Multi-Stage Complex Contagions in Random Multiplex Networks

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***Abstract*—Complex contagion models have been developed to understand a wide range of social phenomena such as adoption of cultural fads, the diffusion of belief, norms, and innovations in social networks, and the rise of collective action to join a riot. Most existing works focus on contagions where individuals' states are represented by *binary* variables, and propagation takes place over a single isolated network. However, characterization of an individual's standing on a given matter as a binary state might be overly simplistic as most of our opinions, feelings, and perceptions vary over more than two states. Also, most real-world contagions take place over multiple networks (e.g., Twitter and Facebook) or involve *multiplex* networks where individuals engage in different *types* of relationships (e.g., co-worker, family, etc.). To this end, this paper studies *multi-stage* complex contagions that take place over multi-layer or multiplex networks. Under a linear threshold based contagion model, we first give analytic results for the expected size of *global* cascades, i.e., cases where a randomly chosen node can initiate a propagation that eventually reaches a *positive* fraction of the whole population. Then, analytic results are confirmed by an extensive numerical study. In addition, we demonstrate how the dynamics of complex contagions is affected by the structural properties of the networks. In particular, we reveal an interesting connection between the assortativity of a network and the impact of *hyper-active* nodes on the cascade size.**

I. INTRODUCTION

Modeling and analysis of dynamical processes in complex networks has been a very active research field in the past decade. This has led to many advances in our understanding and ability to control a wide range of physical and social phenomena. Examples include adoption of cultural fads, the diffusion of beliefs, norms, and innovations in social networks [1], [2], disease contagion in human and animal populations [3], cascading failures in *interdependent* infrastructures [4], *insolvency* and *default* cascades in financial networks [5], and the spread of computer viruses or worms on the Web.

In this work, we focus on *complex* contagions, a class of dynamical processes typically used in modeling the propagation of *influence* in social networks. In particular, complex contagion models are used when *social reinforcement* plays an important role in the propagation process, i.e., when *multiple* sources of exposure is needed for an individual to adopt an activity. Examples include the spread of social movements and radical behavior, the rise of collective action to join a riot, or the decision to support one political candidate versus the other. This differs from the class of models known as *simple* contagions, where propagation often takes place after only a single copy is received; e.g., spread of diseases, viruses, etc.

Complex contagions have typically been studied in the literature using a *linear threshold* model. The original threshold model, proposed by Watts [1], considers binary-state dynamics where each node is in one of the two states, *inactive* or *active*, and is initially assigned a threshold τ in $(0, 1]$. At any point in time, if an *inactive* node has d neighbors of which m are *active*, we determine if it will be activated by checking the relationship between $\frac{m}{d}$ and the pre-assigned threshold τ. If $\frac{m}{d} \geq \tau$, then the node will turn *active*. Otherwise, if $\frac{m}{d} < \tau$, it stays *inactive*. Once a node is activated, it is assumed to remain active forever.

In the Watts threshold model, there are two important assumptions. First assumption is that all active individuals exhibit the same amount of influence on their neighbors. However, individuals' standings on a given matter could vary significantly. For example, followers of a radical organization or a revolutionary movement may have varying levels of commitment to the cause, or varying desire and ability to recruit new members. To cope with the multi-state nature of individuals activity levels, Melnik et al. [6] introduced a *multi-stage* contagion model as a generalization of the Watts threshold model. There, nodes can be inactive or be in one of several active states with different levels of influence; e.g., active, hyper-active, etc.

The second assumption of the Watts model, which is also used in the multi-stage model by Melnik et al. [6], is that contagion is taking place over a *single* network where all edges have the same impact on spreading the influence. However, most real-world influence propagation events take place over multiple networks. For example, individuals may participate in multiple online social networks (e.g., Facebook, Twitter, etc.), and may have different levels of influence in each network. Similarly, within a single network, individuals may form different types of relationships (e.g., friendship, colleagueship, etc.), and each relationship type might have a different impact on the propagation of influence in a given context. For example, video games might be more likely to spread among high-school friends rather than parents, while the opposite might be true for political ideas. That is, if we do not distinguish different types of relationships, dynamics of influence propagation may not be accurately captured. Hence,

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it is natural to consider complex contagions over multiplex networks. With this motivation, [2] proposed and studied a threshold model in *multiplex* networks. However, [2] still used the first assumption mentioned above in the sense that their model is not suitable for multi-stage contagions (where nodes can belong to a rich set of states).

