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Complex Contagion and the Weakness of Long Ties in Social Networks: Revisited

GOLNAZ GHASEMIESFEH, Stony Brook University
ROOZBEH EBRAHIMI, Stony Brook University
JIE GAO, Stony Brook University

Diseases, information and rumors could spread fast in social networks exhibiting the small world property. In the diffusion of these "simple contagions", which can spread through a single contact, a small network diameter and the existence of weak ties in the network play important roles. Recent studies by sociologists [Centola and Macy 2007] have also explored "complex contagions" in which multiple contacts are required for the spread of contagion. [Centola and Macy 2007] and [Romero et al. 2011] have shown that complex contagions exhibit different diffusion patterns than simple ones. In this paper, we study three small world models and provide rigorous analysis on the diffusion speed of a k-complex contagion, in which a node becomes active only when at least k of its neighbors are active. Diffusion of a complex contagion starts from a constant number of initial active nodes. We provide upper and lower bounds on the number of rounds it takes for the entire network to be activated. Our results show that compared to simple contagions, weak ties are not as effective in spreading complex contagions due to the lack of simultaneous active contacts; and the diffusion speed depends heavily on the the way weak ties are distributed in a network.

We show that in Newman-Watts model with $\Theta(n)$ random edges added on top of a ring structure, the diffusion speed of a 2-complex contagion is $\Omega(\sqrt[3]{n})$ and $O(\sqrt[5]{n^4 \log n})$ with high probability. In Kleinberg's small world model (in which $\Theta(n)$ random edges are added with a spatial distribution inversely proportional to the grid distance to the power of 2, on top of a 2-dimensional grid structure), the diffusion speed of a 2-complex contagion is $O(\log^{3.5} n)$ and $\Omega(\log n / \log\log n)$ with high probability. We also show a similar result for Kleinberg's hierarchical network model, in which random edges are added with a spatial distribution on their distance in a tree hierarchy. In this model the diffusion is fast with high probability bounded by $O(\log n)$ and $\Omega(\log n / \log\log n)$, when the number of random edges issued by each node is $\Theta(\log^2 n)$. We also generalize these results to k-complex contagions.

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1. INTRODUCTION

Since social and information networks often have small diameters, diffusion of contagions in these networks could be fast. In a closer examination of the phenomena, Granovetter in 1973 [Granovetter 1973] attributed the fast diffusion in social networks to the *weak ties*, and termed it as '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*

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Author's addresses: Department of Computer Science, Stony Brook University, NY. Email: {gghasemiesfe,rebrahimi,jgao}@cs.stonybrook.edu.

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strong". In particular, social ties are often classified into ***strong ties*** and ***weak ties***. Strong ties represent close relationships such as family members, close friends, relationships that people invest time and energy to actively maintain; while weak ties capture relationships with acquaintances, friends from the old days, relationships that are relatively less invested.

Besides the relational meanings, strong ties and weak ties have structurally different meanings. Strong ties exhibit the *transitive closure* property, or *triadic closure* [Rapoport 1953] – if A and B are close friends, B and C are close friends, then it is very likely that A and C are also close friends. This poses structural limitations, in the sense that information spread in close triads tends to be redundant and does not help with diffusion. Indeed, it has been confirmed through social experiments that close friends are not so much helpful to learn about new opportunities or fresh ideas. Granovetter [Granovetter 1974] carried out experiments in which people were interviewed and asked how they got their current job. Many people learned information leading to their current jobs through personal contacts, not surprisingly. What was interesting is that these personal contacts were often described as acquaintances rather than close friends.

The strength of weak ties is further confirmed by the famous Milgram's experiment that observed the small world phenomena in real social networks, that people are connected by a very short path of length roughly six, i.e., 'six degree of separation' [Milgram 1967]. Thus, in such a network weak ties (which connect people that are otherwise at remote distances) and strong ties (which cluster people into tightly knitted communities) co-exist. The first mathematical model that captures both features is proposed by Watts and Strogatz [D.Watts and S.Strogatz 1998]. In their model the nodes are placed on a ring and nodes within small ring distances to each other are connected by strong ties. The edges are then 'randomly rewired' such that one endpoint is changed to a uniformly randomly chosen node. For a relatively large range of the rewiring probability, the resulted network has both a high clustering coefficient (i.e., closely knit clusters) and a small diameter. Thus, information can diffuse very fast even in networks with tightly clustered communities in which the majority of ties are strong ties. With only a small number of weak ties, information spreads to distant communities and in an exponential way spreads to the entire network.

In a recent paper [Centola and Macy 2007] Centola and Macy remarked that the strength of weak ties is not always so significant in helping diffusions, or, specifically does not help with the diffusion of *complex contagions*. Information or diseases are simple contagions. They could be spread through a single contact. Hence, a single weak tie can help affect remote regions, which greatly speeds up the diffusion. In some other cases, however, diffusion requires multiple confirmations or multiple contacts with affected nodes, to accumulate sufficient influence. Such cases include pricey technology innovations, the change of social behaviors, the decision to participate in a migration, etc [Coleman et al. 1966; Macdonald and Macdonald 1964]. Complex contagions appear due to strategic complementarity, credibility, legitimacy and emotional exchange as explained by Centola and Macy [Centola and Macy 2007]. While weak ties can carry information across long social distances, they are not as effective in spreading complex contagions, simply due to the lack of multiple, collective contacts. Thus, fast diffusion of complex contagion requires not only long bridges, but also "wide" ones, which may or may not exist. Therefore, complex contagions spread using mostly the strong ties and are going to be significantly slower in many settings. Analytic results and simulation results have been shown on the Watts and Strogatz model [D.Watts and S.Strogatz 1998]. Essentially, it was shown that when the contagion is merely minimally complex, i.e. requiring two active neighbors to be affected instead of one, it would require a substantially large number of random ties to even create one single 'bridge' to diffuse the contagion. The second more disturbing theorem says that random ties in fact erode the capability of spreading a complex contagion, holding the network density and size to be constant. This is understandable, as the more edges we randomly rewire, the less likely we are able to find wide and long bridges after all.

Eventually, the following very interesting question was posed in Centola and Macy's article: *How is it possible that complex contagions are able to spread through real social networks?* This is what motivates our work.

1.1. Summary of Models and New Results

In this paper we focus on the diffusion speed of a complex contagion in three social network models: the Newman-Watts model [Newman and Watts 1999], the Kleinberg's small world model [Kleinberg 2000] and Kleinberg's hierarchical network model [Kleinberg 2001]. In Newman-Watts model, n nodes are placed on a ring and nodes within ring distance 2 are connected by a strong tie. On top of that, random edges are *added* as weak ties (instead of rewiring existing edges in the grid as in Watts-Strogatz model [D.Watts and S.Strogatz 1998]). The nodes of each random edge are picked uniformly at random. In Kleinberg's small world model, nodes are placed on a 2-dimensional grid; and all nodes within Manhattan distance of 2 are connected by strong ties. The weak ties are added with a spatial distribution, in the sense that p connects an edge to q with probability proportional to $1/|pq|^{\alpha}$, where $\alpha \geq 0$ is a parameter and $|pq|$ is the grid distance between p and q. It has been shown by Kleinberg [Kleinberg 2000] that when $\alpha = 2$, the network not only has polylogarithmic diameter, but greedy routing based on the grid coordinates can lead to the destination in $O(\log^2 n)$ hops. In Kleinberg's hierarchical network model, nodes are represented as leaf nodes of a hierarchical organization structure. Random edges are added to the leaves with probability dependent on their tree distance. It has been shown by Kleinberg [Kleinberg 2001] that when each node has polylogarithmic out degree, greedy routing based on the tree distance can lead to destination in $O(\log n)$ hops.

