11-2007

Complex Contagions and the Weakness of Long Ties

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Recommended Citation

Centola, D. (2007). Complex Contagions and the Weakness of Long Ties. *American Journal of Sociology, 113* (3), 702-734. https://doi.org/10.1086/521848

Damon Centola was affiliated with Harvard University during the publication of this article.

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Abstract

The strength of weak ties is that they tend to be long—they connect socially distant locations, allowing information to diffuse rapidly. The authors test whether this “strength of weak ties” generalizes from simple to complex contagions. Complex contagions require social affirmation from multiple sources. Examples include the spread of high-risk social movements, avant garde fashions, and unproven technologies. Results show that as adoption thresholds increase, long ties can impede diffusion. Complex contagions depend primarily on the width of the bridges across a network, not just their length. Wide bridges are a characteristic feature of many spatial networks, which may account in part for the widely observed tendency for social movements to diffuse spatially.

Disciplines

Communication | Social and Behavioral Sciences

Comments

Damon Centola was affiliated with Harvard University during the publication of this article.

Complex Contagions and the Weakness of Long Ties[1]

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The strength of weak ties is that they tend to be long—they connect socially distant locations, allowing information to diffuse rapidly. The authors test whether this "strength of weak ties" generalizes from simple to complex contagions. Complex contagions require social affirmation from multiple sources. Examples include the spread of high-risk social movements, avant garde fashions, and unproven technologies. Results show that as adoption thresholds increase, long ties can impede diffusion. Complex contagions depend primarily on the width of the bridges across a network, not just their length. Wide bridges are a characteristic feature of many spatial networks, which may account in part for the widely observed tendency for social movements to diffuse spatially.

Most collective behaviors spread through social contact. From the emergence of social norms (Centola, Willer, and Macy 2005), to the adoption of technological innovations (Coleman, Katz, and Menzel 1966), to the growth of social movements (Marwell and Oliver 1993; Gould 1991, 1993; Zhao 1998; Chwe 1999), social networks are the pathways along which these "social contagions" propagate. Studies of diffusion dynamics have demonstrated that the structure (or topology) of a social network can have

[1] The authors wish to express their gratitude to the National Science Foundation for support of this research through Human and Social Dynamics grant SES-0432917 and through an IGERT fellowship for the first author. We also thank the Robert Wood Johnson Foundation for support of the first author. We thank Duncan Watts, Steve Strogatz, Jon Kleinberg, Vlad Barash, Chris Cameron, and Stephen Moseley for useful comments and suggestions. Direct correspondence to Damon Centola, Institute for Quantitative Social Science, 1730 Cambridge Street, Harvard University, Cambridge, Massachusetts 02138. E-mail: dcentola@rwj.harvard.edu

0002-9602/2007/11303-0003$10.00

important consequences for the patterns of collective behavior that will emerge (Granovetter 1973; Newman, Barabasi, and Watts 2006). In particular, "weak ties" connecting actors who are otherwise socially distant can dramatically accelerate the spread of disease, the diffusion of job information (Granovetter 1973), the adoption of new technologies (Rogers 1995), and the coordination of collective action (Macy 1990). As Granovetter puts it (1973, p. 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." This insight has become one of the most widely cited and influential contributions of sociology to the advancement of knowledge across many disciplines, from epidemiology to computer science.

Nevertheless, the central claim of this study is the need to circumscribe carefully the scope of Granovetter's claim. Specifically, while weak ties facilitate diffusion of contagions like job information or diseases that spread through simple contact, this is not true for "whatever is to be diffused." Many collective behaviors also spread through social contact, but when these behaviors are costly, risky, or controversial, the willingness to participate may require independent affirmation or reinforcement from multiple sources. We call these "complex contagions" because successful transmission depends upon interaction with multiple carriers. Using formal models, we demonstrate fundamental differences in the diffusion dynamics of simple and complex contagions that highlight the danger of generalizing the theory of weak ties to "whatever is to be diffused." Network topologies that facilitate diffusion through simple contact can have a surprisingly detrimental effect on the spread of collective behaviors that require social reinforcement from multiple contacts. This is not to suggest that complex contagions never benefit from weak ties, but to demonstrate the need to identify carefully the conditions under which they can.

FROM WEAK TIES TO SMALL WORLDS

"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 tie as a conduit of information. Weak ties connect acquaintances who interact less frequently, are less invested in the relationship, and are 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, p. 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 diffusion, cohesion, and integration of a social network by linking otherwise distant nodes. Granovetter's insight is that ties that are weak in the relational sense—that the relations are less salient or frequent—are often strong in the structural sense—that they provide shortcuts across the social topology. Although casual friendships are relationally weak, they are more likely to be formed between socially distant actors with few network "neighbors" in common.[2] These "long ties" between otherwise distant nodes provide access to new information and greatly increase the rate at which information propagates, despite the relational weakness of the tie as a conduit.[3]

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 close friends will not help them learn about opportunities, developments, or new ideas in socially distant settings. That is the weakness of their strong ties.

It has become a truism that diffusion over social and information networks displays the regularity that Granovetter (1973) characterized as "the strength of weak ties." This insight has changed the way sociologists think about social networks and has informed hundreds of empirical studies in the four decades since its publication, including studies of adolescent peer group formation (Shrum and Cheek 1987), sex segregation (McPherson and Smith-Lovin 1986), residential segregation (Feld and Carter 1998), banking regulation (Mizruchi and Stearns 2001), collective action (Macy 1990), and immigration (Hagan 1998), to name a few.

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

[2] A "neighbor" refers to any type of social or physical contact—a friend, co-worker, cousin, etc.—and is not limited to a residential neighbor. A "neighborhood" is a focal node plus the set of these contacts, and the size of the set of neighbors is the "degree" of that node.

[3] We use the term "long" rather than "weak" to avoid confusion between length and strength. The length of a tie is its range, which in graph theory is the geodesic that it spans, ranging from two to the diameter of the network (the maximum geodesic). The length of a geodesic is the minimum number of edges between any two nodes. The mean geodesic is the standard measure of the connectivity of a network. The expression "long tie" is shorthand for "long range tie."

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 in-group 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 mean geodesic (the shortest path between two nodes averaged over all pairs).

Granovetter proposed the strength of weak ties as an explanation for the spread of job information through friends and acquaintances. Extensions of the idea to the "small world problem" by physicists and mathematicians (Newman 2000; Watts and Strogatz 1998) have focused on the spread of disease. More recently, the small worlds model has been generalized to the diffusion of collective behavior in a variety of contexts, including political organizing (Hedstrom, Sandell, and Stern 2000), financial markets and banking (Davis, Yoo, and Baker 2003; Stark and Verdes 2006), cultural interaction (Klemm et al. 2003), professional collaboration (Uzzi and Spiro 2005; Burt 2004), and organizational forms (Ruef 2004).

It is understandable that the small world principle would be generalized from information and disease to "whatever is to be diffused." Most diseases are communicable, which means that individuals do not spontaneously generate the infection; they acquire it from a carrier. Similarly, much of what we know is not independently discovered; rather, we obtain the information from others. It is the same with collective behavior. Strikes, fads, social norms, and urban legends do not usually become widespread because individuals independently and spontaneously come up with the same idea or belief. We adopt the idea from someone else who is acting on it and then pass it on to others. Behaviors, like diseases, can be contagious.

Granovetter (1978) and Schelling (1978) modeled this contagion process as a threshold effect in which a small number of "seeds" can trigger a chain reaction of adoption, leading to a population-wide cascade of participation in collective behavior. By "threshold," they mean the number of activated contacts required to activate the target. Except for the seeds, the rest of the population is assumed to have a nonzero threshold of activation. In order to get sick, learn about something, or join in a collective behavior, we need direct or indirect contact with at least one person

who is currently infected, knows about this thing, or is already participating.