In this paper, we drop both of the aforementioned assumptions and study for the first time a multi-stage contagion model on multiplex networks. For simplicity, we assume that the network consists of two types of links, red and blue, and individuals can be in one of three possible states, inactive, active, and hyper-active. Then, we seek to answer several important questions: *In the cases where a global spreading event is possible, could we give analytic answers to the final expected cascade size? Under the newly proposed model, how do topological properties affect the cascade process?*

Our contributions towards answering these questions are summarized as follows:

- For a class of random networks generated by the *colored* configuration model (see Section II-A), we analytically derive the expected size of *global* cascades; i.e., cases where a positive fraction of nodes (in the asymptotic limit) eventually becomes active or hyper-active when a randomly selected node is switched to the active state.
- We explore the intricate relationships between the structural properties of the underlying network and the impact of *hyper-active* nodes on the contagion dynamics. For instance, a particularly interesting scenario is when the hyper-active state is manifested in only one link type. This is motivated by the case where people may be more willing to express their viewpoints to close friends instead of office-mates, or can reach a hyper-influential state only in one social network (e.g., Twitter) versus another (e.g., Facebook). Our main finding is the interesting connection between the assortativity (i.e., the correlation among the degree of neighboring nodes) of the network and the impact of hyper-active nodes on cascade size. For instance, when the network is highly assortative (i.e., when high-degree nodes are likely to be connected with high-degree nodes), the influence exerted by the hyper-active nodes has a much more significant impact on the cascade size as opposed to the case when the network has low assortativity (i.e., when the degrees of neighboring nodes tend to be uncorrelated). This impact is best observed when the cascade size is plotted as a function of the mean degree of the network. There, as the influence of hyper-active nodes increases, the highly assortative networks are shown to exhibit changes on not only the critical transition points (i.e., mean degree values at which expected cascade size changes from zero to a positive value, or vice verse), but also the number and *order* of transitions.

The rest of the paper is organized as follows. In Section II, we introduce the network and contagion models. Then, we describe the problem of interest and our main results in Section III. In Section IV, we present numerical results that demonstrate the accuracy of our analysis in the finite node regime, and discuss the impact of hyper-influencers on complex contagions under different levels of assortativity. We conclude the paper in Section V.

II. Model Definition: Networks and Dynamics

A. Multi-layer and multiplex network models

A multiplex network is a network model where links are classified into different types (or, colors), which can capture the different types of connections between nodes in networks. For convenience, in the following discussion, we focus on a multiplex network with two types of links, *red* and *blue*, but the model and results can be easily extended to an arbitrary number of link types. These two link types can be motivated by the case where one color accounts for edges in Facebook while the other for edges in Instagram. Alternatively, one link color may be representing close friendship links while the other representing "acquaintances" in a social network. In this network model, we let $\mathcal{N} = \{1, 2, \ldots, n\}$ denote the vertex set, with n standing for the number of nodes. We let $\mathcal{N}_r \subset \mathcal{N}$ denote the set of vertices that have *red* edges and $\mathcal{N}_b \subset \mathcal{N}$ denote the set of vertices having *blue* edges. For simplicity, we assume $\mathcal{N}_b = \mathcal{N}$, which means all vertices in the network may have blue edges. To model the possibility that not everyone may have red links, we assume that each vertex in $\mathcal{N}$ has red links with probability $\alpha \in (0, 1]$:

$$\mathbb{P}[i \in \mathcal{N}_r] = \alpha, \quad i = 1, \ldots, n. \tag{1}$$

With this assumption, by the law of large numbers, we can easily conclude that $\frac{|\mathcal{N}_r|}{n} \xrightarrow{\text{a.s.}} \alpha$, where $|\mathcal{N}_r|$ denotes the cardinality of $\mathcal{N}_r$ and $\xrightarrow{\text{a.s.}}$ indicates almost sure convergence.