All three models assume a hidden sociological space/metric, in particular, the ring structure, the grid structure and the hierarchical structure respectively. Distances in these social spaces capture the similarity of users in terms of attributes. Nodes that are nearby on the ring, grid or the hierarchy could be considered as having similar traits, living closer or within the same organizational unit. Thus, naturally we consider the non-random edges connecting nodes nearby on the ring and grid as ***strong ties*** and the randomly initiated edges as ***weak ties***. In the hierarchical model all edges are random. Hence, we just assume that the short distance edges represent the *strong ties* and the long ones represent *weak ties*. Notice that the main difference between the three models is the distribution of weak ties. As we will show later, this is the crucial difference resulting in different diffusion speed.

In all these models, we consider the general case when the networks are *undirected* graphs. Contagion can spread both ways along each edge. However, for the ease of explanation we sometimes care about the node that initiates a random edge and denote this edge as an ***outgoing*** edge. The node who receives a random edge from others would denote this edge as an ***incoming*** edge. Our analysis can be applied to directed graphs and this will be discussed in Section 5.

Complex Contagion

We say that a node is ***activated*** if it has adopted the contagion, otherwise the node is ***unactivated***. In a ***k-complex contagion*** diffusion, a node becomes activated only if it has at least $\boldsymbol{k}$ active neighbors, $k \geq 2$. Also, we assume that when a node becomes activated, it will remain active till the end of the diffusion. In all the three models, we focus on the case where the complex contagion is the only contagion in the network (i.e. no competitor), the same as the model chosen in [Centola and Macy 2007]. The model is synchronized and diffusion progresses in ***rounds***: If at round i, a node has at least k active neighbors, it will become active at round $i + 1$. We thus bound the total number of rounds for the entire network to be activated. Initially, a small number of active nodes, typically nodes connected by strong ties, exist in the graph, upon which diffusion bootstraps. We refer to these nodes as ***initial seeds***. Since our main objective is to examine the ***speed*** of diffusion, we choose network

parameters such that each node has at least k strong ties and k weak ties. A k-complex contagion always spreads to the entire network.

We start with a minimally complex contagion, the same as in [Centola and Macy 2007], i.e. $k = 2$. We show the following results for the diffusion speed of a 2-complex contagions:

(1) ***Newman-Watts model.*** The diffusion speed in this model is boudned by $\Omega(\sqrt[3]{n})$ and $O(\sqrt[5]{n^4 \log n})$ with high probability, which is considerably slow compared to simple contagion on the same graph (which is $\Theta(\log n)$).
(2) ***Kleinberg's small world model.*** If $\alpha = 2$, the diffusion speed is $O(\log^{3.5} n)$ and $\Omega(\log n / \log\log n)$ with high probability. For $0 < \alpha < 2$, the diffusion speed is $O(n^{\frac{4-2\alpha}{10-4\alpha}} \sqrt{\log^3 n})$ and $\Omega(\log n / \log\log n)$ with high probability.
(3) ***Kleinberg's hierarchical network model.*** When each node has $\Theta(\log^2 n)$ random edges the diffusion speed is $O(\log n)$ and $\Omega(\log n / \log\log n)$ with high probability.

These results can be generalized for larger constant values of k as long as we choose the strong ties and weak ties accordingly. Those results also follow the patterns explained above. We would like to emphasize the sharp contrast of the Newman-Watts model with Kleinberg's small world and hierarchical models. The main difference here is that the weak ties for Kleinberg's models are chosen with a non-uniform, spatial distribution, while the weak ties in Newman-Watts model are chosen uniformly at random. The distribution of weak ties thus becomes critical in accelerating complex contagion. Our results also give an interesting comparison of simple contagion and complex contagion in Newman-Watts model. For simple contagions, the diameter of the graph, which is $\Theta(\log n)$ [Flaxman and Frieze 2007; Bollobás and Chung 1988], is a natural bound for the diffusion speed. Hence, complex contagion in the Newman-Watts model is exponentially slower than simple contagion.

1.2. Related Work

Diffusion of information or viruses has been an active research topic for many years and a number of models have been used. A well accepted epidemic model is the SIR model, in which a node may experience one of the three stages: *susceptible*, i.e. a healthy node that could be infected by neighbors; *infectious*, i.e. a node that has caught the disease and has some probability of infecting susceptible neighbors; and *removed*, i.e. a node has recovered from the disease and is no longer infectious. There are many variations to the model, some eliminating the removed state, some adding a new state with temporary immunity. Detailed discussions of these models and their properties in networks can be found in [Easley and Kleinberg 2010]. In many of the previous epidemic studies, the main interest has been the analysis of the life time of a simple contagion in a network.

The closest model to our work is the *threshold model*, in which each node has a threshold on the number of active neighbors to become activated [Granovetter 1978]. It is shown that the problem of choosing the optimal set of initial active nodes to maximize the diffusion in these models is NP-Hard [Kempe et al. 2003, 2005] and remains NP-hard to approximate within a polylogarithmic factor [Chen 2008]. When all nodes have the same threshold and when the initial seeds are randomly chosen as a fraction f of nodes, this setting is termed as the *bootstrap percolation* [Chalupa et al. 1979; Adler 1991]. The focus is to examine the threshold f with which the infection eventually 'percolates', i.e. diffuses to the entire network. Study has been done on grid [van Enter 1987; Holroyd 2003], random regular graphs [Balogh and Pittel 2007], d-regular graphs [Blume et al. 2011], infinite trees [Balogh et al. 2006], random geometric graphs [Bradonjic and Saniee 2012], and more recently on complex networks [Baxter et al. 2010]. Our focus, however, is on the speed of the diffusion and its relationship to the network structure.

The threshold models and the diffusion of simple contagions in them have been studied extensively in the economic literature too, e.g. in [Ellison 1993; Young 2000]. In particular,

they consider the game theoretical setting when two behaviors are in competition and formulate the coordination game to examine how the competition evolves [Montanari and Saberi 2009; Goyal and Kearns 2012].

On the experimental side, complex contagions are observed in real world data sets. In [Romero et al. 2011], a large-scale experimental analysis of diffusion of hashtags on Twitter is presented. They show that beside the *stickiness* of a topic (how interesting the topic is by itself), its *persistence* (the effect of repeated exposure to the topic) plays an important role in diffusion of hashtags. Our paper takes the first step in providing theoretical analysis of complex contagion speed, which may shed light on how to understand/analyze real world data sets.

2. KLEINBERG'S SMALL WORLD MODEL

In this section, we prove polylogarithmic upper/lower bounds for the speed of complex contagion in Kleinberg's small world model introduced in [Kleinberg 2000].

2.1. Model

We start with n nodes in a $\sqrt{n}\times\sqrt{n}$ grid. In order to eliminate the boundary effect, we wrap up the grid into a torus – i.e., the top boundary is identified with the bottom boundary and the left boundary is identified with the right boundary. We consider two types of edges in this network. The *strong ties* are the edges that connect two nodes on the grid with Manhattan distance 2 or smaller. In addition, each node generates 2 random outgoing edges that are considered as *weak ties*. The probability that p chooses q as a neighbor through a weak tie is proportional to $1/|pq|^\alpha$, where $\alpha \geq 0$ is a parameter and $|pq|$ is the Manhattan distance between p and q. We consider a pair of grid neighbor nodes $\{u, v\}$ as the *initial seeds*, i.e. u and v have Manhattan distance of 1 on the grid.

The case of $\alpha = 2$ is the most interesting scenario of Kleinberg's small world model, in which greedy routing using the grid coordinates is able to deliver messages in $O(\log^2 n)$ steps. Thus, our focus is primarily on the $\alpha = 2$ case. The case of $\alpha = 0$ renders a 2-dimensional Newman-Watts network and its analysis is very similar to the analysis of the Newman-Watts model in Section 3.

