bridge ties that can be added to each node. Random ties must be added without replacement of existing ties, otherwise the disruption of local structure precludes structural balance. Since uncontested contagions are unproblematic when perturbation increases network density, we focus only on contested contagions.

As in experiment 1, the parameter p ($0 \leq p \leq 1$) governs the probability of perturbing the network with a random tie, where 0 indicates that no ties are added and 1 indicates that G random ties are added to each node. Thus, $p = 0$ corresponds to a regular network and $p = 1$ corresponds (approximately) to a random network with degree G (Newman 2000). As before, between 0 and 1, there is a critical region for p in which there is high local clustering with low characteristic path length, corresponding to a small world network.

This model becomes a "targeted" random tie model by adding a parameter β that biases the destination of the randomly added ties. Let i and r be a random pair who are not neighbors and who have no neighbors in common. Suppose a tie is randomly added between i and r. Now suppose i is randomly chosen to receive a second random tie. The target for i's new tie will be chosen from among the neighbors of r with probability β. When $\beta = 0$, the model corresponds to a standard small worlds model in which bridge ties are formed between randomly selected targets with probability p. When $\beta = 1$, the first tie added to node i will be randomly targeted, but the next time i is randomly

selected to have an additional tie, the target will be selected from the same destination neighborhood[14] as the first. Thus, increasing β increases the width of the bridge between the two neighborhoods. We want to know if this will in turn allow the bridge to support complex contagions at higher activation thresholds.

Figure 7 shows the effects of random and targeted perturbation with $\beta = 0$ and $\beta = 1$, for contested contagions with $\tau = 2/z$ (for comparability with the random perturbation assumed previously). As in Figure 5, increasing p with $\beta = 0$ produces a phase transition in which cascade frequency (solid line) sharply drops to zero around $p = 0.1$. With $\beta = 1$ (dotted line), as p increases, cascade frequencies drop, but instead of a first order phase transition, cascade frequencies maintain moderate levels (slightly higher than 50%).

While this appears to be a promising result for targeted bridge ties, small reductions in β have large effects on the success of cascades. For $\beta = 0.9$ (dark dotted line), cascade frequency drops gradually to zero as p increases toward 1, and for $\beta = 0.8$ (dashed line), there is a steep transition to zero almost as abrupt as the transition for $\beta = 0$. For lower values of β, there is no effect of targeted versus random bridge ties. Further tests with slightly higher thresholds ($\tau = 3/z$) showed that even with $\beta = 1$, targeting bridge ties has the same effect on cascade frequency as random ties.

[Figure 7]

14 This is a generous interpretation of structural balance theory. A literal interpretation requires that the second tie target any of *r*'s *z* neighbors, which could be a node that is distant from the nodes in *r*'s original neighborhood. Under this condition, targeted perturbation is even less effective in allowing propagation of complex contagions.

These results aver the robustness of the results obtained for the random perturbation model, even with thresholds at the minimum for complex contested contagion ($\tau = 2/z$). Moreover, targeting has no effect if ties are rewired (with constant density) rather than added, for the obvious reason that as targeted ties attempt to create effective bridges, the rewiring process erodes the structure of the local neighborhoods. Further still, targeting has no effect if contagions are uncontested, since uncontested contagions are not disrupted by random perturbation (as shown in Figures 4 and 5). In sum, structural balance in tie formation can constrain perturbation so as to increase the width of bridges in complex networks, and this might seem like a plausible explanation for how complex contagions might propagate on perturbed lattices. Surprisingly, this turns out not to be the case. The results we observe for random perturbation are highly robust as we relax the assumption that network evolution is strictly random.

Hybrid Contagions

This leaves open the question as to how complex contagions might nevertheless spread through social networks. An alternative explanation is suggested by Schelling (1978) and Granovetter (1978) – threshold effects. These occur when thresholds are distributed such that low threshold actors trigger those with higher thresholds, and so on. Following Watts (2002), we refer to these low threshold actors as "vulnerable nodes," that is, nodes that are vulnerable to simple propagation. Watts (2002) showed that the size of the connected cluster of vulnerable nodes must be infinitely large in order for a global cascade to occur in an infinitely large and sparsely connected random network. However, for smaller networks ($N = 2000$) of the same density, even a moderate fraction of vulnerable nodes may be sufficient. Watts's results suggest the possibility that a critical

mass of vulnerable nodes may trigger the activation of high-threshold nodes on complex networks.

The introduction of vulnerable nodes complicates the more elementary cases of simple and complex contagions. While thresholds are a property of nodes, simple and complex are properties of contagions, not nodes. Simple contagions never require confirmation from additional sources. For example, disease has a threshold of one for every member of the population (no one has to be infected by two or more carriers to become infected). Similarly, while complex contagions can involve distributed thresholds, they always require contact with two or more sources. For example, collective behaviors that begin with "milling around" indicate that no one is willing to act unless several others do so as well. Innovations that are useless without other adopters likewise require confirmation that more than one other person has adopted.

Some contagions, however, are "hybrid," in that they can be either simple or complex, depending on an individual's activation threshold. Members of a group with the strongest interest in the public goods may be willing to contribute even if no one else has, or if only one other has contributed, and so on. Hybrid contagions have the important property that a network can contain nodes that are vulnerable to simple contagion (as well as self-activating seed nodes that require no contact at all). If this proportion is sufficiently large to take advantage of random ties, it may become possible to propagate hybrid contagions to the remaining high-threshold members of the population even in a highly randomized network. Moreover, the rate of propagation may increase as the network is perturbed.

We tested this hypothesis by introducing vulnerable nodes into the network used for experiment 1. Keeping $\tau_i = 2$ as the maximum threshold in the population, we then varied the fraction of the population with vulnerable nodes ($\tau_i = 1$), repeating the experiment for $\tau_i = 2/z$. Adding vulnerable nodes allows us to evaluate the effect of p on the dynamics of hybrid contagions in the transitional region between simple and complex.

[Figure 8 about here]

Figure 8 shows the frequency of cascades for distributions of τ_i in which 5% (solid line), 10% (solid with circles), and 15% (dotted line) of the nodes are randomly replaced with vulnerable nodes.[15] With 5% vulnerable nodes, the effect of p is slightly different from what we observe in Figure 5 – instead of a phase transition, there is a gradual crossover between cascade success and failure – yet the frequency of cascades still decays to zero as p increases. Increasing the fraction of vulnerable nodes to 0.1 prevents the drop to zero, but frequencies still show a noticeable decline as p approaches 1. With 15% vulnerable nodes, frequencies dip only slightly as p approaches 1. As the percentage of vulnerable nodes reaches $2/z$ (dashed line), they form a sufficient fraction of most neighborhoods to guarantee activation of their higher threshold neighbors, even in a network that is entirely random.[16]

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15 The introduction of the vulnerable nodes increases the variance in the distribution of thresholds such that contagions are no longer complex. We obtained qualitatively similar results using Gaussian and uniform threshold distributions with enough variance to introduce a moderate fraction of nodes that could be activated by a single contact, and by increasing the proportion of nodes with threshold 0 (instead of 1).