This network model can be interpreted in two different ways. The first one is a multi-layer network where each network layer is generated by the widely used configuration model [7]–[9]. In particular, we use $P(d_r)$ (resp. $P(d_b)$) as the *degree distribution* to determine the number of red (resp. blue) edges that will be assigned to each node in $\mathcal{N}_r$ (resp. $\mathcal{N}_b$). Once the degree of each node is determined, we generate the networks $\mathbb{R}$ and $\mathbb{B}$ by selecting a graph uniformly at random from among all possible graphs that have the same degree sequence; see [7], [8] for more details. Next, we take a union of the edges in $\mathbb{R}$ and $\mathbb{B}$ to create a network $\mathbb{H}$. Equivalently, we can consider a multiplex network model generated by the *colored* configuration model [10]. Let $\mathbf{d} = (d_r, d_b)$ denote the colored degree of a node, where d_r and d_b stand for the number of red edges and blue edges incident on it. Each of the n nodes in the network is assigned a colored degree by independently drawing from the distribution $P_{\mathbf{d}}$. Then, pairs of edges of the same color are randomly chosen and connected together until none is left; see [10] for details.

B. Multi-stage content-dependent linear threshold model for complex contagions

We first introduce the single-stage content-dependent linear threshold model [2] which is a generalization of the vanilla

threshold model [1]. In the content-dependent linear threshold model, links are classified into r types. For a given content (a rumor, product, etc.), scalars c_i, $i = 1, \ldots, r$ represent the weight of type-i edges on spreading this particular content. Nodes belong to either one of the two states, active or inactive, and each node is assigned a threshold τ in $(0, 1]$ drawn from a distribution $P(\tau)$. Given an inactive node with m_i active and $d_i - m_i$ inactive neighbors for each link type-i, $i = 1, \ldots, r$, an inactive node will turn active if $\frac{\sum_i c_i m_i}{\sum_i c_i d_i} \geq \tau$. Namely, an inactive node with $\mathbf{m} = (m_1, \ldots, m_r)$ and $\mathbf{d} = (d_1, \ldots, d_r)$ will turn *active* with probability

$$F[\mathbf{m}, \mathbf{d}] \triangleq \mathbb{P}\left[\frac{\sum_{i=1}^{r} c_i m_i}{\sum_{i=1}^{r} c_i d_i} \geq \tau\right]. \tag{2}$$

Throughout, $F[\mathbf{m}, \mathbf{d}]$ is referred to as the response function. If we do not distinguish the edge types or simply set $c_i = 1$ for all $i = 1, \ldots, r$, then this model reduces to the Watts' threshold model [1]. The content-dependent threshold model enables modeling the case where people's influence on others vary according to their relationship type, or the social network that they are interacting through.

Different from the single-stage threshold model where nodes can be only in two states, the multi-stage linear threshold model [6] allows nodes to be in a richer set of active states. In this work, we assume that nodes can belong to three states, inactive, active, and hyper-active. In the following discussion, we use state-0, state-1, and state-2 to represent the inactive, active, and hyper-active state, respectively. Let τ_1 and τ_2 denote the thresholds associated with transitioning to the active and hyper-active states, respectively. The hyper-active individuals are assumed to be β-times more influential than active nodes in the propagation process (where $\beta \geq 1$). For example, given an individual with d neighbors of which m_1 are active and m_2 are hyper-active, the probability of switching to state-i from the inactive state (i.e., state-0) is given by:

$$F_i[\mathbf{m}, d] \triangleq \mathbb{P}\left[\tau_i \leq \frac{m_1 + \beta m_2}{d} \leq \tau_{i+1}\right], \ i = 0, 1, 2, \tag{3}$$

where $\mathbf{m} = (m_1, m_2)$, $\tau_0 = 0$, $\tau_3 = \infty$, and $\beta \geq 1$. Although we assume there are three states in the contagion process, our analysis can be extended to an arbitrary number of states.