2.2. Upper Bound for $\alpha = 2$

We start with inspecting random edge initiation probabilities. The probability that p initiates a weak tie to q is $\beta \times \frac{1}{|pq|^2} \times \frac{1}{\log n}$, where β is a constant making sure that the probability for all q's sum up to 1.

We look at the edges of a node p and how p may become activated. The edges that can activate p are of the following three types: (1) The weak ties initiated by p; (2) The weak ties initiated by other nodes connecting to p; (3) The strong ties of p.

Since we would like to upper bound the diffusion speed, in the following discussion we will ignore diffusion along the weak ties initiated by other nodes to p. That is, p is only activated through strong ties or through weak ties initiated by p. By artificially disabling diffusion along some edges or some direction of the edges, we will only make the whole diffusion process slower – as nodes may have been activated in earlier rounds if all edges are considered. In addition, when we analyze the process in different phases we may consider diffusion only through the weak ties or the strong ties. Again, this will only possibly make the diffusion process to be slower and our upper bound will hold. We will consider the diffusion process in two phases.

Phase 1: Local Diffusion

Starting from the initial seeds $\{u, v\}$, we define local diffusion to be diffusion through *only* strong ties. This type of diffusion behaves like growing a disk in ℓ_1 around the initial seeds. In each round the periphery of the active nodes are activated through local diffusion (which is a slow process).

CLAIM 1. *By only local diffusion, the number of rounds takes to activate all nodes within Manhattan distance x of an initial seed u is $O(x)$.*

We consider phase 1 as the period of time(rounds) at the end of which all the nodes within Manhattan distance $2r$ from u, denoted by $B_u(2r)$, have been activated, where $\boldsymbol{r = 3\log^{2.5} n/\sqrt{\gamma}}$ (γ is a constant to be determined later). Clearly, the number of rounds for phase 1 is no more than $O(\log^{2.5} n)$.

Phase 2: Exponential Diffusion

A generic scenario in phase 2 is that the set of active nodes is a superset of an active ***core $B_u(R)$***, where $R \geq 2r$. We will show that in polylogarithmic number of rounds, the active core *grows* to $B_u(2R)$ with high probability. Thus, complex contagion speeds up exponentially in the second phase.

THEOREM 2. *Suppose that the nodes within Manhattan distance R from u are active. $R \geq 2r$, $r = 3\log^{2.5} n/\sqrt{\gamma}$ and γ is a constant. With probability $1 - O(1/n^2)$, the number of rounds needed for all nodes within Manhattan distance $2R$ from u to be activated is $O(\log^{2.5} n)$.*

To prove this theorem, we will need the following lemma. Define by $A_u(x, c{\cdot}x)$ the annulus centered at u including all nodes within Manhattan distance $[x, c \cdot x]$ from u, where $c > 1$.

LEMMA 3. *Suppose the nodes inside a core $B_u(x)$ are active. Consider a pair of grid-neighbor nodes q, q' inside $A_u(x, 5x)$. The probability that q, q' are both activated using weak ties to the active core $B_u(x)$ is at least $\Omega(1/\log^4 n) = \gamma/\log^4 n$, for a constant γ.*

PROOF. If we only consider the outgoing edges of q, q', we can lower bound the probability that both q, q' are activated by the nodes in the core.

$$\begin{aligned}\text{Prob}\{q, q' \text{ are activated}\} \geq &\text{Prob}\{2 \text{ weak ties initiated by } q \text{ both fall in the core}\}\\ &\times\text{Prob}\{2 \text{ weak ties initiated by } q' \text{ both fall in the core}\}\end{aligned}$$

For every node q in the annulus $A_u(x, 5x)$ and any node $w \in B_u(x)$, the Manhattan distance from q to w is at most $6x$. The probability that q has initiated a weak tie to w is thus $\Omega\left(\frac{1}{\log n} \times \frac{1}{(6^2x^2)}\right)$. There are $\Theta(x^2)$ points inside the core. Hence, the probability that q initiates a weak tie to the core $B_x(u)$ is $\Omega(1/\log n)$. Since q, q' initiate total four weak ties and these events are independent of each other, the probability that q, q' are both activated is at least $\Omega(1/\log^4 n)$. □

PROOF OF THEOREM 2. The annulus $A_u(R, 2R)$ centered at u can be covered by $\Theta(R^2/r^2)$ ℓ_1-disks, each with radius r, by a packing argument. For example, we can put the centers of these small disks on a diamond shape lattice with diagonal width of each diamond as $2r$ and consider the disks that overlap with $A_u(R, 2R)$. Notice that these ℓ_1-disks are within an annulus $A_u[R - r, 2R + r]$. Since $R \geq 2r$ and $R - r \geq r$, we have:

$$\frac{2R + r}{R - r} = 2 + \frac{3r}{R - r} \leq 5.$$

Now, we consider the following 'balls and bins' problem. Each ℓ_1-disk is considered as a bin. Nodes in the union of the ℓ_1-disks are placed inside these bins. These small ℓ_1-disks are disjoint except at the boundary. For a node that is on the boundary of two adjacent ℓ_1-disks, we place it arbitrarily inside one of the bins. Each bin has $\Theta(r^2)$ nodes. We now consider a partition P of the nodes in a bin into disjoint pairs of grid neighbor nodes. There are at least $r^2/3$ such pairs inside each bin. Let p, q be one such pair in P. If both p, q become active, we say that they constitute a ***new seed***. Let $\mathcal{D}_{p,q}$ be the event that both of p, q have weak ties to the core. Consider an indexing I of the pairs inside P and let χ_i be the indicator random variable corresponding to one of $\mathcal{D}_{p,q}$ s.

Define $X = \sum_{i \in I} \chi_i$ to be the number of new seeds appearing among the pairs of partition P. By Lemma 3, $\text{Prob}\{\chi_i = 0\} \leq \left(1 - \frac{\gamma}{\log^4 n}\right)$, for a constant $\gamma > 0$. Also recall that $r = 3\log^{2.5} n/\sqrt{\gamma}$. Because the collection of $\mathcal{D}_{p,q}$ are defined on the edges initiated by p, q, the χ_is are independent of each other and we get:

$$\begin{aligned}\text{Prob}\{X = 0\} &= \text{Prob}\left\{\bigcap_{i \in I} \chi_i = 0\right\} = \prod_{i \in I} \text{Prob}\{\chi_i = 0\} \leq \prod_{i \in I}\left(1 - \frac{\gamma}{\log^4 n}\right) \\ &= \left(1 - \frac{\gamma}{\log^4 n}\right)^{r^2/3} = \left(1 - \frac{\gamma}{\log^4 n}\right)^{3\log^5 n/\gamma} \leq e^{-3\log n} \leq n^{-3}.\end{aligned}$$

This means that there is at least one new seed in a specific bin w.h.p. We now calculate the probability that each bin has at least one pair of new seeds.

$$\begin{aligned}&\text{Prob}\{\text{Each bin has at least one seed}\} \\ &= 1 - \text{Prob}\{\text{One or more bins have no seeds}\} \\ &\text{By union bound} \\ &\geq 1 - \Theta(R^2/r^2)\text{Prob}\{\text{A particular bin has no seeds}\} \\ &\geq 1 - \Theta(R^2/\log^5 n)(n^{-3}) \\ &\geq 1 - (1/n^2\log^5 n) \Leftarrow \text{Since } R^2 \leq n \\ &= 1 - O(1/n^2).\end{aligned}$$