FROM SIMPLE TO COMPLEX CONTAGIONS

This similarity among different kinds of contagions invites generalization of the small world principle from the spread of information and disease to the spread of collective behavior. The spread of new technologies among farmers (Ryan and Gross 1943) and medical innovations among doctors (Coleman et al. 1966), the growth of strikes (Klandermans 1988) and social movements (Marwell and Oliver 1993; Opp and Gern 1993; McAdam 1988), and the seemingly irrational behavior of a maddening crowd (McPhail 1991) all depend upon social contact between participants and the co-workers, friends, or acquaintances whom they recruit. Observed from a distance, these cascades resemble an epidemic and may have isomorphic functional forms (e.g., an S-shaped adoption curve). It is not surprising, then, that conclusions drawn from research on epidemics and information networks would be generalized to the spread of collective behaviors.

The problem is that while all contagions have a minimum threshold of one, the range of nonzero thresholds can be quite large. For communicable diseases and information, the threshold is almost always exactly one. If you are infected with a rhinovirus from your child, there is no need also to be infected by your spouse. You are likely to catch a cold and to pass it on to others. Similarly, if you are told the score of the afternoon's soccer match, there is no need to keep asking if anyone knows who won before spreading the news further. These are examples of simple contagions, in that contact with a single source is sufficient for the target to become informed or infected. While information and disease are archetypes of simple contagions,[4] some collective behaviors can also spread through simple contact. A familiar example is a highly contagious rumor that spreads on first hearing, from one person to another. Or consider the tendency for cars to travel in clusters on a two-lane highway. The clusters form because the first car in each cluster is traveling slower than the car in front of it, and this normative speed then spreads to the cars behind who slow down to match the speed of the car in front. More generally,

[4] Some viruses may require infection by two different persons before one can contract the disease, and information that involves "putting two and two together" could require exposure to two different persons, each providing one piece of the puzzle. More importantly, while hearing about a job the first time is usually sufficient, gossip may not be believed until confirmed by independent sources. In such cases, information and disease are complex contagions.

the familiar concept of a "domino effect" refers to a tipping process in which each actor responds to a single neighbor through simple contact.

However, many collective behaviors involve complex contagions that require social affirmation or reinforcement from multiple sources. As McAdam and Paulsen (1993, p. 646) observe, "the fact that we are embedded in many relationships means than any major decision we are contemplating will likely be mediated by a significant subset of those relationships." The distinction between simple and complex refers to the number of sources of exposure required for activation, not the number of exposures. A contagion is complex if its transmission requires an individual to have contact with two or more sources of activation. Depending on how contagious the disease, infection may require multiple exposures to carriers, but it does not require exposure to multiple carriers. 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. Each contact with the same carrier counts as an additional exposure.

By contrast, for complex contagions to spread, multiple sources of activation are required since contact with a single active neighbor is not enough to trigger adoption. There are abundant examples of behaviors for which individuals have thresholds greater than one. The credibility of a bizarre urban legend (Heath, Bell, and Sternberg 2001), the adoption of unproven new technologies (Coleman et al. 1966), the lure of educational attainment (Berg 1970), the willingness to participate in risky migrations (MacDonald and MacDonald 1974) or social movements (Marwell and Oliver 1993; Opp and Gern 1993; McAdam and Paulsen 1993), incentives to exit formal gatherings (Granovetter 1978; Schelling 1978), or the appeal of avant garde fashion (Crane 1999; Grindereng 1967) all may depend on having contacts with multiple prior adopters.

Mechanisms of Complex Contagion

There are at least four social mechanisms that might explain why complex contagions require exposure to multiple sources of activation.

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. For example, Coleman et al. (1966) 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 urban legends (Heath et al. 2001) and folk knowledge (Granovetter 1978) generally depends upon multiple confirmations of the story before there is sufficient credibility to report it to others. Hearing the same story from different people makes it seem less likely that surprising information is nothing more than the fanciful invention of the informant. The need for confirmation becomes even more pronounced when the story is learned from a socially distant contact, with whom a tie is likely to be relationally weak.
3. *Legitimacy.* Having several close friends participate in a collective action often increases a bystander's acceptance of the legitimacy of the movement (Finkel, Muller, and Opp 1989; Opp and Gern 1993; McAdam and Paulsen 1993). Decisions about what clothing to wear, what hairstyle to adopt, or what body part to pierce are also highly dependent on legitimation (Grindereng 1967). Nonadopters are likely to challenge the legitimacy of the innovation, and innovators risk being shunned as deviants until there is a critical mass of early adopters (Crane 1999; Watts 2002).
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 Aminzade and McAdam (2002).

The Structural Weakness of Long Ties

The "strength of weak ties" and "six degrees of separation" have become familiar principles across the social sciences and beyond, with practical implications for unprecedented access to online information, as well as sobering implications for the spread of disease. Generalizations of the small world principle to the spread of collective behaviors implicitly assume that network properties conducive to the spread of disease and information are also conducive to the spread of complex contagions. This assumption is appealing for two reasons. First, as noted above, simple and complex contagions depend on social contact to spread and often display similar S-shaped adoption curves. The second reason is methodological. Simple contagions can be studied on random networks, which are highly amenable to analytic treatment (Erdos and Renyi 1959). Moreover, mathematical approximations can be made for simple contagions (Watts 2002), which cannot be used for those that require multiple sources of activation. In short, the assumption that the global properties of complex contagions can be extrapolated from the properties of simple contagions is not only highly intuitive, it is also analytically convenient. If this assumption is correct, then research on a broad range of sociological problems can benefit from epidemiological research and from studies of information flows (such as Granovetter's study of job search). However, if the assumption is wrong, then generalizing the "strength of weak ties" to the spread of collective behaviors could lead to fundamental errors in our understanding of the effects of network topology on diffusion processes.

The present study provides a theoretical test of this assumption, using available empirical research to evaluate our analytical and computational findings.[6] We show that for complex contagions, long ties can be weak in both of Granovetter's meanings, structural as well as relational. The implication of the relational meaning is immediately apparent. A low level of trust and familiarity between socially distant persons means the relationship is weak, and this inhibits the ability of one person to influence the other. What is not at all obvious is that long ties can also have a *structural* weakness—they are nontransitive. For the spread of information, transitive ties between friends tend to be redundant, such that we hear the same thing from multiple friends. However, when activation requires confirmation or reinforcement from two or more sources, the transitive structure that was redundant for the spread of information now becomes an essential pathway for diffusion. Thus, while weak ties are

[6] For a technical introduction to the propagation dynamics of high-threshold contagions, see Morris (2000), Watts (2002), and Centola, Eguiluz, and Macy (2007).

beneficial for the spread of new information precisely because they are nonredundant, for complex contagions uniqueness becomes a weakness rather than a strength.

This structural weakness of long ties reflects a qualitative difference between simple and complex contagions. For simple contagions, what matters is the length of the bridge between otherwise distant nodes. For complex contagions, the effect of bridges depends not only on their length (the range that is spanned) but also on their width.

In network terminology, a "bridge" links two components of an otherwise disconnected network, and a "local bridge" links otherwise disjoint neighborhoods within a component, where disjoint means that the two neighborhoods have no members in common. Simply put, a bridge makes a connection *possible*, while a local bridge makes a connection *shorter* (by reducing the geodesic distance). Because our analysis is limited to networks with a single component, we only consider local bridges, and for brevity, we will refer to these simply as "bridges." A bridge is generally assumed to consist of a single tie, which is sufficient for simple contact between neighborhoods. However, if a connection requires multiple contacts, then a bridge must consist of multiple ties. Hence, we can measure a bridge not only by its length (the range that is spanned by the bridge) but also its width (the number of ties it contains). The range of a bridge is the geodesic between the focal nodes of the neighborhoods connected by the bridge if all of the bridge ties were to be removed.[7]

The importance of bridge width has been overlooked in previous research because it is not relevant for simple contagions. However, propagation of many collective behaviors depends on bridges that are wide as well as long. The structural weakness of long ties is that they form bridges that are too narrow for complex contagions to pass.

EFFECTS OF LONG TIES ON A RING LATTICE

The classic formalization of the small world principle 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 ties (within a cluster), making them into bridges between clusters that reduce the mean distance between arbitrarily chosen nodes in the network.

Our central purpose is to test whether this principle generalizes from the spread of information and disease to the spread of collective behavior.