Finally, we introduce the multi-stage content-dependent linear threshold model. In the content-dependent linear threshold model, links are classified into r types. For a given content (a view, rumor, product, etc.), there are r scalars c_i, $i = 1, \ldots, r$ represent the weight (i.e., relative importance) of type-i edges on spreading this particular content. Assume that there are two types of links in the network, red and blue, and that nodes can be in three states, inactive, active, and hyper-active. We let c_r and c_b denote the weight of red and blue edges, respectively, and set $c = \frac{c_r}{c_b}$. With this notation, the probability of an inactive node switching to state-i is given by:

$$F_i[\mathbf{m}, \mathbf{d}] \tag{4}$$
$$\triangleq \mathbb{P}\left[\tau_i \leq \frac{c(m_{r,1} + \beta m_{r,2}) + m_{b,1} + \beta m_{b,2}}{c d_r + d_b} \leq \tau_{i+1}\right],$$

where $\mathbf{m} = (m_{r,1}, m_{r,2}, m_{b,1}, m_{b,2})$, $\mathbf{d} = (d_r, d_b)$, $m_{r,1}$ and $m_{r,2}$ (resp. $m_{b,1}$ and $m_{b,2}$) denote the number of active and hyper-active neighbors connected through a red (resp. blue) edge, and d_r and d_b denote the number of red and blue neighbors, respectively.

III. Main Results

Assume that all nodes are initially inactive and the contagion process starts by randomly choosing a node and setting it as active. The influence might then propagate in the network according to (4) and other nodes might turn active, and so on. Since the contagion process is monotone (i.e., an active node can never switch back to inactive), it will eventually stop, i.e., a steady-state will be reached. A *global* cascade is said to take place if the fraction of nodes that are activated is *positive* in the limit of large network sizes. Our main goal is calculating the expected size of global cascades when they are possible.

We start the analysis with computing the expected size of global cascades when they occur. Consider a random variable S defined as

$$S \triangleq \frac{\text{\# of active and hyper-active nodes at steady-state}}{n},$$

where n is the number of nodes in the network. Then, a *global* cascade is said to take place if $S > 0$ in the limit $n \to \infty$, and our main goal is to derive

$$\lim_{n \to \infty} \mathbb{E}\left[S \mid S > 0\right],$$

which gives the expected size of global cascades when they exist. For simplicity, in our analysis we omit *self-loops*, i.e., the possibility of having more than one edge between two nodes. It is a simple matter to show that such self-loops occur very rarely in the construction of the configuration model and they have negligible impact on the cascade dynamics; e.g., see [11]. In fact, our experiments also confirm that the impact of this omission is negligible.

According to our definition, the expected cascade size stands for the final fraction of active and hyper-active individuals in the network. Therefore, we can compute it by computing the probability that an arbitrary node is active or hyper-active at the steady-state. We will compute this probability recursively using the "tree-approximation" approach [2], [6], which is a mean-field treatment of the zero-temperature random-field Ising model on Bethe lattices [12]. The tree-approximation approach assumes that the network has a locally tree-like structure, which is valid under the configuration model considered here [7]. Labeling the tree structure from the bottom to the top, it is assumed that the node states are updated starting from the bottom, and continuing to the top, one level at a time. In other words, the nodes at level ℓ will not update their states until the nodes at levels $0, 1, \ldots, \ell - 1$ have finished updating. We define $q_{r,1,\ell}$ (resp. $q_{b,1,\ell}$) as the probability that a node at level ℓ who is connected to its only parent at level $\ell + 1$ by a red (resp. blue) edge turns active. Similarly, we define $q_{r,2,\ell}$ (resp. $q_{b,2,\ell}$) as the probability that a node at level ℓ that is attached to its only parent via a red (resp. blue) edge turns hyper-active.

Given our assumption that nodes in the tree update their states one level at a time, these probabilities will be computed under the condition that the parent nodes at level $\ell+1$ are inactive.

In the interest of brevity, we only explain the derivation of $q_{r,1,\ell+1}$ in details. The derivations of $q_{r,2,\ell+1}$, $q_{b,1,\ell+1}$, and $q_{b,2,\ell+1}$ can be explained very similarly. Since $q_{r,1,\ell+1}$ cannot be expressed explicitly, we derive a recursive relation in terms of $q_{r,1,\ell}$, $q_{r,2,\ell}$, $q_{b,1,\ell}$, and $q_{b,2,\ell}$; see (6) - (9). The validity of the expression (6) for $q_{r,1,\ell+1}$ can be explained as follows. Consider an inactive node at level $\ell+1$ with colored degree $\mathbf{d}=(d_r,d_b)$ that is connected to its unique parent at level $\ell+2$ via a red edge. The probability that this node has i active children connected via red edges, s active children connected via blue edges, j hyper-active children connected via red edges, and t hyper-active children connected via blue edges, *and* that it turns active is given by