16 This result implies that in finite populations the effect of vulnerable nodes is dependent upon z. As neighborhood size increases, the fraction of vulnerable nodes needed to facilitate the propagation of high-threshold contagions decreases.

The effects of vulnerable nodes on propagation rates are consistent with their effects on cascade frequencies. The inset in Figure 8 shows that there is a drop in the number of timesteps required to fully propagate hybrid contagions as p increases from 0 to 0.1, regardless of the number of vulnerable nodes. With 5% and 10% vulnerable nodes, there is no phase transition, but the number of timesteps required to complete a cascade still increases as p approaches 1. For 15% vulnerable nodes, the number of timesteps required for propagation increases only slightly as p approaches 1. When 25% of the nodes are vulnerable, increasing p above 0.1 has the small world effect of approximating the rates on a random network. However, the rates only approach those for simple contagions if the proportion of vulnerable nodes is increased to 50%. And as the maximum threshold increases (e.g., from $2/z$ to $3/z$), so too does the critical mass of vulnerable nodes required to trigger a successful cascade.

Even with these caveats, the experiments with vulnerable nodes suggest how hybrid contagions can spread, even when a majority of nodes have high thresholds. Adding a relatively small fraction (15-20%) of nodes with very low thresholds ($\tau_i = 0$ or $\tau_i = 1$) allows the network to benefit from the addition of a relatively small number of random ties (e.g. $p \approx 0.1$), and vice versa. Just as it takes very few random ties for simple contagions to propagate on a regular lattice about as easily as on a purely random graph, so too it takes only a few innovators (seed nodes) and early adopters (vulnerable nodes) for hybrid contagions to propagate on a perturbed lattice about as easily as would a simple contagion.

A critical mass of low-threshold nodes explains how hybrid contagions can benefit from the perturbation of a lattice even when most nodes have high thresholds.

While most people will only adopt a new fashion or join a new cause after seeing several others do so, there are often a number of "innovators" and "early adopters" who can then provide the critical mass that allows a hybrid contagion to acquire the propagation dynamics characteristic of simple contagions. However, not all populations will contain this critical mass. Some innovations may be so risky to adopt, and some collective actions may be so costly to join, that few if any members are prepared to act alone or to act on first exposure.

Discussion

If targeted perturbation and vulnerable nodes do not explain the spread of complex contagions, the question remains: How do we account for the rapid diffusion of participation in costly collective actions or the adoption of risky innovations? The answer, we suggest, is spatial diffusion.

Rep. Tip O'Neil's insight that "all politics is local" quickly became an aphorism because it resonated with experience in widely divergent political cultures, from ethnic enclaves to high tech boom towns. Our experiments with complex contagions suggest that politics is local not only because of the parochial attitudes of ethnic voters but also because political influence involves relatively high thresholds and therefore depends on the wide bridges that characterize spatial networks.

The local influence principle applies not only to electoral politics but also to the mobilization of social movements. Beginning with McAdam's (1988) seminal study of Freedom Summer, a consistent finding in social movement research is that recruitment is local. Participation in collective action and social movements seems to spread most effectively in populations that are spatially clustered.

A recent example is Hedstrom's (1994) study of the early labor movement in Sweden, in which he shows that participation spread locally, from one residential neighborhood to another. Other studies have also found that social movements tend to spread along spatial contours. In China, the dormitory housing arrangements structured social ties in a way that allowed for easy diffusion of student dissent (Zhao 1998). Similarly, in France, the close quarters of inner city settlements promoted the emergence of violent revolts (Gould 1996). Further, Rogers and Kincaid (1984) show that the diffusion of birth control technology follows spatial patterns of adoption in Korean villages, and Whyte (1954) argues that the diffusion of product adoption in Philadelphia followed spatial residential patterns.

An obvious explanation is the higher probability of propagation between nodes in close physical proximity. As Hedstrom suggests, "The 'closer' that two actors are to one another, the more likely they are to be aware of and to influence each other's behavior" (Hedstrom 1994: 1163). Intuitive support for this claim draws from everyday experience: the spread of disease and fashion requires physical, respiratory, or visual contact. Empirical studies show that social movements tend to spread through contacts between existing participants and their close friends, many of whom are likely to be their neighbors as well.

However, our study suggests an additional advantage of spatial networks that has not been previously noticed. Complex contagions favor spatial networks not only because the bridges between nodes are physically short – i.e., greater physical proximity – but also because the bridges are structurally *wide*. Spatial networks are characterized by overlapping neighborhoods – the ideal medium for the spread of complex contagions.

This structural advantage suggests a new explanation for the widely observed importance of friendship ties as the conduits of recruitment to social movements. The standard explanation is the relational strength of friendship ties. People are more easily influenced by their friends than by strangers or acquaintances. Our study identifies an additional mechanism that has nothing to do with the affective strength of the ties. The mechanism is entirely structural. While friendship networks tend to be highly clustered, they also tend to have substantial overlap between clusters (Hanneman 2005). These overlaps provide the wide bridges needed for complex contagions to propagate.

This principle has a further implication for the spatial diffusion of social movements. While neighbors may be important for the spread of movement participation because they are likely to be friends, it may be the other way around as well: *friends are important because they are likely to be neighbors*. The spatial arrangement of neighborhoods in turn constrains friendship networks to acquire the overlapping clusters needed by complex contagions.

Conclusion

The strength of weak ties is that they tend to be *long* – they connect socially distant locations. Moreover, only a few long ties are needed to give large and highly clustered populations the "degrees of separation" of a random network, in which information and disease can rapidly diffuse. For simple contagions, like the spread of disease or information, a single link between otherwise remote nodes is like a worm hole in social space.

It is tempting to regard this principle as a lawful regularity, in part because it justifies generalization from mathematically tractable random graphs to the clustered

networks that characterize social structures. Nevertheless, our research cautions against generalization. Many social contagions are not like disease or information. The decision to *act* on information is often based not only on the attributes of an innovation but also the observation of prior adopters. The credibility of information or the willingness to adopt a risky innovation often requires independent confirmation. Information about an innovation (including how many have already adopted) can be acquired from a single source, but the *influence* from prior adopters, including direct evidence of their adoption, frequently requires exposure to multiple sources of social reinforcement.

For complex contagions, the relevant attribute of a bridge is not the length of a dyadic tie but the *width* of the bridge, measured by the number of short paths between non-neighbors. Using Watts and Strogatz's original model of a small world network, we found that long ties not only fail to speed up complex contagions, they can even preclude diffusion entirely. Complex contagions benefit not from ties that are long and narrow but from bridges that are wide enough to transmit sufficient social reinforcement.

This finding has profound implications for the strength of weak ties. As a regular lattice is perturbed through the addition of random ties, there are fewer common neighbors to provide multiple sources of confirmation or reinforcement. Thus, while networks with long narrow bridges are useful for spreading information about an innovation or social movement, they can be inefficient for the spread of the decision to *act* on that information if this decision depends on confirmation from multiple sources.