[7] By definition, wide bridges are always composed of short ties. Conversely, long ties always form the narrowest possible bridge.

In order to replicate earlier studies as closely as possible, we begin with the original Watts and Strogatz (1998) small world model. They used a ring lattice to demonstrate the small world effect for a simple contagion. A ring lattice is a one-dimensional spatial network that allows the simplest analytical model of the effects of introducing long ties to an ordered graph.

Watts and Strogatz demonstrated the small world effect by holding the density of the ring constant as local ties were replaced with a tie to a randomly selected node. Long ties can also be introduced by *adding* random ties to the network (Newman and Watts 1999), but this increases both density and the fraction of long ties, which confounds the effects of randomization with the effects of densification. The effects of network density on the rate and frequency of complex contagions is an interesting and important question, but it is not the question that frames the present study. We therefore focus on randomization while holding density constant, using a rewiring method similar to that used by Watts and Strogatz.[8]

Our only departure from Watts and Strogatz is that we raised activation thresholds above the lower limit for propagation through social contact assumed in previous studies. Thresholds can be expressed in two ways—as the *number* (Granovetter 1978) or the *fraction* (Watts 2002) of neighbors that need to be activated. The conceptual distinction reflects an underlying (and often hidden) assumption about the influence of nonadopters. Fractional thresholds model contagions in which both adopters and nonadopters exert influence, but in opposite directions. For example, the willingness to refrain from littering in one's neighborhood may depend not only on the number of others willing to refrain but also on their numbers relative to those who add to the litter. If nonadopters exert countervailing influence, as neighborhood size increases, a greater number of activated neighbors are required to trigger adoption. In contrast, numeric thresholds model contagions in which nonadopters are irrelevant. Hence, an increase in neighborhood size has no effect on the required number of activated neighbors. For example, disease has a threshold of one. No matter how large the neighborhood, infection requires contact with only a single carrier. Uninfected neighbors do not increase resistance to the contagion. Similarly, the credibility of an urban legend may depend only on the

[8] Newman and Watts (1999) show that adding ties to a regular lattice is more robust than the rewiring method (Watts and Strogatz 1998; Watts 1999) because it eliminates the possibility of multiple components forming at high levels of randomization. For this study, we use the rewiring technique proposed by Maslov and Sneppen (2002), which also keeps the network connected, while allowing each node to keep a constant degree at all levels of randomization. Thus, we assume throughout that networks are connected in a single component and focus on the effects of randomization within a component of constant size and density.

number of others from whom it has been heard, regardless of the number who have never mentioned it.

If ties are randomly rewired, holding degree constant, the effect on propagation is the same whether thresholds are expressed as the number or the fraction of activated neighbors.[9] However, we also manipulate degree exogenously, holding it constant at different levels as ties are randomized. We therefore represent the threshold τ as a fraction $\tau = a/z$, where a is the number of activated nodes and z is the number of neighbors. This notation allows us to distinguish, for example, between $\tau = 1/8$ and $\tau = 6/48$. Both thresholds require an identical proportion of activated neighbors, but the former is a simple contagion, and the latter is complex. One of the main findings of our study is that there is a qualitative difference between $a = 1$ and $a > 1$, even when the proportions are identical.

We also followed previous studies (Watts and Strogatz 1998; Watts 1999; Newman 2000) in assuming that

1. The network is sparse.
2. Thresholds are deterministic (the probability of activation goes from zero to one as the threshold is crossed).
3. Every tie has equal weight.
4. Every node has equal influence.
5. Every node has an identical threshold τ.
6. Every node has about equal degree.

These simplifying assumptions are standard in much of the diffusion literature, including the research on small worlds, because they are necessary to identify the structural effects of long ties without the confounding effects of heterogeneity and stochasticity. Nevertheless, homogeneity (of thresholds, influence, and degree) violates our empirical intuition. We therefore began with the simplest possible extension to the basic model—in which everyone has identical influence, thresholds, and neighborhood size—and analyzed the effects of random rewiring as we increased thresholds above $1/z$. Following our analysis of the simple ring structure, we use a two-dimensional lattice to introduce a sequence of complications, including much larger neighborhoods and heterogeneity of degree, thresholds, and influence. We also relaxed the assumption that thresholds are

[9] If random ties are added to a network, the effect on propagation depends decisively on whether thresholds are expressed as the number or the fraction of activated neighbors. Adding random ties increases exposure to both activated and unactivated neighbors. If unactivated neighbors have no countervailing influence, then adding ties can only promote propagation, whether contagions are simple or complex. However, if unactivated neighbors increase resistance to contagion, then adding ties promotes the propagation of simple contagions, but there will be a much stronger inhibiting effect on complex contagions compared to the effect of random rewiring (holding degree constant).

strictly deterministic. These complications require the use of computational methods. However, the highly simplified case we consider first allows an analytical investigation of the effects of network topology on the propagation of simple and complex contagions.

Figure 1 illustrates the width of the bridge between two neighborhoods (I and L) on a ring lattice with $z=4$. Neighborhood I is the ego network containing focal node i and all of i's neighbors [g,k] (black and gray/black nodes). Neighborhood L contains [j,n] (gray and gray/black nodes), where [l,n] $\notin I$. These two neighborhoods have two common members (gray/black nodes). C_{IL} is the set of all common members of both I and L, hence $C_{IL} = [j,k]$. The disjoint set D_{IL} contains the remaining members of L that are not in I, or $D_{IL} = [l,n]$. A bridge from I to L is then the set of ties between C_{IL} and D_{IL}, where the width of the bridge, W_{IL}, is the size of this set. In figure 1, the bridge consists of the three ties jl, kl, and km (shown as bold lines), making $W_{IL} = 3$.

The overlap between the neighborhoods is the number of nodes in C_{IL} (denoted $|C_{IL}|$). In figure 1, the neighborhoods I and L have the maximum possible overlap. Neighborhood M containing [k,o] is one step farther from I, so only node k is shared between them ($|C_{IM}| = 1$), and there is only a single tie (km) between I and M, making the width of the bridge $W_{IM} = 1$.

More generally, on a ring lattice of degree z, $0 \leq | C | \leq z/2$. The widest bridge on the ring is limited by the maximum overlap $|C_{max}| = z/2$. There will be $z/2$ ties from I to the member of L closest to I, $z/2 - 1$ ties to the next closest member of L, and so on, giving:

$$W_{max} = z/2 + (z/2 - 1) + (z/2 - 2) + \ldots + 1, \quad (1)$$

$$W_{max} = z(z + 2)/8. \quad (2)$$

The bridge from I to L is therefore the maximum possible width for $z=4$, giving $W_{max} = 3$.

The width of the bridge between neighborhoods determines the upper bound on the threshold at which a contagion can pass. In figure 1, $W_{IL} = 3$, which imposes an upper bound of $a = 2$. So long as $a \leq 2$, the two ties from j and k will be sufficient to activate l, and l and k can then activate m, and so on.

Conversely, thresholds determine the *critical width* (W_c) of bridges, defined as the minimum number of nonredundant ties required for a contagion to propagate to an unactivated neighborhood.[10] For simple

[10] Ties are nonredundant so long as there are no more than a bridge ties to a single member of D_{IL}. Suppose $a=2$. If there were three bridge ties to any node in D_{IL}, then one of these ties would be redundant.