$$\begin{aligned}&\binom{d_r-1}{i}\binom{d_r-1-i}{j}q_{r,1,\ell}^i q_{r,2,\ell}^j(1-q_{r,1,\ell}-q_{r,2,\ell})^{d_r-1-i-j}\\&\times\binom{d_b}{s}\binom{d_b-s}{t}q_{b,1,\ell}^s q_{b,2,\ell}^t(1-q_{b,1,\ell}-q_{b,2,\ell})^{d_b-s-t}\\&\times F_1\left[(i,j,s,t),\mathbf{d}\right],\end{aligned}\tag{5}$$

where $F_1\left[(i,j,s,t),\mathbf{d}\right]$ is as defined in (4); i.e., it denotes the probability that an inactive node with a colored degree $\mathbf{d}$ and a group of active and hyper-active neighbors for each color represented by $\mathbf{m}=(i,j,s,t)$ switches to state-1. To simplify the notation, we use $\mathbb{F}_1\left[(i,j,s,t),(x,y)\right]$ as defined at (10), so the term given in (5) becomes equivalent to $\mathbb{F}_1\left[(i,j,s,t),(d_r-1,d_b),\ell\right]$.

The intuition behind (5) is as follows. Since we assume that the network is tree-like, the state of each child node at level ℓ is independent from other children at the same level. Thus, we multiply together the probability of being at a specific state for each child node to get the whole expression (5) using a simple combinatorial argument. The reason behind using d_r-1 rather than d_r in (5) is the fact that the node under consideration is attached to its unique parent at level $\ell+2$ through a *red* edge, and by assumption this parent node is inactive; recall that a node at level $\ell+2$ can not update its state until all nodes in level $\ell+1$ finish updating. A node that is known to have at least one red edge can be seen to have colored *degree* $\mathbf{d}=(d_r,d_b)$ with probability $\frac{d_r p_{\mathbf{d}}}{\langle d_r\rangle}$; e.g., see [2], [7] for a discussion on the *excess* degree distribution. Finally, we get the detailed expressions of $q_{r,1,\ell+1}$ (6) after taking the expectation of (5) over the degree of the node at level $\ell+1$. We can use similar arguments to derive expressions for $q_{r,2,\ell+1}$, $q_{b,1,\ell+1}$, and $q_{b,2,\ell+1}$. The expressions of all four probabilities are shown in (6) - (9).

Equations (6) - (9) form a non-linear system. Since our goal is to compute the expected size of global cascades *given that they exist*, we can initialize this dynamical system with $q_{r,1,0},q_{r,2,0},q_{b,1,0},q_{b,2,0}>0$ to obtain the steady-state values (i.e., fixed points), $q_{r,1,\infty}$, $q_{r,2,\infty}$, $q_{b,1,\infty}$, and $q_{b,2,\infty}$. These fixed points account for the probability of being in a corresponding state for the children of the node chosen uniformly at random. We can use them to calculate the expected size of global cascades.

$$\begin{aligned}q_{r,1,\ell+1}=&\sum_{\mathbf{d}}\frac{d_r p_{\mathbf{d}}}{\langle d_r\rangle}\sum_{i=0}^{d_r-1}\sum_{j=0}^{d_r-1-i}\sum_{s=0}^{d_b}\sum_{t=0}^{d_b-s}\\&\mathbb{F}_1\left[(i,j,s,t),(d_r-1,d_b),\ell\right]\end{aligned}\tag{6}$$

$$\begin{aligned}q_{r,2,\ell+1}=&\sum_{\mathbf{d}}\frac{d_r p_{\mathbf{d}}}{\langle d_r\rangle}\sum_{i=0}^{d_r-1}\sum_{j=0}^{d_r-1-i}\sum_{s=0}^{d_b}\sum_{t=0}^{d_b-s}\\&\mathbb{F}_2\left[(i,j,s,t),(d_r-1,d_b),\ell\right]\end{aligned}\tag{7}$$