We can see that once we have a pair of new seeds within each small disk, by local diffusion the number of rounds needed to activate all nodes in $A_{2R}(u)$ is $O(\log^{2.5} n)$, with probability $1 - O(1/n^2)$. □

THEOREM 4. *The number of rounds for the entire Kleinberg's small world network to be activated in a 2-complex contagion starting from a pair of seeds is bounded by $O(\log^{3.5} n)$ with high probability.*

PROOF. We divide phase 2 into sub-phases. In sub-phase i, we start from a core of active nodes within distance $2^i r$ from u, where $r = \Theta(\log^{2.5} n)$ and $1 \leq i \leq \Theta(\log n)$. By the end of this sub-phase the active nodes cover a core of active nodes with radius $2^{i+1}r$ around u. The number of rounds used in sub-phase i is denoted by τ_i. By Theorem 2, $\text{Prob}\left\{\tau_i = \omega(\log^{2.5} n)\right\} = O(1/n^2)$. There are at most $\Theta(\log n)$ sub-phases. Therefore, the probability that any of sub-phases take more than $O(\log^{2.5} n)$ rounds by union bound is at most $O(\log n/n^2)$. Hence, with probability at least $1 - O(\log n/n^2)$ all the sub-phases take at most $O(\log^{2.5} n)$ rounds. Thus, we get:

$$\text{Prob}\left\{\sum_i \tau_i = O(\log^{3.5} n)\right\} = 1 - O\left(\frac{\log n}{n^2}\right) = 1 - O\left(\frac{1}{n}\right)$$

The number of rounds for the diffusion process is dominated by *phase 2* and thus the theorem follows. □

2.3. Upper Bound for $0 < \alpha < 2$

Using a similar technique as in the previous section, we can derive upper bound for Kleinberg's small world model when $0 < \alpha < 2$. In this case, the probability that p links to q through a weak tie issued by p is: $\beta \times \frac{1}{|pq|^\alpha} \times \frac{1}{n^{1-\alpha/2}}$, where β is a constant normalization factor. The proof of the following corollary is in Appendix A.

COROLLARY 5. *For $0 < \alpha < 2$ in the Kleinberg's small world model, the number of rounds for the entire network to be activated in a 2-complex contagion starting from a pair of seeds is bounded by $O(n^{\frac{4-2\alpha}{10-4\alpha}} \sqrt{\log^3 n})$ with high probability.*

2.4. Lower Bound for $0 < \alpha \leq 2$

The diameter of the network is a natural lower bound on the speed of complex contagions. The diameter of Kleinberg's small world model, when weak ties are considered as directed edges, is shown to be $\Theta(\log n)$ [Martel and Nguyen 2004]. Note that the upper bound of $O(\log n)$ on the diameter also applies to the undirected model, as considered in this paper. Here, we give a high probable lower bound of $\Omega(\log n / \log\log n)$ for the diameter.

With all choices of $0 < \alpha \leq 2$, since each node initiates $O(1)$ edges and the graph is symmetric, the expected degree of nodes is $O(1)$. Because nodes initiate their edges independent of each other, we can apply Chernoff bound to get that the indegree of each node is $O(\log n)$ w.h.p. With union bound we get that the indegree of every node is $O(\log n)$ w.h.p.

LEMMA 6. *With high probability, the diameter of Kleinberg's small world network is $\Omega(\log n / \log\log n)$.*

PROOF. We start from a node s and build a breadth first search tree of the graph. Since w.h.p. all the nodes have at most $\phi \log n$ incoming/outgoing edges (each nodes initiates two outgoing edges), for some constant $\phi > 0$, the branching factor of the BFS tree is at most $\phi \log n$. The diameter of the graph is lower bounded by the maximum height of the tree, which solves to $\Omega(\log n / \log\log n)$. □

COROLLARY 7. *The number of rounds for the entire Kleinberg's small world network to be activated in a 2-complex contagion starting from a pair of seeds is $\Omega(\log n / \log\log n)$ with high probability.*

2.5. Generalization to K-Complex Contagion

For larger constant values of k, we can get similar results for k-complex contagions. We start from k initially active nodes in the network, chosen as a connected group of k nodes on the grid. We also change the network parameters such that all nodes within grid distance of $c_0 \geq k$ are connected by strong ties and each node initiates $c_1 \geq k$ weak ties to other nodes. In this case, we can derive similar bounds using the same techniques. We just state the results for such cases and skip their proofs.

COROLLARY 8. *If $c_0 \geq k$ and $c_1 \geq k$, the number of rounds needed for the entire Kleinberg's small world network to be activated in a k-complex contagion starting from k initially active nodes is:*

(1) $\Omega(\frac{\log n}{\log\log n})$ *and* $O(\log^{k+\frac{3}{2}} n)$, *when* $\alpha = 2$.

(2) $\Omega(\frac{\log n}{\log\log n})$ *and* $O\left(\log n \left(n^{2k-k\alpha} \log n\right)^{\frac{1}{4k+2-2k\alpha}}\right)$, *when* $0 < \alpha < 2$.

3. NEWMAN-WATTS MODEL

We present polynomial lower/upper bounds for the speed of complex contagions in the Newman-Watts small world model introduced in [Newman and Watts 1999].

3.1. Model

This model assumes a ring of n nodes. Nodes that are within distance 2 on the ring are connected by *strong ties*. In addition, each node issues two *weak ties*, with the two endpoints uniformly randomly selected among the rest of nodes. This model is very similar to the Watts-Strogatz model [D.Watts and S.Strogatz 1998]. The only difference is that in the Newman-Watts model weak ties are randomly added *in addition* to the strong ties, while in Watts-Strogatz model existing edges are randomly rewired, thus may destroy the ring structure when the probability of random rewiring is high. Like before, we consider complex contagion in which a node is activated by *two* active neighbors. We assume that a pair of neighbor nodes on the ring, $\{u, v\}$, are the *initial seeds*.

3.2. Lower Bound

Define ***F to be an interval on the ring of length $\sqrt[3]{n}$ centered on the initial pair of seeds***. Now, we want to argue that the chance of any node having two weak ties to nodes of F is small. Under this condition complex contagion inside F becomes a ***limited diffusion process***. In this limited process, nodes could become activated using only either (1) strong ties, or (2) a combination of one weak tie and one strong tie. Notice that nodes that become activated using at least one strong tie are in the neighborhood of active nodes in the previous round along the ring.

We consider a ***speed-up diffusion process*** within F. If the process is a *limited diffusion*, in each round the set of active nodes will always be in a format of a set of intervals in F. We know that using strong ties nodes at ring distance 1 on the boundary of an interval of active nodes will surely become activated. In the *speed-up process*, nodes within ring distance 2 from an interval of active nodes (having at least one strong tie) are ***assumed*** to be activated as well. This will only speed up the process and thus the bound we calculate will be a lower bound.

We realize that before the nodes of F become all activated, the activated nodes must have the pattern of a single interval along the ring and each round will grow 4 nodes by extension to two nodes on each direction. Hence, the number of rounds for only the nodes of F to be activated would be $\Theta(|F|) = \Theta(\sqrt[3]{n})$.

First, we examine the following two events: For a node $q \in F$, we define by $\boldsymbol{\mathcal{I}_q(F)}$ the event that q is connected by at least 2 weak ties to $F \setminus \{q\}$. Also, for a node $p \notin F$, define by $\boldsymbol{\mathcal{O}_p(F)}$ the event that p is connected by at least 2 weak ties to F. The following lemma shows the probability of each event. Note that we need to consider weak ties initiated by q (or p) and those possibly initiated by nodes of F to q (or p).