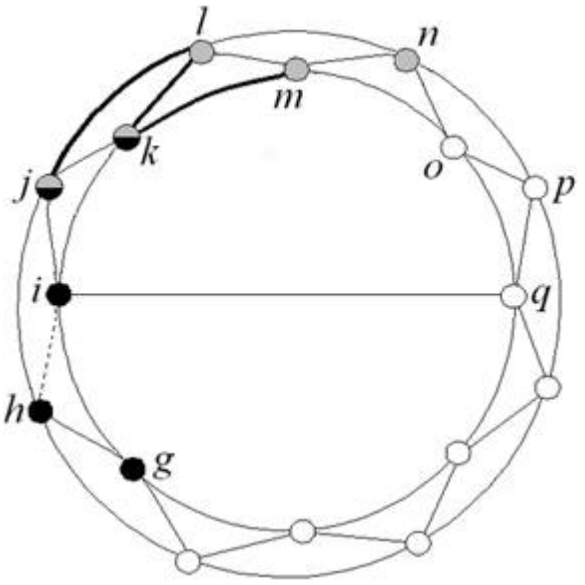

FIG. 1.—A ring lattice with $z = 4$ and one long tie. The figure illustrates the width of the bridge between the neighborhoods of i (black and gray/black nodes) and l (gray and gray/black nodes), showing the two common members (gray/black nodes). The bridge between these two neighborhoods consists of the three ties jl, kl, and km (shown as bold lines). The long tie from i to q provides a shortcut for a simple contagion but not for one that is complex.

contagions, $W_c = 1$, regardless of network topology. On a ring lattice, for minimally complex contagions ($a = 2$), $W_c = 3$. For example, in figure 1, the three ties between C_{IL} and D_{IL} (two to activate l and one to activate m once l is active) allow the contagion to spread from I to L.

More generally,

$$Wc = a + (a - 1) + (a - 2) + \ldots 1, \tag{3}$$

$$W_c = a(a + 1) / 2, \tag{4}$$

giving $W_c = 3$ as the critical width for a minimally complex contagion ($a = 2$). A contagion can propagate around the ring so long as $W_c \leq W_{max}$.

The critical width also determines the minimum number of ties that need to be rewired to create a shortcut across the ring. Figure 1 shows how a single random tie is sufficient to increase the rate of propagation of a simple contagion. Suppose we were to randomly select tie ih to be randomly replaced with tie iq. For a simple contagion ($\tau = 1/z$), the rewiring of the ih tie creates a shortcut across the ring that reduces the time required for a cascade to reach all the nodes. The deleted tie from

h to i (indicated by the broken line) does not hinder the spread of a simple contagion around the ring since the critical width for $a = 1$ is $W_c = 1$, and $W_{max} = 3$ provides sufficient redundancy to support local propagation even with the ih tie removed.

However, the need for bridges that are wider than a single tie implies a qualitative change in propagation dynamics as a increases above one. Figure 2 shows how an increment in the threshold from $\tau = 1/z$ to $\tau = 2/z$ triples the critical width of the bridge required to create a shortcut, from one tie to three. Node j is the focal node of the seed neighborhood J in which j and all four of j's neighbors are activated (indicated by solid black and gray/black nodes). Node s is the focal node of an unactivated neighborhood S (shown in gray and gray/black). For a minimally complex contagion ($\tau = 2/z$), $W_c = 3$, which means that three local ties must be rewired to create a bridge across the ring (indicated by the three bold lines). The two ties from i and k are sufficient to activate s, and the third tie from i to q is sufficient to activate q, given the tie from s to q.

Even for this minimally complex contagion on this very small ring (with only 16 nodes), the probability that three random ties will form a bridge is close to zero. We can expect to need many more random ties before the first bridge is formed across the ring, and that number increases exponentially as N increases. That is because the number of configurations in which all three random ties are between the same two neighborhoods is a very small fraction of the total number of possible configurations.[11] Further, as a increases, there is an exponential increase in the number of ties required to form a bridge, further reducing the likelihood of bridge formation.

An obvious solution to the need for wider bridges is simply to rewire more ties, thereby ensuring that shortcuts across the network will eventually form. However, the problem with extensive rewiring is the potential to erode the existing bridges that allow the contagion to spread locally. Figure 2 shows how this happens. The deleted tie from h to i (indicated by the broken line) would not hinder the spread of a simple contagion. However, even for a minimally complex contagion, the three deleted ties (broken lines) reduce the width of the bridges on either side of i to less than $W_c = 3$, preventing the contagion from spreading locally. The contagion can still spread out in both directions from s, but the JS bridge will not increase the rate of propagation. Moreover, the probability that three random ties will form a bridge (like the one illustrated in fig. 2) is

[11] As the number of adopters increases, random ties can create wide bridges to an unactivated neighborhood from multiple activated neighborhoods, thereby increasing the probability of success. Hence, the farther the contagion spreads, the greater the benefit from random rewiring.

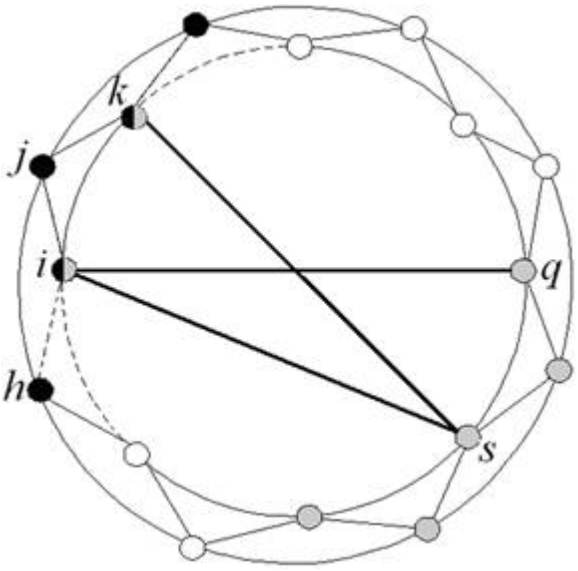

FIG. 2.—A ring lattice with $z = 4$ and three ties. The figure shows the width of the bridge between neighborhood J (black and gray/black nodes, with focal node j) and neighborhood S (gray and gray/black nodes, with focal node s), showing the two common members (gray/black nodes). An increment in the threshold from $\tau = 1/z$ to $\tau = 2/z$ triples the width of the bridge required to create a shortcut (bold lines) between J and S, from one tie to three. The two ties is and ks are sufficient to activate s, and the third tie from i to q is sufficient to activate q, given the tie from s to q.

close to zero, while the probability that three deleted ties will break the ring and block the contagion is close to one.

Simply put, the effect of rewiring depends on whether random ties are more likely to form bridges across the ring than to break bridges along the ring. This in turn depends on the magnitude of W_{max} relative to W_c. If $W_c = W_{max}$, there are no redundant ties in the bridge, and every tie that is removed creates a break along the ring. If $W_c < W_{max}$, some bridge ties may be redundant, and if they were to be rewired to form a new bridge, the rate of propagation would increase.

W_{max} increases exponentially with z while W_c increases exponentially with a. Holding a constant, an increase in z means a smaller fraction of neighbors need to be activated in order for a node to become active. It also means $W_c \ll W_{max}$, hence greater redundancy of bridge ties. As redundancy increases, the network becomes more efficient if some of the redundant ties are randomly rewired to create new bridges.

More generally, the redundancy R refers to the proportion of ties in a bridge that can be rewired without breaking the ring, or

$$R = (W_{max} - W_c) / W_{max}, \quad (5)$$

$$R = 1 - (4a[a + 1] / z[z + 2]). \quad (6)$$

We can see from equation [6] that if $a > z/2$, then $R < 0$, which means that bridges will be too narrow for propagation. Thus, contagions cannot propagate on a ring lattice of any degree if $\tau > .5$ (Morris 2000). If $\tau = .5$, $R = 0$. This means that contagions can now pass, but there is no redundancy (as in fig. 2). The first tie that is randomized will break the ring.[12]

As R increases, more ties can be rewired without creating breaks along the ring, allowing complex contagions to benefit from randomization, just as do simple contagions. However, there is an important difference. For simple contagions, a connected network can never be too randomized. That is not true for complex contagions. Eventually, randomization will reach a critical upper limit (given by R) above which even minimally complex contagions can no longer propagate. For example, on a ring lattice with $z = 10$, $W_{max} = 15$ and $R = .75$. For a minimally complex contagion ($a = 2$ and $W_c = 3$), the high level of redundancy indicates that limited randomization could allow faster propagation than on the unperturbed ring. The unperturbed lattice consists of a chain of bridges that are linked to one another around the ring. As long as randomization does not create a break along this chain, rewiring redundant ties to create a shortcut will allow the contagion to jump across the network and fan out from multiple locations.