How, then, might we account for the spread of high-threshold contagions? Balance theory explains how wide bridges might form through the perturbation of a regular lattice via the tendency for bridge ties to become triadically enclosed. Although

ties that are randomly added in a clustered network tend to be long, long ties are not always randomly formed. A chance acquaintance between individuals from different neighborhoods increases the chances that a second tie will form between one of these individuals and the neighbors of the other. This second tie increases the likelihood that yet more ties will form between these neighborhoods, and so on, until the bridge grows wide enough for a complex contagion to pass.

The balance-theoretic social mechanism underlying this tie formation process indicates that a small random process (a chance acquaintance between individuals from different neighborhoods) can lead to the formation of a wide bridge between them. Intuitively, this would seem to suggest an explanation for the spread of complex contagions on social networks: tendencies toward structural balance widen the bridges formed by random ties. Surprisingly, this turns out not to be the explanation. The effects we observe for random perturbation are highly robust, even when network evolution is tightly constrained by the need for structural balance.

An alternative explanation for the spread of high-threshold contagions in complex networks is a “threshold effect” caused by a critical mass of vulnerable nodes. The fraction turns out to be surprisingly small, a result that parallels the remarkable discovery made by Watts and Strogatz (1998). They showed that surprisingly few random ties are needed to make simple contagions propagate on highly clustered networks at close to the same rate as on random graphs. We found that comparably few vulnerable nodes are needed to make hybrid contagions propagate on complex networks (although the rates remain somewhat slower than for simple contagions). Vulnerable nodes show that what matters for cascades is not only the mean threshold but the distribution. Schelling (1978)

and Granovetter (1978) proved this for cascades in a fully connected population. We now see that this is true for embedded populations as well. As the mean threshold increases, the effects of perturbation on a clustered network depend decisively on a critical mass of innovators and early adopters. Above critical mass, the effect of random ties is similar to what we observe for simple contagions like information and disease. Below critical mass, the effect is the opposite.

The possibility for "threshold effects" does not mitigate the danger in taking disease and information as archetypes for social diffusion. For complex contagions, the intuitions generated by the small world model can be highly misleading. Higher thresholds fundamentally alter the effects of perturbation on clustered networks in ways that have gone unnoticed in the excitement generated by the discovery of the small world effect. Our findings clearly suggest the need for future studies of social diffusion to take into account the possibility that contagions may be complex, based on thresholds requiring exposure to more than a single activated neighbor. The qualitative differences we discovered between simple and complex contagions should caution network theorists about extrapolating from the spread of disease or information to the spread of participation in political, religious, or cultural movements. These movements may not benefit from "the strength of weak ties" and may even be hampered by processes of global integration that stretch social ties out much "longer" while making them "thinner."

Interestingly, these results may have important implications for the development of public health policies that aim to prevent the spread of infectious diseases. The channels along which disease can spread the quickest may well be similar to the channels along which preventative information can propagate the fastest. However, our study

suggests that these may not be the best pathways for effecting social change. Health reform, especially in cases where public health innovations contravene existing social norms, requires social reinforcement, not simply access to information (Friedman et al. 1993, Latkin et al. 1995, Pulerwitz and Barker 2004). While word of mouth transmission of new ideas may travel as quickly as the spread of a disease, without the social reinforcement necessary to propagate behavioral norms, these ideas may have very little effect on risky behavior (CDC 1997, p. 3-2). Simply put, information may diffuse faster through the long ties found in small worlds, but the information may be less effective in changing resistant or inertial behaviors.

This problem is particularly relevant for public health organizations that rely on peer networks to relay information about disease prevention. Our findings suggest that efforts to change behavioral norms through peer influence should target residential networks rather than more complex structures like friendship or employment networks. Residential neighborhoods have the overlapping clusters that are essential for transmitting social reinforcement.

This applies as well to the spread of social movements. Our study suggests a mechanism that may explain why social movements seem to favor spatial diffusion. For complex contagions, it is not only the physical proximity of the nodes that is important, but also the width of the bridges. Spatial neighborhoods are not only clustered, but more importantly, they overlap. These overlapping nodes create wide bridges between neighborhoods. Spatial proximity – the shortness of the bridge – makes the connection relationally strong, but it is the width of the bridge – not its length – that makes the connection structurally strong.

Appendix

Theorem 1. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2$, holding network density constant and adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for an uncontested contagion approaches 0 for $N >> z$.

Proof

Let S be the set of $z+1$ nodes already activated in a ring lattice of size N and density $z/(N-1)$, where every node has z ordered neighbors (not including neighbors that are randomly added), and $z \geq \tau$. We assume that density is held constant by replacing regular (non-random) ties as random ties are added. We also assume that all nodes have threshold $\tau = 2$, the minimum threshold for complex contagions, and that unactivated nodes do not exert countervailing influence, such that contagions are uncontested. Let A be the number of currently activated nodes. Let i be any unactivated node, none of whose neighbors are activated.

Given $\tau = 2$, there must be two random ties to i from any two nodes in A (see Figure 2). Given activation of i, for a neighbor j of i to then become activated, there must be one random tie to j from any node in A. Given activation of both i and j, their shared neighbor k will have two activated neighbors and will also become activated, and so on. Hence, for $\tau = 2$, the number of random ties $R = 2+1 = 3$. More generally, $R = \tau(\tau+1)/2$.

The probability that $R = 3$ ties form an effective bridge is given by the number of effective configurations divided by the total number of possible configurations. For any z, an effective configuration must include one node i which can be any of the N-A unactivated nodes. This node must have 2 ties to any 2 of the A activated nodes, giving

$(N\text{-}A)A(A\text{-}1)/2$ combinations. There must also be 1 tie from any activated node to any one of i's z neighbors, or Az. The total number of effective combinations C_E is then

$$C_E = (N\text{-A})\text{A}(\text{A-1})Az/2 \quad [1.1]$$

For the limiting case of a very large and sparse network with minimal activated nodes

$$(N >> A = z+1) \text{ and } z = 4, \quad [1.2]$$

$$C_E` = (N\text{-}5)(20)(20)/2 = 200N\text{-}1000 \quad [1.3]$$

For convenience, we can assume that random ties can be formed between any pair of nodes, including those already connected. Thus, each of the three random ties can connect any of the $N(N\text{-}1)/2$ pairs of nodes, giving

$$C_P = (N(N\text{-}1)/2)^3 \quad [1.4]$$

Assuming very large N, we can approximate

$$C_P \approx N^6/8 \quad [1.5]$$

The probability P of an effective bridge between activated nodes and any two inactivated neighbors is then

$$P = C_E / C_P \quad [1.6]$$

$$P \approx 8(200N\text{-}1000)/N^6 \quad [1.7]$$

$$P \approx (1600N\text{-}1000)/N^6 \quad [1.8]$$

Thus, P approaches zero under conditions in which a bridge across the ring will have maximum impact, that is, where $N >> A = z+1$ and $z = 4$. As the proportion of activated nodes increases, P increases but the need for an effective bridge declines. In the limiting condition where all nodes are activated except two,

$$C_E = 2N(N)Nz/2 \quad [1.9]$$

$$C_E = 4N^3 \quad [1.10]$$

$$P = 32N^3/N^6 \quad [1.11]$$

$$P = 32/N^3 \quad [1.12]$$

Thus, where only the seed nodes are activated and bridges are most likely to speed up propagation, the probability that three random ties will form an effective bridge is very close to zero. As the contagion spreads along regular ties, and more nodes are activated, the probability increases, but the need for the bridge declines. Even when all but two nodes are activated, and a bridge will have no effect on propagation, the probability remains extremely small. The smaller the probability, the more random ties that will be needed to create at least one effective bridge.