LEMMA 9. *Let F be any interval on the ring of length $s \leq n/2$ centered on the initial pair of seeds. We have:*

$$Prob\{\mathcal{I}_q(F)\} = Prob\{\mathcal{O}_p(F)\} = \Theta\left(\frac{s^2}{n^2}\right)$$

The detailed proofs of lemma 9 is based on direct probability computation and is given in appendix B. Plug in the size of $|F| = \Theta(\sqrt[3]{n})$ and we have that $\text{Prob}\{\mathcal{I}_p(F)\} = \text{Prob}\{\mathcal{O}_p(F)\} = \Theta\left(1/(n^{4/3})\right)$. Now we are ready to prove the lower bound result.

THEOREM 10. *With probability at least $1 - \Theta\left(1/\sqrt[3]{n}\right)$, it takes at least $\Omega(\sqrt[3]{n})$ rounds for a 2-complex contagion to activate all nodes in the Newman-Watts Network.*

PROOF. We give a lower bound on the number of rounds needed to activate all the nodes in F. We show that with probability at least $1-\Theta\left(1/\sqrt[3]{n}\right)$, no node inside/outside of F has two weak ties to F.

Nodes inside F. Let the random variable X be the number of nodes $q \in F$ that have at least two random edges to/from $F \setminus \{q\}$. By linearity of expectation, $E[X] = |F| \times \text{Prob}\{\mathcal{I}_q(F)\} = |F| \times \Theta\left(1/(n^{4/3})\right) = \Theta\left(1/n\right)$. By Markov inequality we have: $\text{Prob}\{X \geq 1\} = \Theta\left(1/n\right)$. Thus, $\text{Prob}\{X = 0\} = 1 - \Theta\left(1/n\right)$.

Nodes outside F. Let the random variable Y be the number of nodes $q' \notin F$ that have at least two random edges to/from F. Again by linearity of expectation, $E[Y] = (n - |F|) \times \Theta\left(1/(n^{4/3})\right) = \Theta\left(1/\sqrt[3]{n}\right)$. By Markov inequality we have: $\text{Prob}\left\{Y \geq 1\right\} = \Theta\left(1/\sqrt[3]{n}\right)$. Thus, $\text{Prob}\left\{Y = 0\right\} = 1 - \Theta\left(1/\sqrt[3]{n}\right)$.

We are now able to give the lower bound on the number of rounds needed to activate nodes of F. By the above analysis, the probability that no node has at least two weak ties to F is, by union bound, at least $\left(1 - \Theta\left(1/\sqrt[3]{n}\right)\right) \times \left(1 - \Theta\left(1/n\right)\right) = 1 - \Theta\left(1/\sqrt[3]{n}\right)$. Hence with the same probability, the *speed-up diffusion process* will only grow 4 active nodes in each round before all nodes of F become activated. Thus, the number of rounds needed just to activate F is $\Theta(\sqrt[3]{n})$ w.h.p. □

THEOREM 11. *With probability at least $1 - \Theta\left(1/\log n\right)$, it takes at least $\Omega(\sqrt{n/\log n})$ rounds for a 2-complex contagion to activate all nodes in the Newman-Watts network.*

PROOF. By setting the size of F to be $\sqrt{n/\log n}$, we get a probability of $\text{Prob}\left\{\mathcal{I}_q(F)\right\} = \text{Prob}\left\{\mathcal{O}_q(F)\right\} = \Theta(1/n\log n)$ from lemma 9. Repeating the argument of the above theorem gives us that $\text{Prob}\left\{X \geq 1\right\} = \Theta(1/\sqrt{n/\log n})$ and $\text{Prob}\left\{Y \geq 1\right\} = \Theta(1/\log n)$. The rest of the argument is the same as above. □

3.3. Upper Bound

To get the upper bound result, we use a similar technique as in Section 2. We partition the nodes on the ring into continuous intervals $\mathcal{G}_i$ of $2\sqrt[5]{n^4 \log n}$ nodes each. In particular, u and v are in the middle of $\mathcal{G}_1$.

Each interval $\mathcal{G}_j$ has a set of nodes consecutively located on the ring. Consider a partition Φ_j of the nodes in each $\mathcal{G}_j$ into disjoint pairs of ring neighbor nodes: We pair up the k-th and $(k+1)$-th node into $|\mathcal{G}_j|/2$ number of disjoint pairs, denoted by $\mathcal{P}_i^j$, $i \in I = \{1, \cdots, |\mathcal{G}_j|/2\}$. For each pair $\mathcal{P}_i^j = (p, q)$, define the event $\mathcal{A}_i^j$ as the event that p and q both have at least two weak ties to $\mathcal{G}_1$. By considering only weak ties initiated by p and q (that are chosen independently by p, q), we can lower bound $\text{Prob}\left\{\mathcal{A}_i^j\right\}$ by:

$$\text{Prob}\left\{\mathcal{A}_i^j\right\} \geq \left(\binom{|\mathcal{G}_1|}{2} / \binom{n-1}{2}\right)^2 = \Omega\left(\frac{|\mathcal{G}_1|}{n}\right)^4 = \Omega\left(\frac{16\log^{4/5} n}{n^{4/5}}\right)$$

Let $\chi(\mathcal{A}_i^j)$ be the indicator random variable of $\mathcal{A}_i^j$. Consider the sum $Z_j = \sum_{i \in I} \chi(\mathcal{A}_i^j)$.

LEMMA 12. *With high probability there exists one pair of new seeds in $\mathcal{G}_j$. That is w.h.p. we have $Z_j > 1$ for all j.*

PROOF. Since $\mathcal{A}_i^j$ are defined on outgoing edges, they are independent of each other. Therefore, we can write:

$$\begin{aligned}\text{Prob}\{Z_j = 0\} &= \text{Prob}\left\{\bigcap_{i\in I} \chi(\mathcal{A}_i^j) = 0\right\} = \prod_{i\in I} \text{Prob}\left\{\chi(\mathcal{A}_i^j) = 0\right\} \\ &\leq \prod_{i\in I}\left(1 - \frac{16\log^{4/5} n}{n^{4/5}}\right) = \left(1 - \frac{16\log^{4/5} n}{n^{4/5}}\right)^{|\mathcal{G}_i|/2} \\ &\leq e^{-16\log n} \leq 1/n^{16}.\end{aligned}$$

□

Now, we can provide an upper bound for a speed of a complex contagion in Newman-Watts model.

THEOREM 13. *With high probability, it takes at most $O(\sqrt[5]{n^4\log n})$ rounds for a 2-complex contagion to affect all the nodes in Newman-Watts network.*

PROOF. If we neglected propagation through weak ties, then it would take at most $t_1 = O(\sqrt[5]{n^4\log n})$ rounds for $\mathcal{G}_1$ to become active using strong ties only. By Lemma 12, for each j, with probability at least $1 - O(1/n^{16})$, at least one pair of neighbor nodes in $\mathcal{P}_i^j$ has two weak ties to $\mathcal{G}_1$ and thus is activated in round $t_1 + 1$. Hence, by union bound the probability that there is at least one pair of new seeds in every interval $\mathcal{G}_j$, for all j, is at least $1 - n/n^{16} = 1 - 1/n^{15}$. Again, if we neglected propagation through weak ties each set $\mathcal{G}_j$ will be activated no later than round number $t_1 + O(\sqrt[5]{n^4\log n}) = O(2t_1)$. Therefore, it takes at most $O(\sqrt[5]{n^4\log n})$ rounds for a complex contagion to activate the whole network. □

3.4. Generalization to K-Complex Contagion

Again, similar results can be shown for complex contagion for other constant values of k. We start from an interval of k active nodes on the ring. We assume that a node has strong ties to nodes within ring distance of $c_0 \geq k$ and each node initiates $c_1 \geq k$ weak ties. We just state the results for such cases and skip their proofs.

