Co-Contagion Diffusion on Multilayer Networks

Ho-Chun Herbert Chang\textsuperscript{1,2*} and Feng Fu\textsuperscript{2}

Abstract
We study the interface of three elements of diffusion: the synergy between contagions, the dormancy rate of each individual contagion, and the multiplex network topology. Dormancy is defined as a weaker form of "immunity," where dormant nodes no longer actively participate in diffusion, but are still susceptible to infection. The proposed model extends the literature on threshold models, which we use to show the intricate interdependencies between different graph structures. Our comprehensive simulations show that first, the faster diffusion contagion induces branching on the slower diffusion contagion; second, there exists a positive relationship between network radius and dormancy in lowering dormancy; third, when two long-range graphs are paired, the faster diffusing contagion depends on both dormancy rates, whereas the slower contagion depends on its own; lastly, synergistic contagions are less sensitive to dormancy, and have a wider window to diffuse. Furthermore, when long-range and spatially constrained graphs are paired, ring vaccination occurs on the spatial graph, which describes partial diffusion due to dormant, surrounding nodes. The spatial contagion depends on both dormancy rates whereas the long-range contagion depends on only its own.

Keywords: complex contagions; network diffusion; stochastic modeling

1 Introduction
From misinformation to health interventions, product diffusion to technological uptake, interconnected domains increasingly demand sophisticated models to understand their diffusion phenomenon. Contemporary research in social contagions diffusion follows three directions: an ecology of contagions, the mechanisms of diffusion, and population structures [1]. This paper addresses the first two, where we examine the simultaneous diffusion of two contagions on different pairs of multiplex networks. Specifically, we study the interaction of synergy and dormancy in multi-layer contagion diffusion.

Synergy and dormancy are naturally opposing forces but their interaction is not as simple as additive cancellation. For instance, synergistic contagions should diffuse faster together, since greater density implies greater infection probability. However, if one diffuses much faster than other contagions and introduces immunity or dormancy within the population, then it effectively "vaccinates" the population against subsequent contagion and produces percolation.

Prior results show probabilistic branching and percolation based on synergy and dormancy ratios [2]. While insightful from a complex network perspective, prior analysis only encapsulates lattice and regular-random-graphs, thus limits direct application to real social systems today. This paper aims to bridge that gap—we generalize diffusion behavior based on common graph structures, and demonstrate
case-specific phenomenon based on network properties such as shortest path, clustering, and degree distribution.

Co-infection is a notion in epidemiology that describes multiple contagions interfering with each other, and can simultaneously infect a host [3]. Thus, co-infection can be extended to model social contagions as, like infectious diseases, behaviors do not spread in isolation.

In the domain of epidemiology, Cai et al. have proposed a co-evolutionary spreading model whose dynamics depend on the SIR model[4]. They determine the conditions that induce phase transitions on nodes belonging to a giant component. Grassberger et al. further gives a thorough review of network topology influencing phase transitions [5]. They note the importance of long-range dependencies on producing discontinuous phase transitions. Hebert-Dufresne and Althouse show on clustered networks, synergistic diffusion behaves differently than equivalent random networks. This suggests that clustering bolsters, rather than percolates diffusion in comparing the synergistic and single contagion case [6]. These studies have been largely constrained to single layer networks. For the case of multiplex networks, Azimi-Tafreshi used general percolation theory to compute the fraction of nodes that are infected at equilibrium. The author describes multiplex networks using joint-degree distribution, then, given the overlapping edges, computes the final state and shows the emergence of a tri-critical point [7]. Synergy in this field has been defined many ways, for instance, dynamically inferred from the neighborhood of susceptible-infected pairs [8].

While co-diffusion has its roots in epidemiology, contemporary systems benefit greatly from contagion interaction models. Multilayer models are can be also better understood in the context of infrastructural, institutional, or functional divisions. Examples include the internet and power grids [9], transportation systems [10], and information on different social media platforms [11]. Models have been developed to encapsulate a fraction of all nodes, with respect to real networks.

For social contagions, prior research has been divided between successive and simultaneous contagions; this research focuses on the latter. A common paradigm has been through evolutionary games, which not only provides a vocabulary for characterizing cooperation versus competition, but generalizes well across different mechanisms and domains [12] [13]. For instance, Jiang [14] models information diffusion as a game on social networks, where mutations are interpreted as new information. Teaching activity and information sharing has been modeled similarly by Szolnoki [15] [16], with concentrated efforts are being directed towards multilayer networks [14] [17] [18]. In particular, research has shown the importance of topological features, such as collective influence by degree [19], stochasticity and noise [20], and strategy/topology coevolution [21]. Shu et al. [22] study contagions on two interdependent lattices

More closely related to our work are complex contagion threshold models. Zarazade et al. have discovered diffusion synergy between correlated platforms, such as Youtube and Google Play when a new album is released [23]. On the other hand, they note URL sharing is competitive. The focus of theoretical models have been diverse, from a pair of simple and complex layers [24], and trusted and distrusted edges between layers [25]. These studies focus on competing contagions,
though recently there has been directed research effort toward synergy. Liu et. al consider diffusion of two contagions constrained to two layers [26] [27], considering diffusion density of other contagions as synergy. Chang and Fu build on this model by quantifying the different types of synergy using a formulation similar to Loewe Additivity, and introduce the effects of dormancy on diffusion behavior [2].