Corollary 1.1. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2/z$, holding network density constant while adding the minimum number of random ties to bridge across the ring, the probability of creating an effective bridge for a contested contagion approaches 0 for $N >> z$.

Proof

The proof is identical to Theorem 1 except that $\tau = 2/z$. Since density is held constant, it remains the case that $R = 3$.

Corollary 1.2. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2/z$, allowing network density to increase while adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for a contested contagion approaches 0 for $N >> z$.

Proof

The proof is identical to Theorem 1, except that $R > 3$. That is because $\tau = 2/z$ and z remains constant, while the number of neighbors increases from z to $z+1$ if a random tie is added to a node. Let r_i be the minimum number of random ties from A to i that must be created in order for i to be activated by ties to A. Node i can only be activated if the proportion of activated neighbors equals i's threshold $\tau = 2/z$, or

$$r_i/(r_i+z) = 2/z \quad [1.3.1]$$

$$r_i = 2z/(z-2), \quad [1.3.2]$$

If $z = 4$ (the lower limit), $r_i(4) = 4$. If $z = 6$, $r_i(6) = 3$. As z becomes indefinitely large (as a multiple of 2), r approaches 2, but since r_i is an integer, it follows that

$$r_i(z) = 3 \text{ for } z \geq 6. \quad [1.3.3]$$

Keeping in mind that the probability of forming an effective bridge of width 4 is smaller than for a bridge of width 3, we simplify the proof by assuming $r_i = 3$ for all $z \geq 4$.

For a neighbor j of i to become activated, given activation of i,

$$(r_j+1)/(r_j+z) = 2/z \quad [1.3.4]$$

$$r_j = z/(z-2), \quad [1.3.5]$$

If $z = 4$, $r_j = 2$. As z becomes indefinitely large (as a multiple of 2), r_j approaches 1, but since r_j is an integer, it follows that

$$r_j = 2 \quad [1.3.6]$$

Thus, for any $z \geq 4$, 2 ties are needed from A to activated i's neighbor j (who then has $z+2$ neighbors, of whom 3 are activated including i). Given activation of both i and j, their shared neighbor k will have two out of z activated neighbors and will also become activated, and so on. Hence, for $\tau = 2/z$, the number of nodes outside A that must be

activated to form an effective bridge to a node that is not tied to A is $\tau_z = 2$ nodes, and the number of random ties $R = 3+2 = 5$.

The remainder of the proof is identical to Theorem 1 but the probabilities of an effective bridge are even smaller, given that 5 ties are now required instead of 3.

Corollary 1.3. For a ring lattice of size N, degree $z \geq 4$, and threshold $\tau = 2$, allowing network density to increase while adding the minimum number of random ties to bridge across the ring, the probability of forming an effective bridge for an uncontested contagion approaches 0 for $N >> z$.

Proof

The proof is identical to Theorem 1. Although density is allowed to increase as random ties are added, it remains the case that $R = 3$ since the contagion is uncontested, and thus $\tau = 2$, regardless of the number of neighbors.

Theorem 2. For a ring lattice identical to that for Theorem 1 (including constant density and uncontested contagion), if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Proof

Let R be the minimum number of random ties required for an effective bridge between activated and unactivated nodes. From Theorem 1, the probability that these ties create an ineffective bridge approaches unity as N increases and $N >> z$ and $N >> A$. Suppose the bridge is ineffective and a random tie happens to link two nodes, i and j,

each with $z/2$ neighbors in S and on opposite ends of S. Both i and j then each have $z/2$ activated neighbors in S. In order to hold density constant, the addition of the ij tie requires that one of the existing ties to either i or j must be deleted.

Suppose the break occurs between i and one of its activated neighbors in S, such that i cannot be activated. (Note that the reasoning is identical if the break occurs on a tie to j rather than i.)The cascade can then only proceed beyond i if a second random tie is formed, either to i or to i's closest unactivated neighbor k (see Figure 2). Both i and k have one activated regular neighbor, and the addition of a random tie to a second activated neighbor allows the cascade to proceed.

Suppose instead the break occurs between i and k, such that k cannot be activated. The cascade can then only proceed beyond k if a second random tie is formed, either to k or to $\underline{k}$'s closest unactivated neighbor l. Both k and l will already have one activated regular neighbor (since k and i share one activated neighbor and l remains tied to i), and the addition of a random tie to a second activated neighbor allows the cascade to proceed.

However, a random tie anywhere else will not circumvent the broken ik tie, because a single activated neighbor is not sufficient. This applies as well if the break occurs before the cascade reaches i. Regardless of the location of the break (before or after i), the repair tie must be formed between an activated node and a node that is within $z/2$ nodes of the break. The probability of such a repair is simply the number of possible repairs ($z/2$) divided by the number of dyads ($N(N\text{-}1)/2$), or $z/(N(N\text{-}1)$. The probability approaches zero as N increases and $N >> z$.

A random tie between i and j is the worst-case scenario for the effect of random rewiring on the extent of propagation. More generally, the expected effect is determined

by the longest tie that is randomly created in R tries, where length is measured as the number of nodes from one end to the other, not passing through S. Each node has $1/N$ chance of being selected in each of R tries. For a ring lattice with any degree z and size $N >> z$, the length of the longest tie can be expected to be approximately $RN/(R+1)$. For $R = 3$, cascades can be expected to reach only about $N/4$ nodes, for any N and z with $N >> z$.

Corollary 2.1. For a ring lattice identical to that for Theorem 1 except that network density increases as random ties are added, and if random ties fail to create an effective bridge, each additional random tie has no effect on the number of nodes that a cascade can be expected to reach.

Proof

Corollary 2.1 shows that there is no disruption of local propagation if the contagion is uncontested and the addition of random ties leads to increased density rather than deleted ties. Suppose a random tie happens to link two nodes, i and j, whose immediate neighbors are in S and on opposite ends of S. Both i and j then each have two activated neighbors in S. With the addition of a random tie between i and j, these nodes now each have $z+1$ neighbors, of whom two (the two neighbors within S) are activated. In the absence of countervailing influence by unactivated nodes, both i and j can be activated, and the cascade can escape the seed neighborhood.

Corollary 2.2. For a ring lattice identical to that for Theorem 1 except that the contagion is contested, if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Proof

The proof is identical to that for Theorem 2. It makes no difference whether $\tau = 2/z$ or $\tau = 2$ when density is held constant as random ties are formed.