COROLLARY 14. *If $c_0 \geq k$ and $c_1 \geq k$, then the number of rounds needed for the entire Newman-Watts network to be activated in a k-complex contagion is bounded from above and below:*

(1) With probability at least $1 - \Theta(1/\sqrt[3]{n})$, the number of rounds is $\Omega(n^{1-\frac{4}{3k}})$ and with probability at least $1 - \Theta(1/\log n)$, the number of rounds is $\Omega\left(\left(n^{k-1}/\log n\right)^{\frac{1}{k}}\right)$.

(2) With high probability, the number of rounds is $O\left(\left(n^{2k}\log n\right)^{\frac{1}{2k+1}}\right)$.

4. KLEINBERG'S HIERARCHICAL MODEL

Finally, we analyze complex contagion in Kleinberg's hierarchical network model introduced in [Kleinberg 2001].

4.1. Model

In Kleinberg's hierarchical network model, the set of nodes, V, is embedded as leaves of an organization hierarchy, represented by a complete b-ary tree T, where $b \geq 2$ is a constant. We denote the height of the least common ancestor of two leaf nodes u and v in T by $h(u, v)$, where $1 \leq h(u, v) \leq \log_b n$. Each node of v initiates j random edges to other nodes

and the chance of v connecting to w is proportional to $1/b^{h(v,w)\cdot\alpha}$, where α is a constant parameter. In this section we focus on the case of $\alpha = 1$, in which greedy routing using the distance on the hierarchy guarantees to find a short path of $O(\log n)$ hops [Kleinberg 2001]. In this case, the probability that u chooses to build an edge to w is $\psi/b^{h(u,w)}$, where $\psi = \frac{b}{b-1}\frac{1}{\log_b n}$ is a normalization factor to ensure that the probabilities sum up to 1. In our analysis we assume that $j = c\log_b^2 n$ for some $c > 3(b-1)\ln b$. For such values of j, the graph has polylogarithmic greedy routing and thus must be connected w.h.p. [Kleinberg 2001]. To simplify the analysis we allow repetitions[1] and assume that a node becomes activated if it has two random edges that connect to active nodes (or the same active node). The contagion process starts with 2 *initial seeds* $\{u, v\}$ who share a common parent.

4.2. Upper Bound

Similar to the other upper bound proofs presented earlier, we only consider the activation of a node p through edges initiated by p. Again this will only make the diffusion process slower. Consider a family of fixed sets $\{\mathcal{F}_i\}$ in the graph as follows: $\mathcal{F}_1$ includes u, v and their siblings. For all $1 \le i \le \lceil \log_b n \rceil$, inductively define $\mathcal{F}_{i+1}$ as the set of nodes in the subtree rooted at the parent of $\mathcal{F}_i$' root. Hence $\mathcal{F}_{\lceil \log_b n \rceil}$ contains all the nodes in the network.

For a node $p_{i+1} \in \mathcal{F}_{i+1} \setminus \mathcal{F}_i$, it has a common ancestor of height $i+1$ with all nodes in $\mathcal{F}_i$. Thus, a specific edge initiated by p_{i+1} connects to nodes in $\mathcal{F}_i$ with probability: $\psi\frac{|\mathcal{F}_i|}{b^{i+1}} = \frac{1}{(b-1)\log_b n}$. The proof of the following lemma is in appendix C.

LEMMA 15. *With high probability, p_{i+1} initiates at least 2 edges to $\mathcal{F}_i$.*

THEOREM 16. *If $j = c\log_b^2 n$ for a constant $c \ge 3(b-1)\ln b$, with high probability it takes at most $\lceil \log_b n \rceil$ rounds for the 2-complex contagion to affect all the nodes in Kleinberg's hierarchical network.*

PROOF. We are going to inductively prove that each set $\mathcal{F}_i$ will be activated no later than round number i with probability at least $\left(1 - \gamma \log_b n/n^\beta\right)^{|\mathcal{F}_i|-2}$, for a constant $\gamma = c/(b-1)$ and $\beta = \frac{c}{(b-1)\ln b}$.

For the induction base, we know that the initial seed nodes $\{u, v\} \in \mathcal{F}_1$. If $b > 2$, $\mathcal{F}_1$ contains other nodes than u, v. Consider a node $q \in \mathcal{F}_1 \setminus \{u, v\}$, q has the same parent as $\{u, v\}$. A specific edge initiated by q is connected to u (or v) with probability: $\psi/b = \frac{1}{(b-1)\log_b n}$. Hence, similar to Lemma 15, we can prove that with probability $1 - \gamma\log_b n/n^{2\beta} \ge 1 - \gamma\log_b n/n^\beta$, q has at least 2 random edges to u, v. Therefore, with probability at least $\left(1 - \gamma\log_b n/n^\beta\right)^{b-2}$ all the nodes in $\mathcal{F}_1$ will be affected in round 1.

Assume inductively that the set $\mathcal{F}_i$ is affected no later than round i with probability at least $\left(1 - \gamma\log_b n/n^\beta\right)^{|\mathcal{F}_i|-2}$. By Lemma 15, the probability that all nodes in $\mathcal{F}_{i+1} \setminus \mathcal{F}_i$ are activated in the next round is at least $\left(1 - \gamma\log_b n/n^\beta\right)^{|\mathcal{F}_{i+1}|-|\mathcal{F}_i|}$. Thus, all nodes in $\mathcal{F}_{i+1}$ are activated in round $i+1$ with probability at least:

$$\left(1 - \gamma\log_b n/n^\beta\right)^{|\mathcal{F}_{i+1}|-|\mathcal{F}_i|} \times \left(1 - \gamma\log_b n/n^\beta\right)^{|\mathcal{F}_i|-2} = \left(1 - \gamma\log_b n/n^\beta\right)^{|\mathcal{F}_{i+1}|-2}.$$

Hence, the whole graph is going to be affected in $\lceil \log_b n \rceil$ rounds with probability at least:

$$\left(1 - \gamma\log_b n/n^\beta\right)^{n-2} = 1 - O\left(\log_b n/n^{\beta-1}\right).$$

We choose $c \ge 3(b-1)\ln b$ such that $\beta \ge 3$. In this case the whole network can be activated within $\log_b n$ rounds with probability at least $1 - O(1/n)$. □

[1] Alternatively, we can also sample without repetition and use negative dependencies with Chernoff bounds.

We remark that a by-product of theorem 16 is an upper bound of $O(\log_b n)$ on the diameter of the network.

4.3. Lower Bound

The diameter of the network is a natural lower bound for the speed of complex contagions. In this section, we give a lower bound for the diameter of the hierarchical model. We know that each node has $j = c \log_b^2 n$ outgoing random edges.

Similar to the analysis of the diameter of Kleinberg's small world model, we are going to bound the number of *incoming random* edges of a node. Since each node initiates $O(\log^2 n)$ edges and the graph is symmetric, the expected degree is $O(\log^2 n)$. Because nodes initiate their edges independent of each other, we can apply Chernoff bound to get that the indegree of each node is $O(\log^2 n)$ w.h.p. With union bound we get that the indegree of every node is $O(\log^2 n)$ w.h.p.