However, if randomization rewires more than R of the ties in an existing bridge, the chain will be cut. Although this rewiring may also create new bridges, the advantage of these shortcuts depends on the existence of other bridges to which the shortcut is linked. Bridges that are randomly created as the ring is perturbed are only useful to the extent that they are linked to other bridges. Otherwise the random rewiring creates a bridge to nowhere. As the links of the chain become increasingly disconnected, the probability increases that a random bridge will lead into a cul de sac.

To review, the analysis of the ring lattice reveals two qualitative differences between simple and complex contagions:

1. While a single random tie is sufficient to promote the spread of simple contagions, complex contagions require more rewiring in order to benefit from randomization. The number of ties that need

[12] Note that it is also the case that $R = 0$ if $a = 1$ and $z = 2$, giving $W_c = W_{max} = 1$. The rewired tie now creates a break along the ring but also creates a bridge across the ring, allowing the contagion to fan out from three locations instead of just two (prior to rewiring). However, if $a \geq 1$ and $R = 0$, the first tie that is randomly rewired will break the ring but cannot create a shortcut.

to be randomly rewired increases exponentially with the number required to form a bridge (W_c), and the number of ties needed to form a bridge in turn increases exponentially with the required number of activated neighbors (a).

2. As the ring becomes increasingly randomized, the width of the bridges that make up the lattice structure may be eroded below the critical width required for the contagion to spread. Simple contagions can propagate on a connected network even if every tie is random, and the rate of propagation increases monotonically with the proportion of random ties. In contrast, there is a critical upper limit of randomization above which complex contagions cannot propagate. As thresholds increase, this critical value decreases.

LONG TIES ON HIGHER DIMENSIONAL NETWORKS

These conclusions for a one-dimensional lattice do not necessarily generalize to higher dimensional structures, which provide detours around local ties that have been deleted. However, higher dimensional structures lack the analytical simplicity of the ring lattice. For networks with more complicated geometries, we used computational models to confirm and extend the analysis of the ring lattice.[13]

We began by replicating the small worlds experiments on the spread of simple contagions, using a two-dimensional lattice with Moore neighborhoods instead of the ring lattice used in earlier studies (Watts and Strogatz 1998; Watts 1999; Newman and Watts 1999).[14] We then repeated the experiment with only one change—we increased activation thresholds above the theoretical minimum ($1/z$) for propagation through social contact.

We used two values of degree ($z = 8$ and $z = 48$) so that we could independently manipulate a and z. Network density was held constant as degree increased by increasing N from 40,000 with $z = 8$ to 240,000 with $z = 48$. Thresholds ranged from $a = 1$ to the critical upper limit for propagation on a Moore lattice,[15] which is $a_c = 3$ for $z = 8$ and $a_c =$

[13] All of the reported results for the Moore lattice were replicated on the ring lattice as well, and any differences are noted.

[14] Moore neighborhoods include nine nodes: a focal node and its 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 $z = 4r(r+1)$. Qualitatively similar results are found for $r = 1$ and $r \geq 1$.

[15] The upper limit for propagation on a Moore lattice is $a_c = 2r^2+1$, where r is the radius of the neighborhood.

19 for $z = 48$. In each condition, the model was seeded with a sufficient number of activated nodes to allow a contagion to spread on a lattice network. With simple contagions, a single node was randomly chosen as the seed. With higher thresholds, a focal node was randomly selected and then that node plus its neighbors were activated. At each time step, an unactivated node was randomly selected (without replacement) and its state updated based on its threshold relative to the proportion of its activated neighbors.[16]

Figures 3 and 4 report results for Moore lattices with $z = 8$ (fig. 3) and $z = 48$ (fig. 4), a range that is sufficient to illustrate the effects of neighborhood size. The ordinate shows the rate of propagation as the time steps t required for the contagion to saturate the network (99% of nodes). Time steps were recorded exclusively for successful cascades. The abscissa in figures 3 and 4 represents the proportion p of ties that are rewired, where $p = 0$ corresponds to a regular lattice (all network ties are spatially constrained) and $p = 1$ corresponds to a random network (individuals are tied with equal probability to everyone in the network). Between 0 and 1, there is a region of p in which there is high local clustering with low mean geodesic, corresponding to a small world network.

The results for $\tau = 1/z$ (at the bottom of figs. 3 and 4) confirm the results from previous studies of propagation on small world networks. As ties are rewired, propagation rates approach those of random networks while the network still has abundant local structure ($p < .1$)—the network remains highly clustered and yet is now also highly connected (Watts and Strogatz 1998). Watts and Strogatz (1998) showed that only a modest fraction of random ties are needed to allow propagation rates to approach those observed on a random graph. This is confirmed by the results for $\tau = 1/z$.

However, this small world effect does not generalize to complex contagions, even when contagions are minimally complex ($\tau = 2/8$ in fig. 3), and two activated neighbors are a very small fraction of the neighborhood ($\tau = 2/48$ in fig. 4). Instead, the results in figures 3 and 4 mirror the analytical results for the ring lattice by showing qualitative differences between simple and complex contagions. The analysis of the ring revealed a critical upper limit of randomization, corresponding to R, above which propagation will be precluded, and also a lower limit of randomization, below which there are too few long ties to create a shortcut. Similarly, the results for Moore neighborhoods show that random ties do not help complex contagions at very low and very high levels of randomization,

[16] Asynchronous updating with random order and without replacement eliminates potential order effects and guarantees that every node is updated within a round of decision making, which we define as N time steps.

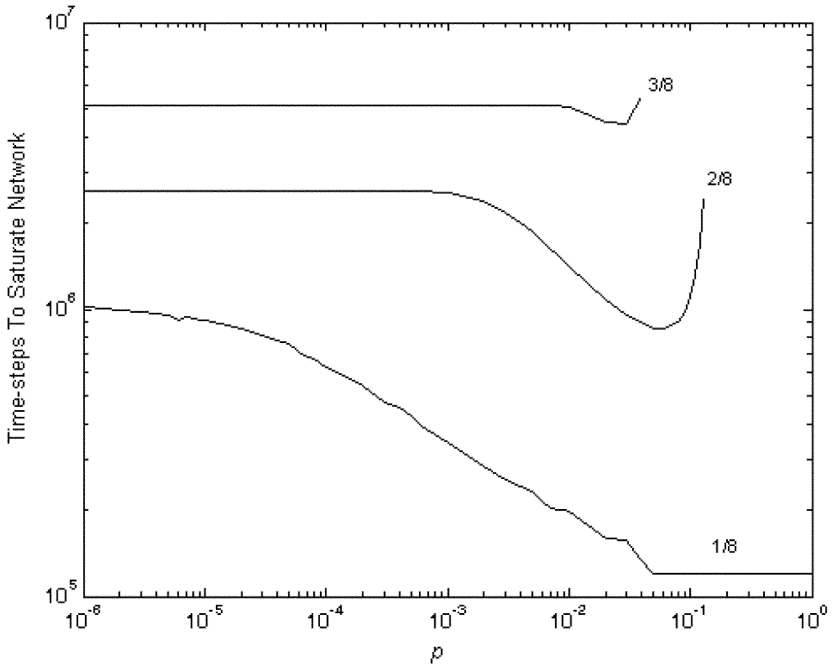

FIG. 3.—Propagation times for simple and complex contagions ($z = 8$; $N = 40{,}000$, averaged over 100 realizations). Lines show the number of time steps required for cascades to saturate the network (reach 99% of nodes) as p increases from 10^{-6} ($p \approx 0$ on a log scale) to 1 along the abscissa. For simple contagions (bottom line, $\tau = 1/8$), small increases in p facilitate propagation, and further increases monotonically reduce propagation time. For minimally complex contagions (middle line, $\tau = 2/8$), small increases in p do not have any effect on propagation, while greater increases have a nonmonotonic effect, first decreasing propagation time, then increasing it. For complex contagions with higher thresholds (top line, $\tau = 3/8$), the "small world window" narrows. The lines end at the critical point above which randomization precludes propagation entirely.

between which there is a "small world window." Inside this window, propagation can benefit from randomization if thresholds are relatively low. As thresholds increase, bridges need to be wider, which means more ties must be rewired to form shortcuts randomly, and existing bridges become more vulnerable to perturbation. In combination, these effects reduce—and ultimately eliminate—the small world window.