Corollary 2.3 For a ring lattice identical to that for Theorem 1 except that network density increases as random ties are added and the contagion is contested, if random ties fail to create an effective bridge, each additional random tie reduces the number of nodes that a cascade can be expected to reach.

Proof

The proof is identical to that for Theorem 2, except that disruption of local propagation cannot be repaired by formation of a random tie to k or l. Let R be the minimum number of random ties required for an effective bridge between activated and unactivated nodes. From Theorem 1, the probability that these ties create an ineffective bridge approaches unity as N increases and $N >> z$ and $N >> A$. Suppose the bridge is ineffective and a random tie happens to link two nodes, i and j, each with $z/2$ neighbors in S and on opposite ends of S. Both i and j then each have $z/2$ activated neighbors in S. With the addition of a random tie between i and j, i and j now each have $z+1$ neighbors, of whom $z/2$ are activated. Since $(z/2)/(z+1) < 2/z$, neither randomly connected node can be activated, and the cascade is unable to escape the seed neighborhood.

Now suppose, in addition to the *ij* tie, another random tie is created from *S* to *k*, the neighbor closest to *i* along the regular lattice and not in *S.* Since *i* lies between *S* and *k, k* is one node farther from *S* than is *i* and thus *k* has one less neighbor in *S* than does *i*, prior to the addition of a random tie to *k*. However, the random tie from *S* to *k* gives *k* one additional activated neighbor, bringing the total to $z/2$. Node *k* now has $z+1$ neighbors, of whom $z/2$ are activated. Since $(z/2)/(z+1) < 2/z$, the random tie to *k* does not allow the cascade to escape the *ij* block. The remainder of the proof is identical to that for Theorem 2.

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Figure 1. Many social diffusion processes involve complex contagions, whether contested or uncontested. All simple contagions are necessarily uncontested.

Figure 2. Simple and complex contagions on a ring lattice with $z = 4$. Solid nodes are activated. An increase in thresholds from one to two triples the width of the bridge needed from s to k via i and j, from $w_{ks}(1) = 1$ to $w_{ks}(2) = 3$. Dashed lines indicate deleted ties, which have no effect on the simple contagion but prevent leftward propagation of the complex contagion.

Figure 3. Random ties increase the rate of simple contagions ($N = 2000$, averaged over 100 realizations). The dashed line shows the average number of nodes activated per timestep t in a regular lattice ($p = 0$). For $p = 0.1$ (solid line), the growth of the activated nodes is steeply concave, activating the entire network in many fewer timesteps. The inset shows the change in the rate of propagation as the network is rewired. With increasing numbers of random ties, the timesteps required for the contagion to saturate the network decreases until, at $p \approx 0.1$, the rate of propagation is approximately that of a random network.

Figure 4. Random ties inhibit minimally complex contagions ($\tau = 2$, $\tau = 2/z$, $N = 2000$, averaged over 100 realizations). Dotted lines indicate contested contagions and solid lines indicate uncontested contagions in a small world network ($p = 0.1$), with lighter lines for constant density and heavier lines for increasing density. The solid line with circles provides a benchmark ($p = 0$). With constant density, both contested and uncontested

contagions initially spread more slowly on a randomized network, but network saturation requires slightly fewer timesteps. With increasing density, contested contagions also spread more slowly on a randomized network than on a regular lattice, while uncontested contagions grow much more rapidly on the randomized networks. The inset shows the average number of timesteps to saturate the network. As the network is rewired, there is a slight increase in the propagation rates of minimally complex contagions with constant density. However, with increasing numbers of random ties, propagation rates steeply increase toward infinity. For increasing density, contested contagions propagate more slowly on randomized networks then on a regular lattice, while rates for uncontested contagions increase monotonically with p.

Figure 5. Random ties reduce the frequency of minimally complex contagions ($\tau = 2$, $\tau = 2/z$, $N = 2000$, averaged over 100 realizations). The frequency of cascades for contested contagions (dotted line) and uncontested contagions (solid line) is unaffected for low values of p. However, as p increases above 0.1, the introduction of random ties reduces the width of local bridges, and the frequency of cascades drops off sharply. Above $p = 0.13$, cascades are entirely inhibited.

Figure 6. Effects of random ties become more pronounced as thresholds increase ($\tau = 3$, $N = 2000$, averaged over 100 realizations). The rate and frequency of complex contested contagions (dotted line) and uncontested contagions (solid line) begin to deteriorate rapidly at lower levels of randomization as compared with Figures 3 and 4 (at around $p = 0.03$, compared to $p = 0.1$ with $\tau = 2$).

Figure 7. Perturbation inhibits propagation of complex contagions, even when targeted ($\tau = 0.25$, $N = 2000$, averaged over 100 realizations). For $\beta = 0$ (solid line) ties are added randomly, and the results show the expected steep decline in cascade frequencies as p increases. For $\beta > 0$, the evolution of network topology is not random. Instead, new ties are "targeted" to form closed triads, as predicted by structural balance theory. For $\beta = 1$ (dotted line), there is a decrease in cascade frequency with increasing p, but approximately 50% of cascades succeed even with $p = 1$. However, for $\beta = 0.9$ (dark dotted line) cascade frequency goes to zero as p approaches 1, and for $\beta = 0.8$ (dashed line) we observe only a slight improvement compared to $\beta = 0$.

Figure 8. Perturbation promotes propagation of hybrid contagions ($\tau = 2$, $N = 2000$, averaged over 100 realizations). For 5% vulnerable nodes (solid line), we observe a gradual decline from high cascade frequencies to zero cascades for $p > 0.1$. The inset shows that a decrease in timesteps for small values of p is followed by a steep increase above $p = 0.1$. For 10% vulnerable nodes (solid line with circles), cascade frequencies remain positive but drop markedly as p approaches 1, and timesteps level off for $p > 0.3$. For 15% vulnerable nodes (dotted line), p has little effect on cascade frequency, except for a small dip as p approaches 1. Timesteps required for propagation increase only slightly as p approaches 1. For 25% vulnerable nodes (dashed line), there is no effect of random ties on cascade frequencies, and increasing p does not reduce propagation rates.