2 Model and Methods

Suppose we have two contagions, Contagion A and Contagion B. Associated with the two contagions are two networks who share nodes, but different edges—a multiplex network. While the diffusion of each contagion is constrained to its own network layer, the "infection" probabilities depend on both contagions.

Each node on can attain four possible states: naïve (Θ), A, B, and AB, which denotes co-infection. Additionally, each node is either active or dormant, represented by a binary variable. Thus, each node $i$ is represented by the tuple (State, Activity). Each node is also assigned a threshold value within (0, 1), aligned with the threshold model. The probability of diffusion is described by the multivariate Hill function [2], which governs canonical logistic growth, whose concavity determines whether the additivity between contagions is synergistic or antagonistic.

This model draws influence from pharmacology [28], where researchers consider the efficacy of drugs when they are used in conjunction. Drugs interact synergistically if they yield better results together, or antagonistically if reduced efficacy is observed. This parametrization of density-dependent performance can be extended naturally to the diffusion of complex contagions. Equation 1 gives the general form of the adoption function.

$$P(i \leftarrow A \text{ or } B) = \frac{(1 - S_A(i)) \left( \frac{|A|}{K_A} \right)^\alpha + (1 - S_B(i)) \left( \frac{|B|}{K_B} \right)^\alpha}{1 + (1 - S_A(i)) \left( \frac{|A|}{K_A} \right)^\alpha + (1 - S_B(i)) \left( \frac{|B|}{K_B} \right)^\alpha}$$

(1)

The $K_j$’s denote the attractiveness of given contagion $j$, $|A|$ denotes the density of neighbor nodes with status $A$, and the $S_i$’s are indicator functions that returns 1 if the infection value is true. This reduce the diffusion probability to uni-variate sub-cases within canonical logistic diffusion, for instance with $S_A(i) = 1$, the terms containing contagion $A$ drop away. The $k_i$’s can be parametrized to reflect the canonical linear threshold model if set to 1. The left arrow denotes node $i$ adopting $A$ or $B$, and the choice between the two is settled by a coin-toss, weighed by their relative densities in Equation 2.

$$P(i \leftarrow A) = \frac{\left( \frac{|A|}{K_A} \right)^\alpha}{ \left( \frac{|A|}{K_A} \right)^\alpha + \left( \frac{|B|}{K_B} \right)^\alpha}$$

(2)

Additionally, contagion $A$ and $B$ is associated with a dormancy constant $\tau_A$ and $\tau_B$ respectively. At a given time step, $\tau_A$ denotes the probability a node infected with $A$ will go dormant and no longer actively diffuse the contagion. Dormant nodes are thus removed from the counts for density. Another way to interpret $\tau_A$ is at a given time step, $\tau_A$ percent of the population infected with $A$ will go dormant.
2.1 Parallel Optimization
To optimize the simulation algorithm, a few steps to parallelize the experimentation can be made since updates are synchronous. Node neighbors are stored in memory, and in addition to the graphs, we maintain three state vectors that record the following: nodes infected with $A$, nodes infected with $B$, and nodes that are active. We use two change vectors also of dimension equal to the number of nodes, denoted $\Delta$ and $\gamma$. $\Delta$ denotes whether a node will change state based on Equation 1, and $\gamma$ is the choice of $A$ over $B$ given in Equation 2. Then the status update rule for time step $t+1$ can be written as follows, in Equation 3.

$$S_{A,t+1} = S_{A,t} + \Delta(1 - S_{A,t})\gamma$$
$$S_{B,t+1} = S_{B,t} + \Delta(1 - S_{A,t})(1 - \gamma)$$

(3)

This tends to perform better when memory is not the primary constraint, but can lead to computational redundancies without heuristics.

2.2 Simulation Setup
Experiments were run on pairwise multiplex networks with the following layers: periodic lattices (LAT), regular-random-graphs (RRG), Erdős-Rényi random graphs (ERG), powerlaw-cluster graphs (PLG), and small-world graphs (WSG). We fix the total number of nodes to 6400, then graphs parameters were computed such that the average degree was four. Table 1 shows the parameters used to initialize the graphs.

| Graph Type                     | Parameters                      |
|-------------------------------|---------------------------------|
| Regular Random Graph (RRG) [29]| Degree = 4                      |
| Erdős-Rényi Graph (ERG) [30]  | Edges = 12800                   |
| Powerlaw Graph (PLG) [31]     | Random Edge per Node = 2        |
| Lattice Graph (LAT)           | Degree = 4, Periodic = True     |
| Watts-Strogatz Small World (WSG) [32]| Nodes = 6400, Extend = 4, $P_R = 0.001$ |

Table 1: Initialization Parameters

3 Results
3.1 Shortest path and primacy jointly determines the impact of dormancy
To investigate the phenomenon of branched diffusion, we consider the dynamics of primacy. Watts-Strogatz Graphs are useful in simulating small-world phenomenon, and through adjustment of the rewiring probability $P_{R,j}$, we investigate the influence of long-range dependencies in the diffusion. The subscript $j$ denotes the probability for the specific contagion-network. We fix the rewiring probability of Contagion $A$ to 0.01, then vary the rewiring probability of Contagion $B$ between 0.00125 and 0.2.