$$\begin{aligned}q_{b,1,\ell+1}=&\sum_{\mathbf{d}}\frac{d_b p_{\mathbf{d}}}{\langle d_b\rangle}\sum_{i=0}^{d_r}\sum_{j=0}^{d_r-i}\sum_{s=0}^{d_b-1}\sum_{t=0}^{d_b-1-s}\\&\mathbb{F}_1\left[(i,j,s,t),(d_r,d_b-1),\ell\right]\end{aligned}\tag{8}$$

$$\begin{aligned}q_{b,2,\ell+1}=&\sum_{\mathbf{d}}\frac{d_b p_{\mathbf{d}}}{\langle d_b\rangle}\sum_{i=0}^{d_r}\sum_{j=0}^{d_r-i}\sum_{s=0}^{d_b-1}\sum_{t=0}^{d_b-1-s}\\&\mathbb{F}_2\left[(i,j,s,t),(d_r,d_b-1),\ell\right],\end{aligned}\tag{9}$$

where for $k=1,2$, we define

$$\begin{aligned}\mathbb{F}_k\left[(i,j,s,t),(x,y),\ell\right]=&F_k\left[(i,j,s,t),(x,y)\right]\\&\times\binom{x}{i}\binom{x-i}{j}q_{r,1,\ell}^i q_{r,2,\ell}^j(1-q_{r,1,\ell}-q_{r,2,\ell})^{x-i-j}\\&\times\binom{y}{s}\binom{y-s}{t}q_{b,1,\ell}^s q_{b,2,\ell}^t(1-q_{b,1,\ell}-q_{b,2,\ell})^{y-s-t}.\end{aligned}\tag{10}$$

$$\begin{aligned}\lim_{n\to\infty}\mathbb{E}\left[S\mid S>0\right]=&\sum_{\mathbf{d}}p_{\mathbf{d}}\sum_{i=0}^{d_r}\sum_{j=0}^{d_r-i}\sum_{s=0}^{d_b}\sum_{t=0}^{d_b-s}\\&\left\{\mathbb{F}_1\left[(i,j,s,t),(d_r,d_b),\infty\right]+\mathbb{F}_2\left[(i,j,s,t),(d_r,d_b),\infty\right]\right\}.\end{aligned}\tag{11}$$

We give the expected size of the cascades (given that they exist) in (11). The validity of (11) can be seen as follows: First, we randomly choose a node, whose colored degree is $\mathbf{d}=(d_r,d_b)$, with probability $p_{\mathbf{d}}$. The probability that each of its d_r neighbors (via red links) is active (resp. hyper-active) is given by $q_{r,1,\infty}$ (resp. $q_{r,2,\infty}$). Similarly, each of the d_b neighbors (connected via blue links) of this randomly chosen node is active with probability $q_{b,1,\infty}$ and hyper-active with probability $q_{b,2,\infty}$, independently from each other. Then, with each possible combination of numbers of active and hyper-active neighbors, we can calculate the probability of being active or hyper-active for the node by the response function (4). Taking the expectation with respect to the degree $\mathbf{d}$ yields (11). As discussed in details in [2], [6], this method, based on the *tree-approximation* technique, gives precise results in the asymptotic limit $n\to\infty$, when the underlying network is generated according to the configuration model. We present extensive numerical studies in Section IV that supports our results even in the finite node regime.

IV. Numerical Results

In this section, we first present numerical results to support our analysis on the expected size of global cascades. We are particularly interested in checking the accuracy of our asymptotic results when the number of nodes is finite. Due to the page limit, we include the results in supplementary materials. Supplementary materials are at https://bit.ly/2vtD2aD.

Next, we will investigate the impact of hyper-influencers (i.e., the additional influence exerted by them) on the contagion dynamics. In particular, we aim at exploring if structural properties of networks will change the impact of hyper-influencers. We consider a case where hyper-active nodes are restricted to appear only through one type of edges, red or blue, rather than allowing them to exert additional influence through both types of edges. This setting is motivated by cases where people can reach a more active/influential state only in one network, or one relationship type. For example, some people may be reluctant to express their opinions freely in person (e.g., physical networks), but may be much more active on online networks (e.g., Twitter) due to anonymity. This raises an interesting question: which network or edge type would facilitate the influence propagation process most when hyper-influencers are allowed there. In what follows, we conduct several experiments to answer this question: 1) we only allow hyper-activity in red edges, i.e., hyper-active neighbors connected by blue edges will be counted as merely active when checking the response function; 2) we only allow hyper-activity in blue edges. As discussed in some previous studies [13], [14], assortativity is one of the most important structural properties on multiplex networks. Assortativity is defined as the Pearson correlation coefficient between the degree of nodes that are connected by a link [15]. If a network is assortative, then nodes of high degree in the network tend to attach to high degree nodes; it was noted in [15] that social networks tend to have high assortativity. Therefore, it is interesting to see if assortativity has any impact on the answer to the above question.