Figure 1

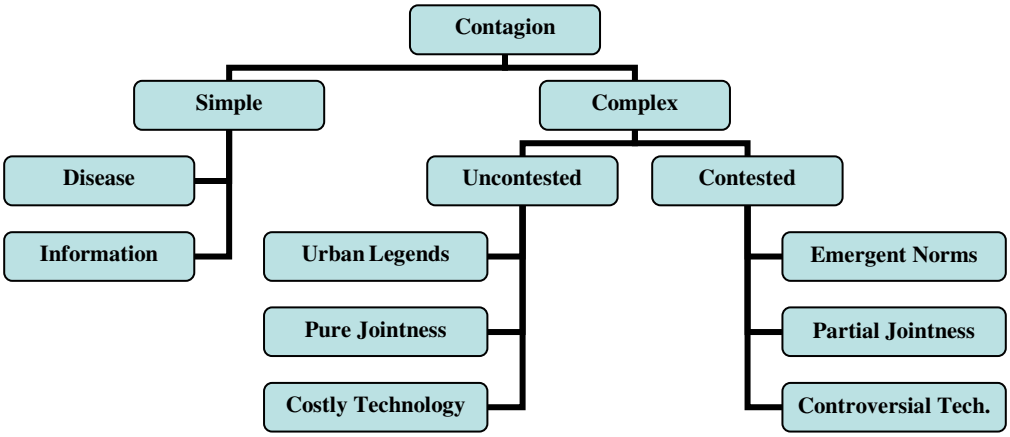

Contagion
Simple
Complex
Disease
Information
Uncontested
Contested
Urban Legends
Pure Jointness
Costly Technology
Emergent Norms
Partial Jointness
Controversial Tech.

Figure 2

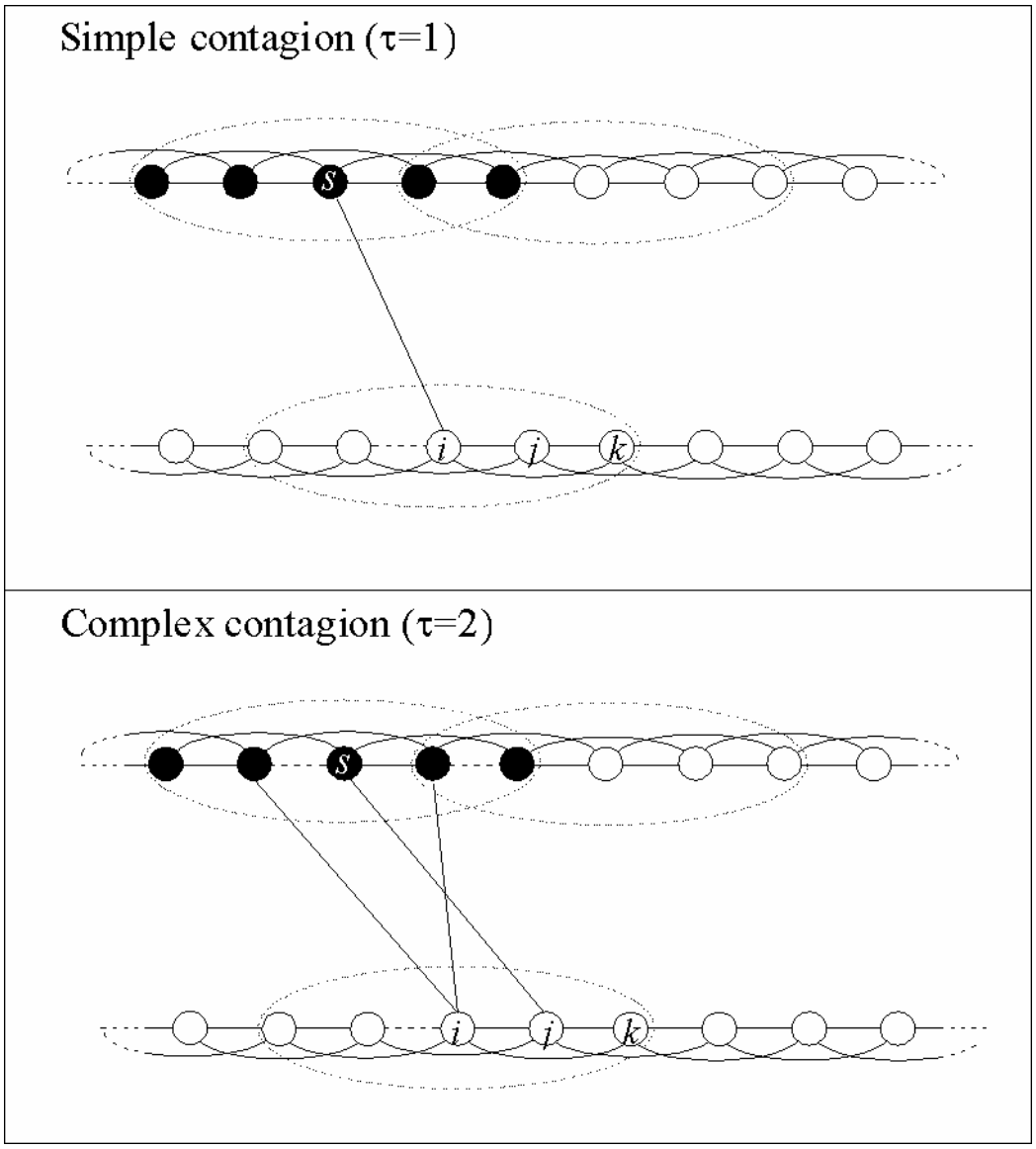


Figure 3

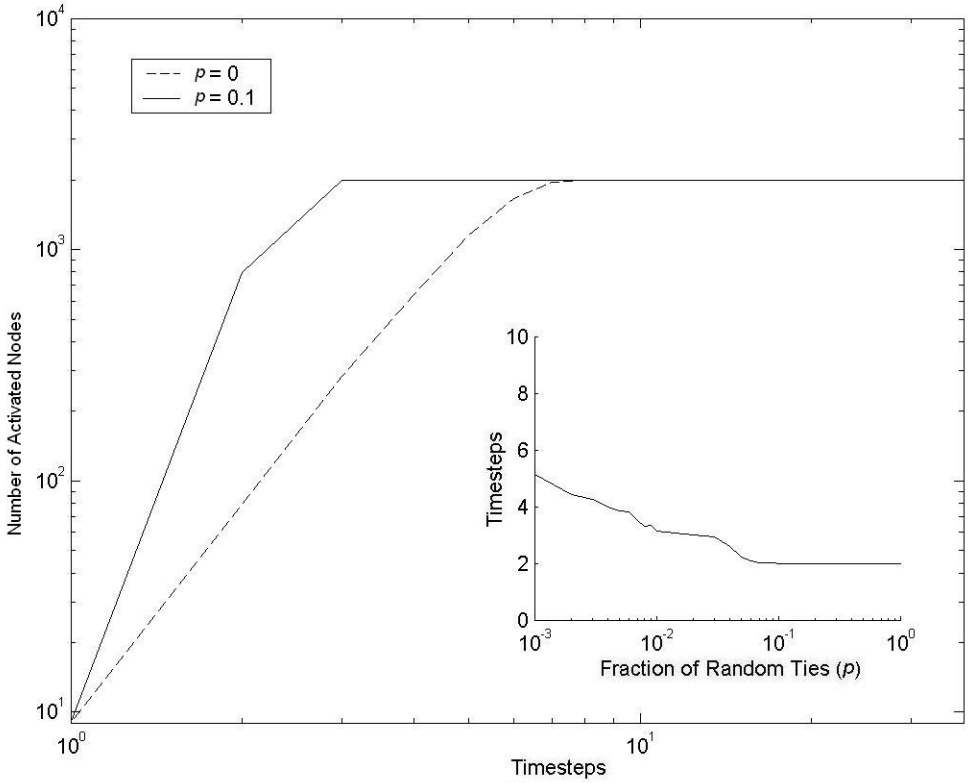

p = 0
p = 0.1
Number of Activated Nodes
Timesteps
Timesteps
Fraction of Random Ties (p)