These differences between simple and complex contagions are evident in figures 3 and 4, which display three principal results:

1. Complex contagions fail to benefit from low levels of randomization, as shown by the initial failure of propagation rates to improve as p increases above zero.
2. Increasing p has a nonmonotonic effect on complex contagions, exhib-

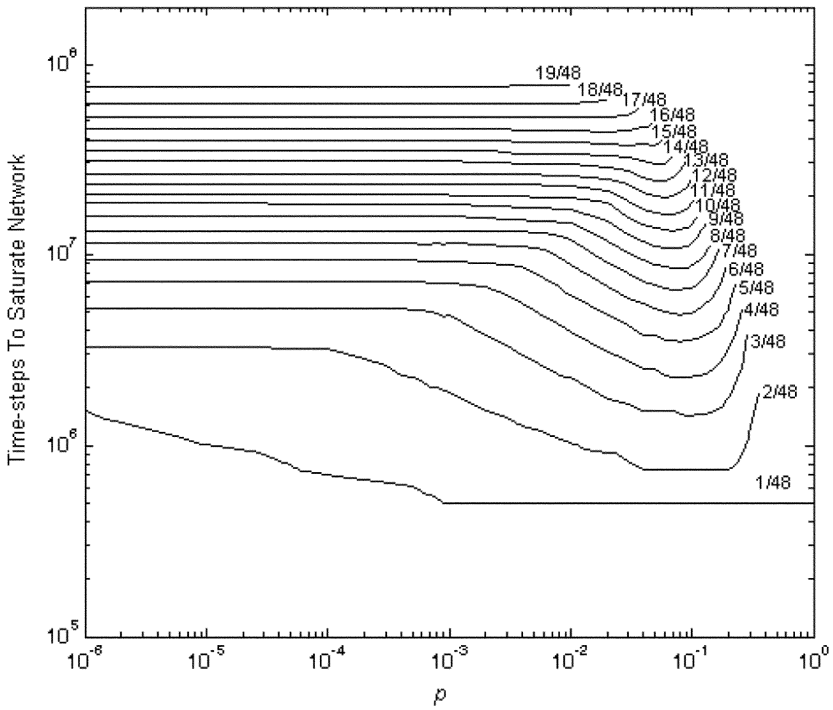

FIG. 4.—Propagation times for simple and complex contagions ($z = 48$; $N = 240{,}000$, averaged over 100 realizations). Lines show the number of time steps required for cascades to saturate the network (reach 99% of nodes) as p increases from 10^{-6} ($p \approx 0$ on a log scale) to 1 along the abscissa. For complex contagions, increasing thresholds narrows the "small world window" of p values for which random ties facilitate propagation. For thresholds above $\tau = 16/48$, the window closes, leaving only the inhibiting effect of random ties.

iting a U-shaped effect, in which randomization starts to help—but ultimately impedes—propagation.

3. As p exceeds a critical upper limit, complex contagions entirely fail to propagate.

Figure 5 provides a more detailed view of cascade failure for the results shown in figure 3.[17] In figure 5, the abscissa represents p, and the ordinate indicates the frequency of successful cascades over 100 realizations.[18] As shown in figure 5, there is a highly nonlinear effect of network pertur-

[17] The qualitative behavior shown in fig. 5 also holds for all results shown in fig. 4.

[18] Across all realizations at all values of p, cascades either reached 99% or more of the population or less than 1%—there were no partial successes. Given this extreme bimodal distribution in the proportion of nodes that are reached, the proportion of realizations in which cascades succeeded conveys more information than the average proportion of nodes that were reached.

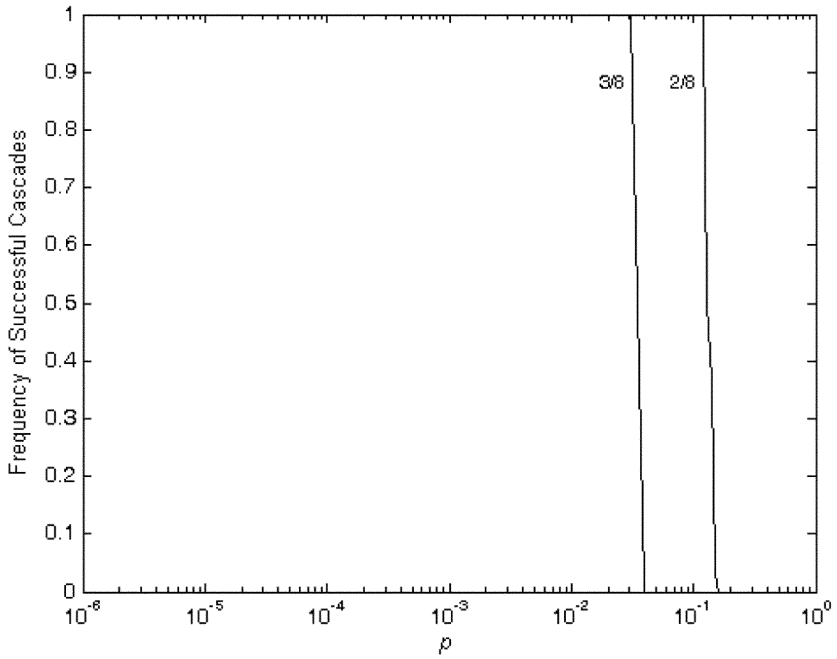

FIG. 5.—Critical transition in cascade frequency ($z=8$; $N=40{,}000$, averaged over 100 realizations). Lines show the average frequency of cascades as the fraction of random ties increases from $p\approx 0$ to $p=1$ (corresponding to the propagation times shown in fig. 3). The sharp drop in frequency as p reaches a critical value indicates a first-order phase transition. Below this value, cascades spread to the entire population, while above it, cascades reach less than 1% of the nodes. For minimally complex contagions ($\tau=2/z$), this critical value occurs at $p\approx .1$. For slightly higher thresholds ($\tau=3/z$), the transition occurs at $p\approx .03$.

bation on the frequency of successful cascades as p increases from 0 to 1. We observe not a steady decline in cascade frequency, but a dramatic shift from near-complete success on each trial, to near-zero success.

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 the macrolevel properties of a system. 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 successfully. However, once the fraction of random ties exceeds this critical point, the network can no longer

support the spread of complex contagions. 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 social diffusion.

To sum up, these experiments with Moore neighborhoods of varying degree confirm the analysis based on the ring lattice. A few long ties facilitate simple contagions, which is why small world networks are a highly effective topology for the spread of information and disease. However, as thresholds increase, complex contagions depend increasingly on network topologies with wide bridges—such as might be observed in residential networks and social networks with overlapping clusters. The higher the thresholds, the lower the likelihood that random ties will form the wide bridges that provide social reinforcement. Ultimately, as more random ties are added, a phase transition transforms the network abruptly from one that can sustain complex contagions to one that cannot.

We also found that randomization can promote the spread of complex contagions so long as the randomization is not too great and the thresholds are not too large. This U-shaped effect of randomization is an important extension of the small world principle. Watts and Strogatz (1998) discovered that simple contagions could spread as fast on a highly clustered small world network as on a more randomized topology. This was important because social networks tend to be highly clustered and rarely (if ever) random. Figures 3 and 4 reveal that this "small world effect" becomes more pronounced as thresholds increase slightly above the level of simple contagions. That is, complex contagions with relatively low thresholds can actually spread *faster* on a highly clustered small world network than on either a network that is more random or one that is more clustered. However, as thresholds get higher still, the small world effect disappears entirely. In short, the essential difference between simple and complex contagions can be distilled as follows. For simple contagions, too much clustering means too few long ties, which slows down cascades. For complex contagions, too little clustering means too few wide bridges, which not only slows down cascades but can prevent them entirely.