With the same argument as in lemma 6, we can show the following:

COROLLARY 17. *If $j = c \log_b^2 n$ for a constant $c \geq 3(b-1)\ln b$, w.h.p. the diameter of Kleinberg's hierarchical network is $\Omega(\log_b n / \log_b \log_b n)$.*

COROLLARY 18. *If $j = c \log_b^2 n$ for a constant $c \geq 3(b-1)\ln b$, w.h.p. it takes at least $\Omega(\log_b n / \log_b \log_b n)$ rounds for the 2-complex contagion to affect all the nodes in Kleinberg's hierarchical network.*

4.4. Generalization to K-Complex Contagion

We can derive the same result for k-complex contagion (for a constant k):

COROLLARY 19. *If $j = c \log_b^2 n$ for a constant $c \geq 3(b-1)\ln b$, w.h.p. a k-complex contagion takes at least $\Omega(\log_b n / \log_b \log_b n)$ and at most $O(\log_b n)$ rounds to affect all the nodes in Kleinberg's hierarchical network.*

5. COMPLEX CONTAGION IN DIRECTED NETWORKS

We can extend our results to directed graphs. First, we need to refine contagion and influence by directed edges. In the context of complex contagion and information diffusion, an edge from p to q typically means that p actively follows the information of q. Therefore, it is natural to assume that influence propagates in the ***reverse*** direction of a directed edge – when p follows q, p can be influenced by behaviors or opinions of q. In a ***k-complex directed contagion***, we say that a node p becomes *active* if p follows at least k activated nodes through its ***undirected*** or ***outgoing*** edges.

The initial model for Kleinberg's small world graph is a directed graph, in which the random edge initiated by p to q is single directional pointing to q. The stong ties edges are still bi-directional. In Kleinberg's hierarchical model, all edges are random and directional. The Newman-Watts model, is by default an undirected graph. We can follow the same rule and consider the ring edges as undirected and the random edges as directed ones. In all the upper bound proofs, we only relied on the outgoing edges and given our interpretation of complex contagion, all the analysis remain intact for the directed case. In the lower bound proofs, the results still hold/or get stronger for the following reasons.

(1) Kleinberg's small world model: It has been shown that the diameter of Kleinberg's *directed* small world model is $\Theta(\log n)$ [Martel and Nguyen 2004]. Hence, if the network is directed, the lower bound on complex contagion becomes $\Omega(\log n)$ instead of $\Omega(\log n / \log \log n)$.
(2) Kleinberg's heirarchical model: The analysis of the diameter relies on the degree of a node in the undirected case. If only outgoing edges are considered, the proof of Corol-

lary 17 would not change and the diameter would be the same. Therefore, the lower bound analysis would also work here.

(3) Newman-Watts Model: For the *lower bound* analysis, we have to notice that if we only consider the outgoing edges in Lemma 9, then inequality 2 becomes an equality as follows ($|\mathcal{F}| = s$): $\text{Prob}\{\mathcal{I}_q(F)\} = \text{Prob}\{\mathcal{O}_q(F)\} = \binom{s}{2}/\binom{n-1}{2} = \Theta\left(s^2/n^2\right)$. This would result to the same bounds for $\mathcal{I}_q(F)$ and $\mathcal{O}_q(F)$ in Lemma 9 and thus the same lower bound would hold.

6. CONCLUSION

Our results show significant differences in the diffusion of simple and complex contagions. While the diffusion speed of simple contagion is simply dependent on the diameter of the graph, the diffusion speed of complex contagion is much more sensitive to other network properties, i.e. the topology of the network and the way weak ties are distributed in the network. This improves our understanding of diffusion in random networks.

Closing the gap between the given upper and lower bounds in all three models remains an open question. A particular special case is to tighten upper/lower bounds for the Kleinberg's small world model for $0 < \alpha < 2$ and figure out the regime of the speed of complex contagion (polynomial or polylogarithmic). In the future, we would also like to study other social network models such as networks with power law distribution, i.e. the preferential attachment model, as well as the diffusion speed dependent on the number of initial seeds.

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A. PROOFS IN KLEINBERG'S SMALL WORLD MODEL

PROOF COROLLARY 5. Similar to the analysis of the case of $\alpha = 2$, we divide the diffusion into two phases. Phase 1 stops when all the nodes within Manhattan distance $2r$ from u have been activated, where $r = n^{\frac{4-2\alpha}{10-4\alpha}}\sqrt{\log n}$. The number of rounds for phase 1 is no more than $O\left(n^{\frac{4-2\alpha}{10-4\alpha}}\sqrt{\log n}\right)$. At the end of phase 1 a core $B_u(2r)$ is activated.

At phase 2, suppose that a core $B_u(R)$ is active, $R \geq 2r$. We can show in a similar argument to Theorem 2, that with high probability the number of rounds needed for all nodes within Manhattan distance $2R$ from u to be activated is $O\left(n^{\frac{4-2\alpha}{10-4\alpha}}\sqrt{\log n}\right)$.

For a node $q \in A_u(R, 2R)$, the probability that q initiates a node to nodes inside $B_u(R)$ is $\Omega\left(\frac{1}{n^{1-\alpha/2}} \times \frac{1}{R^\alpha}\right) \times \Theta(R^2) = \Omega\left(\frac{R^{2-\alpha}}{n^{1-\alpha/2}}\right)$. Thus, the probability that two grid neighbor nodes q, q' both initiate weak ties to the core is $\Omega\left(\frac{R^{8-4\alpha}}{n^{4-2\alpha}}\right)$.

Again, each bin is of size r^2 and there are $\Theta(R^2/r^2)$ in the annulus. The expected number of *new seeds* in each bin is thus $\Omega\left(r^2\frac{R^{8-4\alpha}}{n^{4-2\alpha}}\right) = \Omega\left(\frac{r^{10-4\alpha}}{n^{4-2\alpha}}\right)$. Replacing the value of r, we get that each bin gets $\Omega(\log n)$ new seeds in expectation and at least 1 new seed w.h.p.

Therefore, analogous to the proof of theorem 4, there will be $O(\log n)$ rounds of doubling the radius of core, each of which taking at most $O\left(n^{\frac{4-2\alpha}{10-4\alpha}}\sqrt{\log n}\right)$ rounds. Hence, we can prove that with high probability it takes at most $O\left(n^{\frac{4-2\alpha}{10-4\alpha}}\sqrt{\log^3 n}\right)$ number of rounds for the entire network to be activated starting from a pair of seeds. □

B. PROOFS IN THE NEWMAN-WATTS MODEL

The statement of lemma 9 is the following: For all sets s of size at most $n/2$

$$\text{Prob}\left\{\mathcal{I}_{\mathbf{p}}(\mathbf{F})\right\} = \text{Prob}\left\{\mathcal{O}_{\mathbf{p}}(\mathbf{F})\right\} = \Theta\left(\frac{s^2}{n^2}\right).$$

These probabilities are in fact equal to each other. In oder to compute Prob$\{\mathcal{I}_q(F)\}$, we divide it into disjoint events: $\{\mathcal{I}_q^k(F)\}$ = The event that a specific node q has *exactly* k *random* edges to/from F.

LEMMA 20. *Assume that the size of F is s. Then we have:*

$$Prob\{\mathcal{I}_q^k(F)\} \leq \left(\left(\frac{2e}{k}\right)^k + 2\left(\frac{2e}{k-1}\right)^{k-1} + \left(\frac{2e}{k-2}\right)^{k-2}\right)\frac{s^2\left(\frac{s}{n-1}\right)^{k-2}}{(n-1)(n-2)}.$$

PROOF. The event $\{\mathcal{I}_q^k(F)\}$ can happen if one of the following happens:

(1) $\{\alpha_q^k(F)\}$: Node q has *exactly* 2 *outgoing random edges* and *exactly* $k-2$ *incoming random edges* from F.
(2) $\{\beta_q^k(F)\}$: Node q has *exactly* 1 *outgoing random edges* and *exactly* $k-1$ *incoming random edges* from F.
(3) $\{\gamma_q^k(F)\}$: Node q has *exactly* 0 *outgoing random edges* and *exactly* k *incoming random edges* from F.