Figure 4

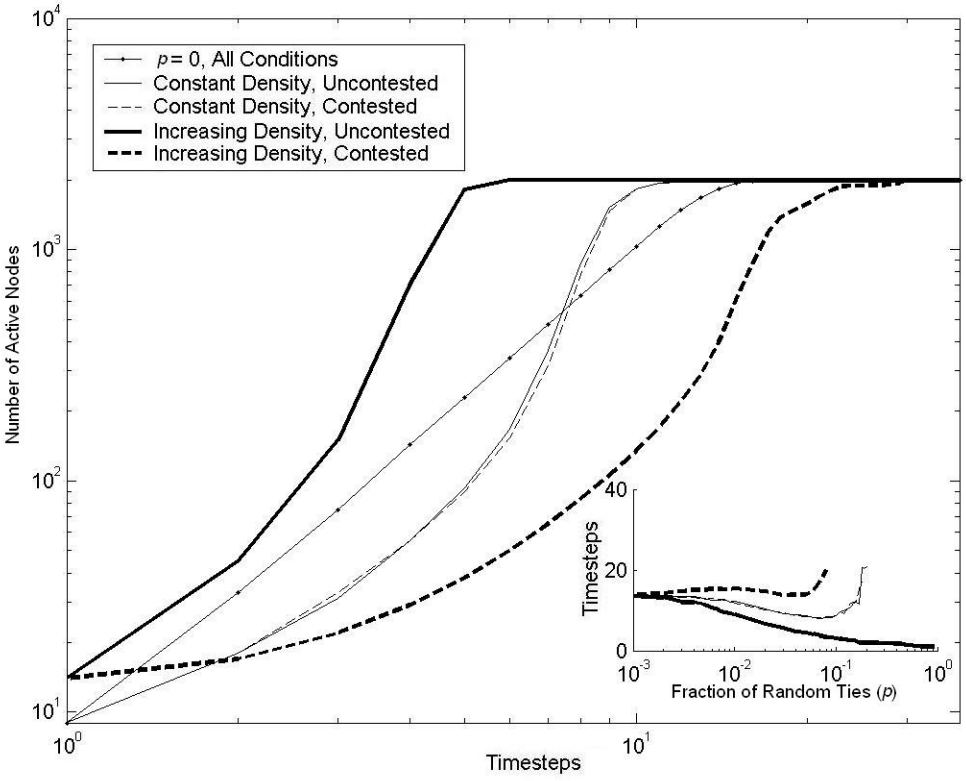

p = 0, All Conditions
Constant Density, Uncontested
Constant Density, Contested
Increasing Density, Uncontested
Increasing Density, Contested
Number of Active Nodes
Timesteps
Timesteps
Fraction of Random Ties (p)

Figure 5

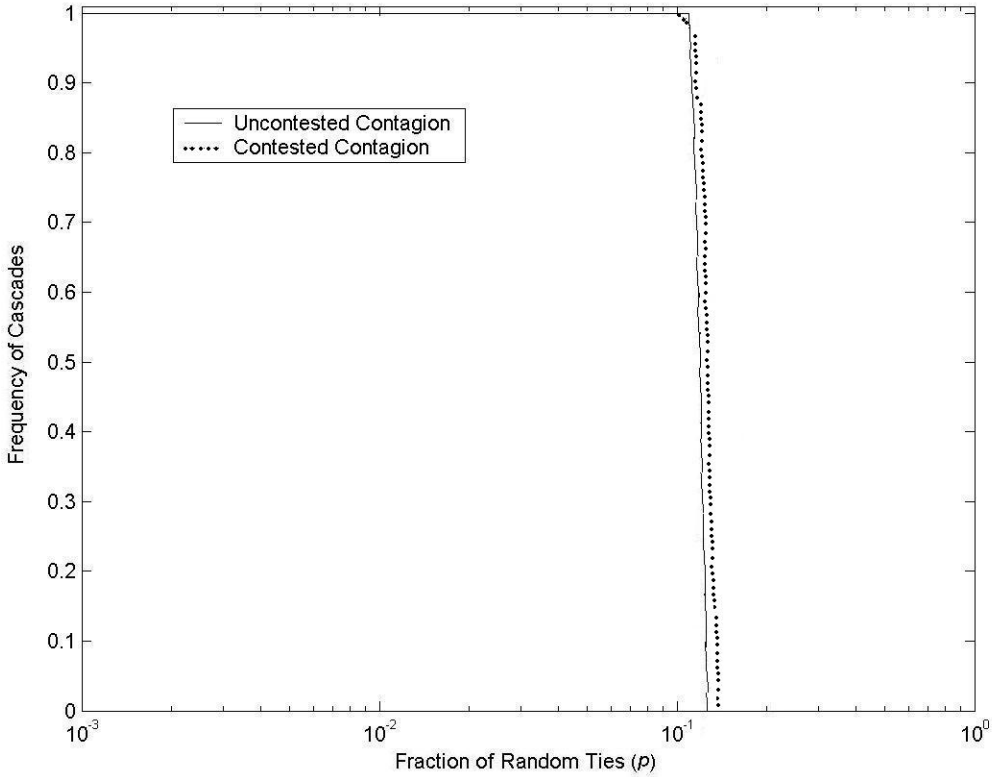

Uncontested Contagion
Contested Contagion
Frequency of Cascades
Fraction of Random Ties (p)
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
10^-3
10^-2
10^-1
10^0