TESTS FOR ROBUSTNESS OF THE SIMPLIFYING ASSUMPTIONS

So far we have been careful to change only a single assumption of the theoretical model of small worlds while holding all else constant. We merely raised activation thresholds to levels that empirical research suggests are likely to characterize many collective behaviors. The results clearly demonstrate the danger of generalizing from the spread of disease and information to the spread of collective behaviors.

Threshold Heterogeneity

We now take the analysis a step further, to test the robustness of these results as we relax some of the simplifying assumptions in previous research, namely the homogeneity of degree, social influence, and activation thresholds. We first consider the effects of threshold heterogeneity in a model that is otherwise identical to that in figures 3, 4, and 5 but with a Gaussian distribution of thresholds. The primary finding is that cascade times and frequencies behave much as they do for fixed thresholds. Initial perturbations to the network have no effect on cascade dynamics. As p increases, propagation times exhibit the characteristic U-shaped pattern evident in figures 3 and 4, and cascade frequency exhibits the same first-order phase transition that was observed for fixed thresholds. The phase transition occurs for increasingly lower values of p as $\bar{\tau}$ increases, equivalent to what we observe with fixed thresholds. Finally, as with fixed thresholds, as thresholds increase, the small world window disappears, and there is a monotonic increase in propagation time as p increases.[19]

As an additional test, we investigated the effects of heterogeneity within nodes as well as between nodes. Within-node heterogeneity relaxes the assumption that thresholds are stationary by allowing thresholds to change over time, which we implemented by randomly reassigning thresholds after each round of decision making (i.e., after all nodes had been given a chance to become activated). We assigned thresholds using the same Gaussian distribution as with stationary threshold heterogeneity. The results are similar to what we observed with stationary thresholds, confirming the robustness of the distinction between simple and complex contagions.

We also tested stochastic thresholds in which nodes are activated with a probability that increases with the number of activated nodes in the neighborhood. Using the cumulative logistic function (Macy 1990), nodes have a 50% chance of activation when the proportion τ of the neighborhood is activated. Below τ, the probability approaches zero as a convex function of the number of active neighbors, and above τ the probability approaches one as a concave function.[20] The results for stochastic thresholds were similar to those for deterministic thresholds—random rewiring

[19] These results are consistent with analytic predictions derived from the generating function method developed in Watts (2002), in which the effects of threshold heterogeneity are reduced to the fraction of nodes susceptible to activation by a single neighbor. With $z = 48$, surprisingly few susceptible nodes are needed to allow minimally complex contagions to spread as if they were simple. However, as mean thresholds increase, contagions no longer benefit from randomization.

[20] In infinite time, stochastic thresholds have a nonzero probability of activating the entire population. However, these results are for finite time scales comparable to those used for deterministic thresholds.

slowed complex contagions and ultimately prevented them from spreading. This surprising result is due to the fact that as thresholds increase, the probability that an activated node will stochastically "turn off" also increases, making the diffusion of complex contagions more difficult as random rewiring reduces pathways of local reinforcement.

Heterogeneity of Influence

We also tested the effects of heterogeneity of influence. Influence is the complement of a threshold, in that it determines the ability of activated nodes to propagate the contagion instead of the susceptibility of their neighbors to becoming activated. We implemented heterogeneity of influence in two ways, as ties to low- and high-status neighbors and as ties to friends and acquaintances.

Status differences were created by assigning a few random nodes the ability to activate their neighbors without the need for social affirmation or reinforcement from additional sources. This enhanced influence might reflect higher social prestige, power, wealth, persuasiveness, and so on. For convenience, we will refer to these as "high-status nodes."

Of course, if there are enough high-status nodes to activate the remainder of the population in one step, the problem reduces to that of a simple contagion. The interesting case is one in which a few high-status nodes must trigger a cascade in order to activate the population. As a conservative test, we randomly assigned N/z of the nodes to be high status (e.g., 5,000 high-status nodes in a population of 40,000). On average, this means that every neighborhood in the network can now be expected to have one high-status member. High-status nodes were given sufficient influence, ι, to activate all of their neighbors (i.e., $\iota \geq a$ for $\tau = a/z$). In order not to conflate the effects of influence heterogeneity with an increase in mean influence (equivalent to a reduction in the average threshold), we held mean influence constant by reducing the status of all other nodes sufficiently to compensate for doubling the influence of a few "opinion leaders" (Katz and Lazarsfeld 1954).

Results show that introducing a small fraction of high-status nodes does not mitigate the need for wide bridges. Under the assumption that the distribution of status is highly unequal, there is no improvement in the propagation of complex contagions as p increases.

To see why, suppose the high-status nodes are sufficiently influential to activate all their Moore neighbors on a network with $z = 48$. The problem is what happens next. Assuming $\tau = 2/z$ and influence homogeneity, an activated node would need only one other activated node to activate a common neighbor. However, with influence heterogeneity (and holding mean influence constant), two activated low-status nodes no longer have

combined influence sufficient to activate a common neighbor with threshold $2/z$. Now three low-status nodes must be activated in order to extend the contagion beyond the reach of their high-status activated neighbor. This increases the width of the bridge needed to propagate the contagion. Thus, status inequality can make the propagation of complex contagions even more vulnerable to network perturbations that remove ties from existing bridges. However, as the number of high-status nodes increases, the propagation of complex contagions can begin to resemble that for simple contagions.

Strong and Weak Ties

Up to this point we have assumed that all ties have equal strength, regardless of range. That is a reasonable assumption for the acquisition of information and disease, which does not depend on the relationship with the source. However, many social contagions not only have higher thresholds than disease and information, but also, the threshold may depend on whether the source is a close friend or an acquaintance. Thus, Granovetter (1983, p. 202) distinguishes between relationally weak ties connecting "acquaintances" located in "distant parts of the social system" and "close friends most of whom are in touch with one another." Following Granovetter (1983, p. 201), we used a binary coding in which closed triads are assumed to connect "close friends," and open triads are "acquaintances" who "are less likely to be socially involved with one another than are our close friends (*strong ties*)." We assigned regular unperturbed ties a weight of 1 and random ties a weight of .5. This 2:1 ratio is convenient in that it parallels the distinction between simple and minimally complex contagions. It means that a single close friend is now sufficient to activate a neighbor with a threshold of $1/z$, but it will take two acquaintances.

As expected, the effects of randomization for simple contagions now resemble what we previously observed for minimally complex contagions. In addition, when we repeated the experiment with heterogeneous thresholds and $z = 48$ (see fig. 4), we observed inhibitory effects of long ties even for populations in which $\bar{\tau} = 2/z$. More generally, the weaker the ties to acquaintances compared to friends, the wider must be the bridges connecting otherwise distant neighborhoods.

Heterogeneity of Degree

Barabasi (2002) has shown that high degree nodes (or "hubs") dramatically improve the diffusion of simple contagions (see also Barabasi, Albert, and Jeong 2000). This suggests the need to test the robustness of the results in figures 3, 4, and 5 by replacing the regular lattice with a highly clustered

scale-free network (Klemm and Eguiluz 2002), which more closely resembles empirically observed small world networks (Barabasi 2002; Barabasi et al. 2000; Newman et al. 2006). We tested our findings using a scale-free degree distribution with $N = 40{,}000$ and $\gamma = 2.3$, in which many nodes have relatively low degree ($z < 5$), and only a few have very high degree ($z > 100$).[21] As noted above, the rewiring procedure that we used reduces clustering through randomization without altering the degree of any node. Thus, we are able to isolate the effects of randomization while preserving the scale-free degree distribution.

All nodes required the same number of activated neighbors (a), but since degree varied, so too did the necessary proportion of activated neighbors. The very low degree in most nodes precluded the spread of contagions with $a > 2$, even with $p = 0$. However, for thresholds of $2/z$, the results were similar to those for the regular lattice in figure 5, except that the drop-off in cascade frequency is noticeably more gradual. This is because the activation of large neighborhoods ($z > 100$) occasionally allows cascades to spread through part of the network. However, even when an activated neighborhood is very large, the bridges between peripheral neighborhoods must remain intact in order for complex contagions to spread. The low degree in most neighborhoods means that there are very few if any redundant ties between neighborhoods, making bridges especially vulnerable to randomization. Thus, a scale-free network can be even more sensitive to perturbation than the regular lattices used in figures 3, 4, and 5. For example, with $\tau = 2/z$, minimally complex contagions were almost entirely inhibited above $p = .001$ (compared to $p \approx .1$ for the regular lattice in fig. 5).