In the following experiments, we conduct these experiments on a network with *low* assortativity and then a network with *high* assortativity We use the degree distributions (12) and (13) to assign red and blue degrees. Namely, with p_k^r (resp. p_k^b) denoting the probability that a node is assigned k red (resp. blue) edges, we let

$$p_k^b = e^{-\lambda_b}\frac{(\lambda_b)^k}{k!}, \quad \text{k = 0, 1, } \ldots, \tag{12}$$

$$p_k^r = \alpha e^{-\lambda_r}\frac{(\lambda_r)^k}{k!} + (1-\alpha)\delta_{k,0}, \quad \text{k = 0, 1, } \ldots. \tag{13}$$

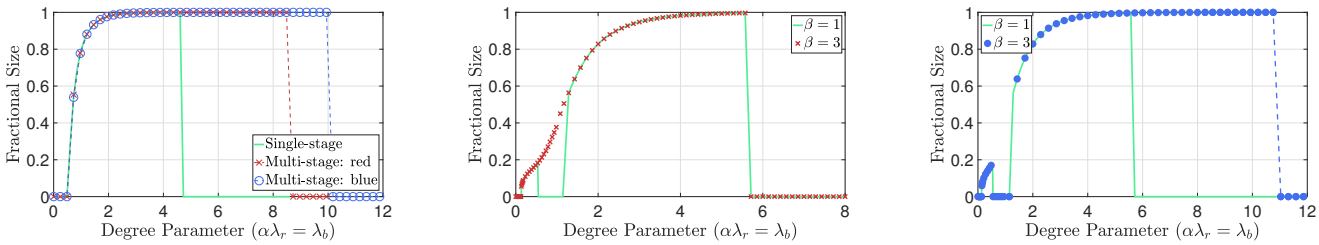
Here, λ_r (resp. λ_b) denotes the mean number of red (resp. blue) edges assigned per node, α denotes the fraction of nodes that have red edges (i.e., the relative size of the *red* network $\mathbb{R}$), and δ denotes the Kronecker delta.

To be able to control the assortativity of networks without changing the first moment of degree, we set $\alpha\lambda_r = \lambda_b$ rather than $\lambda_r = \lambda_b$. With this setting, when α is large, e.g., 0.99, nearly all of the nodes will have a similar number of red and blue edges, which leads to networks with limited assortativity. On the contrary, when α is *low*, e.g., 0.1, only 10% of the nodes will have extra red edges. In addition, these nodes will have a significantly larger number of edges, since λ_r is ten times larger than λ_b. The nodes with extra red edges will tend to be connected together, which results in the network to have *high* assortativity. See [14] for a more detailed discussion.

We start with the limited assortativity case, i.e., $\alpha = 0.99$. As shown in Figure 1(a), we observe that regardless of which network hyper-influencers are constrained to exist, there are two phase transitions as in the case of single-stage complex contagions. However, we see that the existence of hyper-influencers delays the second phase transitions to higher mean degrees. The reason behind this delay can be explained as follows. As mentioned before, the second phase transition occurs due to *high* local stability of nodes making their states hard to change by only few active neighbors. However, hyper-influencers help increase the value of the perceived influence, i.e., $\frac{c(m_{r,1}+\beta m_{r,2})+m_{b,1}+\beta m_{b,2}}{cd_r+d_b}$, so that the response function could be exceeded even with few active and hyper-active neighbors, in the high mean degree region. Besides, allowing hyper-activity in blue edges leads to a larger region where global cascades take place, in comparison with the case where hyper-activity exists only in red edges. This can be explained as follows. When $\alpha = 0.99$, there are more nodes connected by blue edges in the network than red edges. That is, the impact of blue edges on impeding global cascades is more than that of red edges. Thus, allowing hyper-influence to be exerted in blue edges delays the second phase transition further.