Figure 6

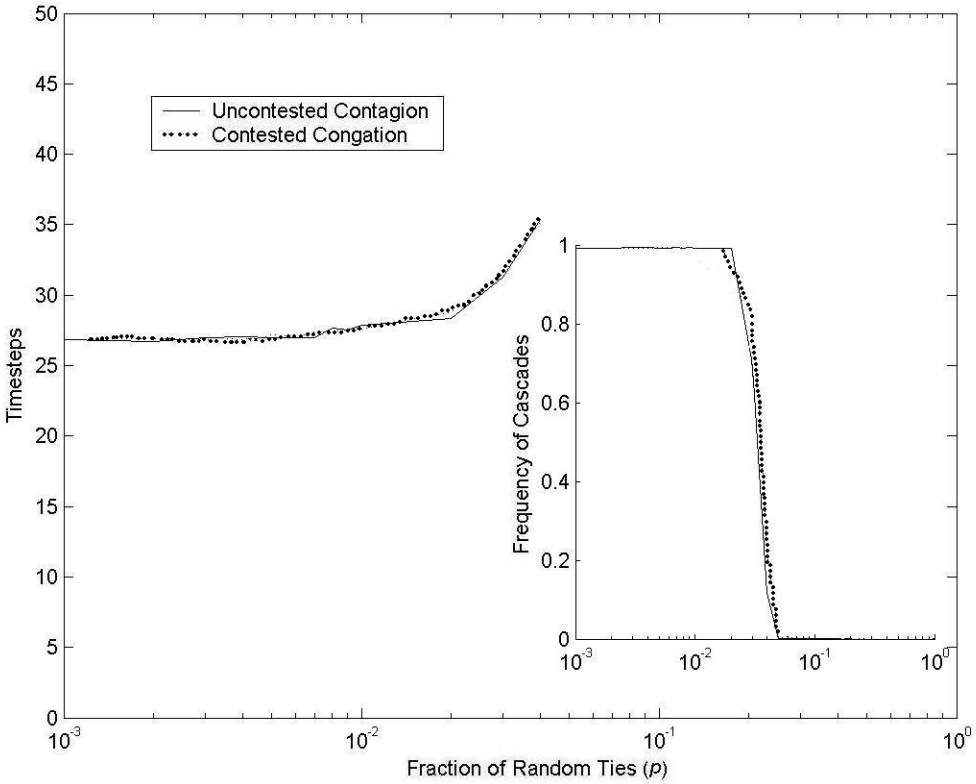

Uncontested Contagion
Contested Congation
Timesteps
Fraction of Random Ties (p)
Frequency of Cascades

Figure 7

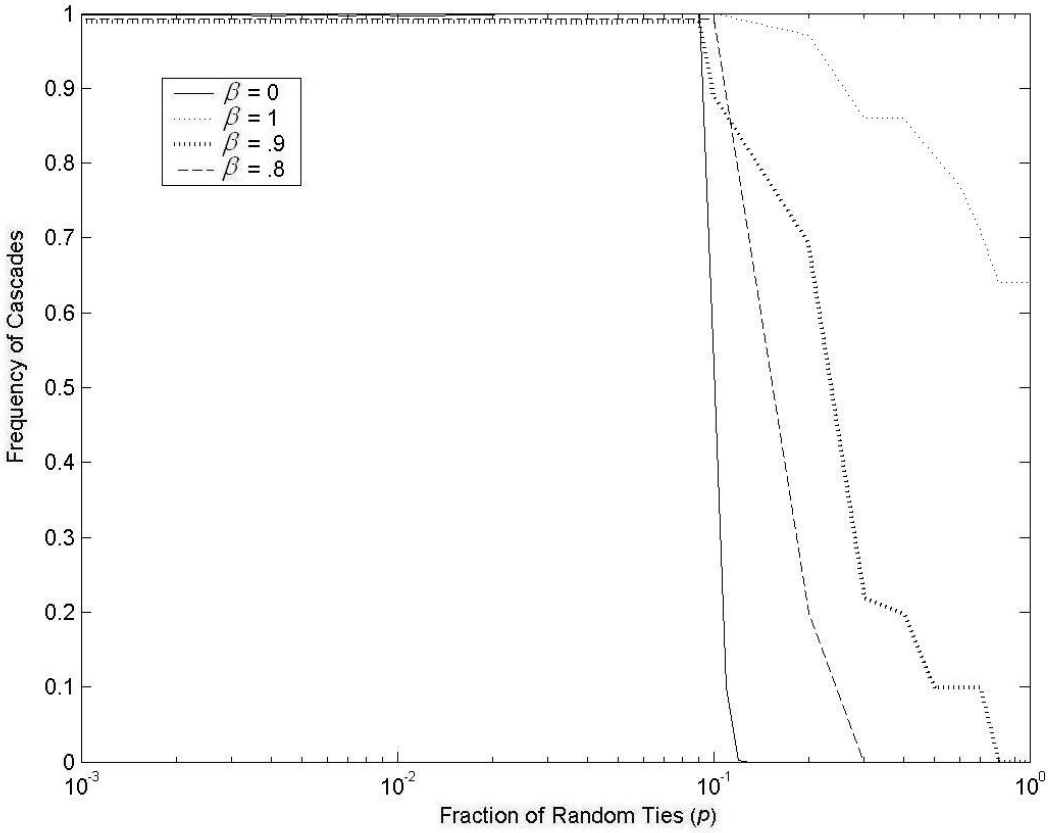
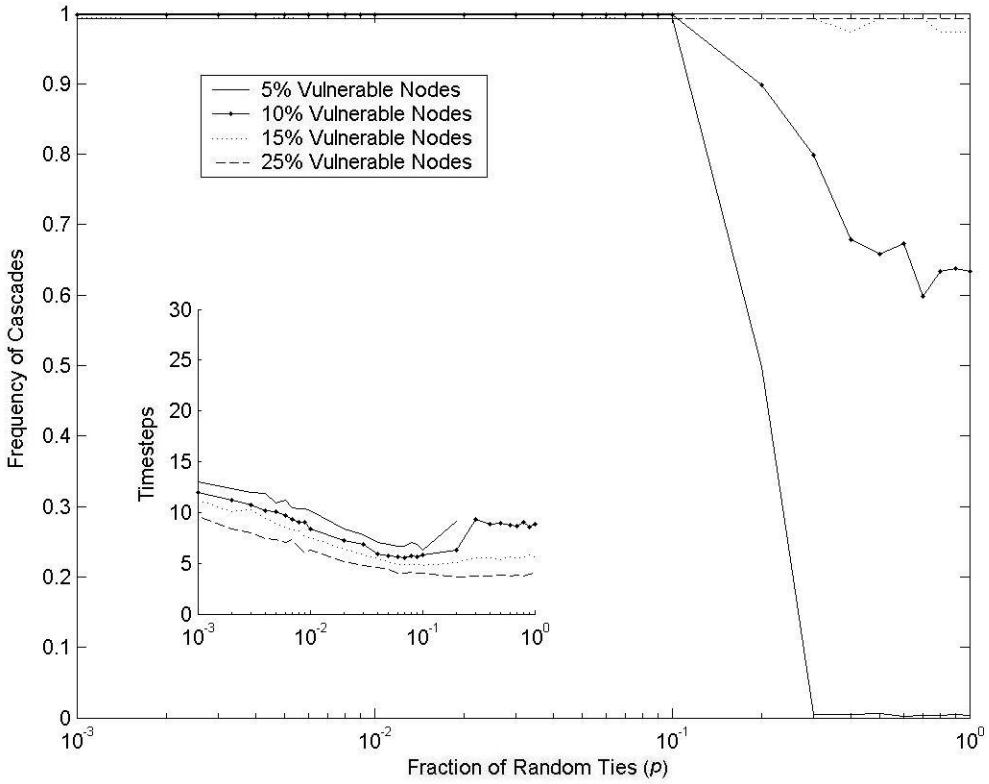

β = 0
β = 1
β = .9
β = .8
Frequency of Cascades
Fraction of Random Ties (p)
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
10^-3
10^-2
10^-1
10^0

Figure 8


5% Vulnerable Nodes
10% Vulnerable Nodes
15% Vulnerable Nodes
25% Vulnerable Nodes
Frequency of Cascades
Fraction of Random Ties (p)
Timesteps