As such, we can write $\{\mathcal{I}_q^k(F)\}$ as: $\{\mathcal{I}_q^k(F)\} = \{\alpha_q^k(F)\} \cup \{\beta_q^k(F)\} \cup \{\gamma_q^k(F)\}$.

We now compute the probability of these three sub-events:

(1) For Prob$\{\alpha_q^k(F)\}$ we can write:

$$\begin{aligned}
\text{Prob}\{\alpha_q^k(F)\} &= \frac{\binom{s}{2}}{\binom{n-1}{2}}\binom{s}{k-2}\left(\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{k-2}\left(1-\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{s-k+2} \\
&= \frac{s(s-1)}{(n-1)(n-2)}\binom{s}{k-2}\left(\frac{2}{n-1}\right)^{k-2}\left(\frac{n-3}{n-1}\right)^{s-k+2} \\
&\leq \frac{s(s-1)}{(n-1)(n-2)}\left(\frac{se}{k-2}\right)^{k-2}\left(\frac{2}{n-1}\right)^{k-2} \\
&\leq \frac{s^2}{(n-1)(n-2)}\left(\frac{2e}{k-2}\right)^{k-2}\left(\frac{s}{n-1}\right)^{k-2}.
\end{aligned}$$

(2) For Prob$\{\beta_q^k(F)\}$ we can write:

$$\begin{aligned}
Pr\{\beta_u^k(S)\} &= \frac{\binom{s}{1}\binom{n-s-1}{1}}{\binom{n-1}{2}}\binom{s}{k-1}\left(\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{k-1}\left(1-\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{s-k+1} \\
&= \frac{2s(n-s-1)}{(n-1)(n-2)}\binom{s}{k-1}\left(\frac{2}{n-1}\right)^{k-1}\left(\frac{n-3}{n-1}\right)^{s-k+1} \\
&\leq \frac{2s(n-s-1)}{(n-1)(n-2)}\left(\frac{se}{k-1}\right)^{k-1}\left(\frac{2}{n-1}\right)^{k-1} \\
&\leq \frac{2s^2}{(n-1)(n-2)}\left(\frac{2e}{k-1}\right)^{k-1}\left(\frac{s}{n-1}\right)^{k-2}\frac{n-s-1}{n-1} \\
&\leq \frac{2s^2}{(n-1)(n-2)}\left(\frac{2e}{k-1}\right)^{k-1}\left(\frac{s}{n-1}\right)^{k-2}.
\end{aligned}$$

(3) And finally for Prob$\{\gamma_q^k(F)\}$:

$$\begin{aligned}
Pr\{\gamma_u^k(S)\} &= \frac{\binom{n-s-1}{2}}{\binom{n-1}{2}}\binom{s}{k}\left(\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{k}\left(1-\frac{\binom{n-2}{1}}{\binom{n-1}{2}}\right)^{s-k} \\
&= \frac{(n-s-1)(n-s-2)}{(n-1)(n-2)}\binom{s}{k}\left(\frac{2}{n-1}\right)^{k}\left(\frac{n-3}{n-1}\right)^{s-k} \\
&\leq \frac{(n-s-1)(n-s-2)}{(n-1)(n-2)}\left(\frac{se}{k}\right)^{k}\left(\frac{2}{n-1}\right)^{k} \\
&\leq \frac{s^2}{(n-1)(n-2)}\left(\frac{2e}{k}\right)^{k}\left(\frac{s}{n-1}\right)^{k-2}\frac{n-s-1}{n-1}\frac{n-s-2}{n-1} \\
&\leq \frac{s^2}{(n-1)(n-2)}\left(\frac{2e}{k}\right)^{k}\left(\frac{s}{n-1}\right)^{k-2}.
\end{aligned}$$

By summing all the probabilities we arrive at the statement of the lemma. □

Proof of Lemma 9. We proceed using the derived bound from lemma 20. A node q can have between 2 and $s+2$ random edges to/from F. Therefore, to get the probability

of having at least 2 random edges to F, we write the following sum:

$$\begin{aligned}\text{Prob}\{\mathcal{I}_q(F)\} &= \sum_{k=2}^{s+2} \text{Prob}\{\mathcal{I}_q^k(F)\} \\ &\leq \sum_{k=2}^{s+2} \left(\left(\frac{2e}{k}\right)^k + 2\left(\frac{2e}{k-1}\right)^{k-1} + \left(\frac{2e}{k-2}\right)^{k-2} \right) \frac{s^2 \left(\frac{s}{n-1}\right)^{k-2}}{(n-1)(n-2)}.\end{aligned}$$

Notice that for all $2 \leq k \leq s+2$,

$$\left(\left(\frac{2e}{k}\right)^k + \left(\frac{2e}{k-1}\right)^{k-1} + \left(\frac{2e}{k-2}\right)^{k-2} \right) \leq 4(\frac{e}{2})^4 \leq 16.$$

Hence, we derive:

$$\begin{aligned}\text{Prob}\{\mathcal{I}_q(F)\} &\leq \frac{16s^2}{(n-1)(n-2)} \sum_{k=2}^{s+2} \left(\frac{s}{n-1}\right)^{k-2} = \frac{16s^2}{(n-1)(n-2)} \frac{1-\left(\frac{s}{n-1}\right)^{s+1}}{1-\left(\frac{s}{n-1}\right)} \\ &\leq \frac{16s^2}{(n-1)(n-2)} \frac{1}{1-\left(\frac{s}{n-1}\right)} = \frac{16s^2}{(n-1)(n-2)} \frac{n-1}{n-1-s}.\end{aligned}$$

For all $s \leq n/2$, we have that $\frac{n-1}{n-1-s} < 2$. So we get:

$$\text{Prob}\{\mathcal{I}_q(F)\} \leq \frac{32s^2}{(n-1)(n-2)}. \tag{1}$$

To get the lower bound, notice that q chooses among the $\binom{n-1}{2}$ pairs of other nodes as the other end of its outgoing random edges with uniform probability: $2/(n-1)(n-2)$. Hence

$$\begin{aligned}&\text{Prob}\{\mathcal{I}_q(F)\} \geq \frac{\binom{s}{2}}{\binom{n-1}{2}} = \frac{s(s-1)}{(n-1)(n-2)} = \Omega\left(\frac{s^2}{n^2}\right) \qquad (2) \\ &\text{from eq. 1 we have: } \text{Prob}\{\mathcal{I}_{\mathbf{q}}(\mathbf{F})\} = \Theta\left(\frac{s^2}{n^2}\right).\end{aligned}$$

All the above argument also holds for $\text{Prob}\{\mathcal{O}_p(F)\}$ without any change, since we didn't use the fact that $q \in F$ at all. So for $p \notin F$, all the probabilities are the same and the we get the statement of the lemma. □

C. PROOFS IN KLEINBERG'S HIERARCHICAL MODEL

Proof Lemma 15. The probability that p_{i+1} initiates no edges to $\mathcal{F}_i$ is $\left(1 - \frac{1}{(b-1)\log_b n}\right)^{c \log_b^2 n} = \Theta(1/n^\beta)$, where $\beta = \frac{c}{(b-1)\ln b}$. The probability that p_{i+1} initiates exactly one edge to $\mathcal{F}_i$ is $\left(\frac{c \log_b n}{b-1}\right)\left(1 - \frac{1}{(b-1)\log_b n}\right)^{c \log_b^2 n - 1}$ which is $\Theta(\log_b n/n^\beta)$. Thus, the probability that p_{i+1} initiates at least 2 edges to $\mathcal{F}_i$ is $1 - \gamma \log_b n/n^\beta$ for a constant $\gamma = c/(b-1)$. □