More generally, degree heterogeneity can exacerbate the effects of randomization by increasing the exposure of hubs to large numbers of unactivated nodes. As degree becomes more skewed, the odds become much higher that a peripheral node will be randomly chosen as the seed. This might seem to make it more likely that a hub will become activated because of its greater access to the network. However, it is very difficult for a single peripheral node to activate a hub when all the other peripherals are exerting countervailing influence. For example, a manager with larger numbers of peer contacts may be more likely to hear about an innovation than a manager with less social capital, but the well-connected manager will also be exposed to the inertial effects of countervailing pressures by nonadopters. Hence, the well-connected manager may be faster to hear about the innovation but slower to adopt it, compared to a socially isolated

[21] Scale-free distributions are specified with the slope parameter γ, where $P(k) = k^{-\gamma}$. $\gamma = 2.3$ is commonly used for social networks (see Klemm and Eguiluz 2002).

manager who hears about the innovation from one of a very small number of peers.

Moreover, even if a hub is already activated, the hub still cannot activate peripherals who require social affirmation or reinforcement from others. Complex contagions can spread on hub-and-spoke structures only in the special case that hubs can compel activation of the peripheral nodes without reinforcement. Otherwise, the diffusion of complex contagions requires wide bridges, even on networks with skewed degree distributions (Centola et al. 2007). These limited explorations of heterogeneity suggest that the differences between simple and complex contagions can be even more pronounced than the differences observed in figures 3, 4, and 5.

DISCUSSION

Granovetter (1973, p. 1366) provided a succinct statement of the strength of weak ties: "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." Our results show that it can be very dangerous to generalize from the spread of information and disease to whatever is to be diffused. Network topologies that make it easy for everyone to know about something do not necessarily make it likely that people will change their behavior. In this section, we point to empirical studies that provide support for this conclusion. We also consider the implications of our results for future empirical research and for public policy.

We know of no empirical studies that have directly tested the need for wide bridges in the spread of complex contagions. The closest is a recent study (Backstrom et al. 2006) on the influence of friends on the probability of joining a LiveJournal blogging community. By comparing the influence of friends scattered across the network with friends concentrated in a single network neighborhood, the Backstrom et al. study provides an indirect test of the need for wide bridges in the propagation of complex contagions. Having friends in a community who are also friends with one another increases the probability of joining, compared to friends in a community who do not know one another. This suggests that the growth of a community depends less on weak ties that span longer social distances and more on wide bridges, such that "the individual will be supported by a richer local social structure" (Backstrom et al. 2006, p. 5).

Our theoretical results also provide new insight into the widely observed tendency for social movements to spread over spatial networks. Beginning with McAdam's (1988) seminal study of Freedom Summer, a consistent finding in social movement research is that participation spreads most effectively in populations that are spatially clustered, such as ethnic en-

claves. Hedstrom's (1994) study of the early labor movement in Sweden has similar findings, which show that participation spreads locally, from one residential neighborhood to another. In China, the dormitory housing arrangements structured social ties in a way that allowed for easy diffusion of student dissent (Zhao 1998). Similarly, the close quarters of inner-city settlements in the Paris Commune promoted the emergence of violent revolts (Gould 1996). Spatial patterns of adoption have also been shown to describe the diffusion of birth-control technology in Korean villages (Rogers and Kincaid 1981), and Whyte (1954) argues that the diffusion of product adoption in Philadelphia followed spatial residential patterns.

Empirical studies have pointed to the relational property of spatial networks that makes them conducive to social, political, and cultural diffusion. The relational property is *physical proximity*, which is needed for the spread of communicable diseases that require physical or respiratory contact, fashions that require visual contact, and sensitive information that requires face-to-face communication. 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, p. 1163).

Our study reveals a structural property of spatial networks—wide bridges—that has received far less attention. Complex contagions may favor spatial networks not only because the ties between nodes are physically short but also because the bridges between neighborhoods are structurally wide. While spatial proximity can make the connection relationally strong, it is the width of the bridge that makes the connection structurally strong for the propagation of complex contagions.[22]

These results have implications for the effects of different network topologies on the recruitment to what McAdam (1986) calls high risk/high cost activism. Macy (1990) has shown that long ties can be useful for solving coordination dilemmas in collective action when all that is needed is the transfer of information between group members. However, the optimal topology for the spread of collective action may depend on the costs and risks of participation and thus on the relative importance of information versus social reinforcement in mobilizing action. For students trying to organize a protest under a totalitarian regime (Zhao 1998), or for a movement facing state oppression (Opp and Gern 1993; Tilly 1978), simply having information about a collective action will be insufficient

[22] This structural property does not apply to all spatial networks. If neighborhood boundaries are dictated by the contours of physical space (such as streets, train tracks, rivers, or mountains), every member of a neighborhood will have the same set of neighbors; hence neighborhoods will not overlap. By comparing diffusion on spatial networks with bounded vs. overlapping neighborhoods, future studies can tease apart the relational effects of physical proximity from the structural effects of wide bridges.

to convince people to join. Resolving the coordination dilemma then requires multiple contacts who reinforce both the credibility of the information and the normative importance of taking action. Thus, consistent with McAdam (1986), our results show that the optimal networks for coordinating action will depend upon the costs and risks of participation.

Finally, these results have related implications for public health strategies for preventing the spread of infectious diseases. The long ties that accelerate the spread of disease are also the channels along which preventative information can quickly propagate. However, where public health innovations contravene existing social norms, health reform is likely to require social reinforcement, not simply access to information (Friedman et al. 1993; Latkin 1995; Pulerwitz, Barker, and Segundo 2004). While word-of-mouth transmission of new ideas may travel as quickly as the spread of a disease, the information may have little effect in changing entrenched yet risky behaviors without the social reinforcement provided by additional contacts (CDC 1997, pp. 3-2). Thus, while public health organizations may rely on peer networks to relay information about disease prevention (Friedman et al. 1993), these may not be the best pathways for effecting behavioral change that requires strong social reinforcement. Our results suggest that efforts to change behavioral norms through peer influence may reach greater numbers with greater speed by targeting tightly knit residential networks rather than the complex networks through which disease is more rapidly transmitted (like acquaintance or employment networks).

In sum, these theoretical results are consistent with recent findings on recruitment to Internet communities and with numerous case studies that document the importance of spatial diffusion and local recruitment to social movements. Although our study suggests novel insights into why diffusion often proceeds through spatial networks, other explanations are also possible, and new empirical studies are needed to test these alternative causal mechanisms directly.

In addition, much more work remains to be done to understand fully the effects of heterogeneity of thresholds, influence, and degree on the diffusion dynamics of complex contagions. In particular, we emphasize the need for research that carefully examines what happens as degree becomes correlated with influence and when there is homophily of degree and influence. Studies of nonspatial networks are also needed before we can generalize from the effects observed on lattices. In particular, randomization may promote propagation of complex contagions in sparse networks with few ties connecting large numbers of small but dense clusters. The present research clearly demonstrates the need for caution in generalizing the "strength of weak ties" from simple to complex contagions, but also suggests the need for further research on the social and

structural conditions that allow contagions to spread most effectively as thresholds increase.

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 simple contagions, like disease or information, can rapidly diffuse. It is tempting to regard this principle as a lawful regularity, in part because it justifies generalization from mathematically tractable random graphs to the structured networks that characterize patterns of social interaction. Nevertheless, our research cautions against uncritical generalization. Using Watts and Strogatz's original model of a small world network, we found that long ties do not always facilitate the spread of complex contagions and can even preclude diffusion entirely if nodes have too few common neighbors to provide multiple sources of confirmation or reinforcement. While networks with long, narrow bridges are useful for spreading information about an innovation or social movement, too much randomness can be inefficient for spreading the social reinforcement necessary to act on that information, especially as thresholds increase or connectedness declines.

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