Next, we discuss the case where $\alpha = 0.1$ that leads to a highly assortative network [14]. In Figure 1(b), we present numerical results for the first setting where the hyper-active state is manifested in only red edges. When $\beta = 1$, i.e., when there are no hyper-influencers in the network, four phase transitions take place. However, if we increase β from one to three, then only two phase transitions are observed. This can be explained as follows. When $\beta = 1$, multi-stage complex contagions is reduced to single-stage complex contagions, in which case four phase transitions might occur when assortativity is high [14]. As explained in [14], the first pair of phase transitions are mainly due to the red edges. When λ_b is *small*, there are too few blue edges to trigger a global cascade. However, since we have $\lambda_r = 10\lambda_b$, there are still enough red edges to have global cascades. As we increase λ_b, we observe a parameter interval where red edges are too many while blue edges are too few to have a global cascade. If we keep increasing λ_b further, global cascades start appearing again when the network has enough connectivity in blue edges to propagate the influence. However, further increasing in λ_b leads to high local stability of nodes w.r.t. both blue and red edges and global cascades become impossible again. A more detailed discussion can be found in [14].

The reason why increasing β changes the number of phase transitions is as follows. From the definition (4) of the response function, we observe that it is monotonically increasing with respect to β. Thus, when β is higher, an inactive node is easier



(a) (b) (c)

Fig. 1. *We fix $\tau_1 = 0.18$ and $\tau_2 = 0.32$, then vary the mean degree. (a) Hyper-activity only appears in either red or blue edges. When $\alpha = 0.99$, the assortativity is negligible. (b) Hyper-activity only appears in red edges. When $\alpha = 0.1$, the assortativity of the network is around 0.8. (c) Hyper-activity only appears in blue edges. When $\alpha = 0.1$, the assortativity is high (be up to 0.8).*

to be activated by a hyper-active node, which makes it possible to have global cascades at higher levels of connectivity; i.e., the second phase transition tends to appear at larger λ. This leads to the second and the third phase transitions seen in Figure 1(c) disappear when $\beta = 1$; i.e., the interval where we have too many red and too few blue edges disappears.

Next, we focus on the second setting where hyper-activity is only manifested in blue edges. The results are shown in Figure 1(c). Allowing hyper-activity in blue edges does not change the connectivity of the network, so the first and the second phase transitions caused by the connectivity w.r.t. red edges remain the same. However, the gap between the second and third transitions still exists, which results from the second transition w.r.t. red and the first transition w.r.t. blue edges. A high β only shifts the second transition to the right but does not affect the first transition much. Thus, the gap disappears quickly with increasing β when we allow it in red edges, but remains when we only allow it in blue edges. Besides, compared with the case $\beta = 1$, the fourth transition is significantly delayed when $\beta = 3$. The reason behind the delay of the fourth phase transition is similar to the previous discussion: A higher β makes it easier to exceed the threshold even when the degree parameter is at a high level, so the original fourth phase transition has been extended to a larger mean degree.

From these experiments, we conclude that depending on the assortativity of the network, the impact of hyper-activity in red or blue edges on complex contagions are different: when the network is highly assortative, the additional influence exerted by the hyper-active nodes may change not only the critical transition points, but also the number and order of phase transitions, while for networks that have little or no assortativity, the additional influence mainly enlarge global cascade regions.

and supported by a numerical study. An interesting finding is that depending on the assortativity of the network, the existence of hyper-influencers affect the expected size of global cascades differently. For instance, when the network is highly assortative, the additional influence exerted by the hyper-active nodes may change not only the critical transition points, but also the number and order of phase transitions; while the effect is much more limited in networks with low assortativity.

V. Conclusion and Future Work

In this work, we study the propagation of influence in multiplex networks under a *multi-stage* complex contagion model. We derive recursive relations characterizing the dynamics of influence propagation to compute the expected size of *global* cascades, i.e., cases where a single individual can initiate a propagation that eventually influences a positive fraction of the population. The analytic results are also confirmed

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