Results show that as the rewiring probability increases, the smaller the expected radius of the graph and hence the faster the contagion diffuses. Next in Figure 2a, we introduce dormancy to investigate the effects of primacy in branch induction.

When $P_{R,B}$ is low as in Figure 2a, Contagion $B$ diffuses more slowly than $A$. However, as $P_{R,B}$ increases in (b), even when Contagion $A$ still diffuses faster its ceiling has lowered. When $P_{R,B} > P_{R,B}$ then Contagion $B$ diffuses more rapidly and hence lowers the diffusion ceiling of Contagion $A$. Additionally, note the sensitivity
Figure 1: Fixing $P_{R,A}$ to 0.01, we increase $P_{R,B}$. As expected, when $P_{R,B} = 0.005 < P_{R,A}$, diffusion is slower (left). When $P_{R,B} = P_{R,A}$ they diffuse at equal rate (mid). When $P_{R,B} = 0.05 < P_{R,A}$, contagion $B$ is faster.

Figure 2: Increasing $P_{R,B}$ with a low $T_B = 0.02$. When $P_{R,B}$ is low (a), Contagion $B$ diffuses more slowly than $A$. However, as $P_{R,B}$ increases in (b), even when Contagion $A$ still diffuses faster its ceiling has lowered. When $P_{R,B} > P_{R,A}$ then Contagion $B$ diffuses more rapidly and hence lowers the diffusion ceiling of Contagion $A$.

Contagion $B$ has towards its own dormancy rate, when $P_{R,B}$ is low. This is likely due to clustering in small-world graphs, where ring vaccination is more likely, which is consistent with the observation that clustering induces partial diffusion.

The heat map in Figure 3 show the phase transitions induced by $B$ on $A$. We observe if $\tau_B = 0$, then Contagion $A$ diffuses fully and uniformly, since even if Contagion $B$ diffuses faster, it does not introduce dormancy. However, even with small amounts of $\tau_B$, when the rewiring probability of $B$ exceeds that of $A$, then Contagion $A$ undergoes percolation. This marks a phase transition. At $\tau_B = 0.10$, the standard deviation is maximal, which indicates the point for which it is more likely $A$ transitions from the upper branch to the lower branch. For Contagion $B$, there is a linear relationship between the density of long-range relations and dormancy in determining diffusion depth.
3.2 Degree Distribution Influences Long-Long Diffusion Dynamics

Having established the effects of radius and primacy, we compare the diffusion behavior between long-range graph pairing: RRG-ERG, ERG-PLG, and RRG-PLG. These graphs have comparable radii, and differ mostly in degree distribution—RRG degrees are uniform, ERGs are Poisson distributed, and PLGs by the power law. In order, these distributions increase in variance and skew.

With no dormancy, we observe the following order in diffusion speed: $\text{RRG} > \text{ERG} > \text{PLG}$. Figure 4 shows the diffusion means of each of the graph pairings. The left heat map column shows the faster diffusing contagion all things equal, the right the slower diffusing contagion. Bi-modal diffusion curves and kernel density estimates of their branch values are shown on the very right.

The first column shows the faster contagion. It is evident that the faster contagion depends on both $T_A$ and $T_B$, as shown by the diagonal line, whereas the subsequent contagion is much more sensitive to its own dormancy rate, on the y-axis. The right-most column shows diffusion outcomes. Compared to prior results on the lattice, narrow bands of bi-modal or tri-modal branching does not occur. This is likely due to the higher variance in degree distribution.

Diffusion on these three graphs illustrate the influence of the variance and skew of degree distribution. Given both the ERG and PLG exhibit right-skew in their degree distribution, this means a higher probability that a node with lower degree distribution will be sampled, on average, than on the uniformly distributed RRG. Relatively speaking, $\text{ERG} > \text{PLG}$ is a necessary conclusion as well.

3.3 Synergy Widens the Diffusion Window

We observe the more synergistic contagions are, the faster both diffuse, which agrees with prior research [2]. Additionally, as synergy increases ($\alpha$ decreases), the resultant heat-maps grow less compressed. That is, the diffusion depth grows more sensitive to both $\tau_A$ and $\tau_B$ as $\alpha$ increases, and the lighter regions in the heatmap diminish. This implies the window for diffusion is wider for synergistic contagions.

3.4 Clustering Induces Ring Vaccination in Long-Short Diffusion

Prior studies have shown tri-modal branching occurring between the RRG and LAT' multilayer graphs [2]. We show this is true on other long-short range combinations,
Figure 4: Heat-maps of diffusion depth by network pairings and diffusion curve samples. Each row is a network pairing. The first column shows the faster diffusing contagion (all things equal), and second the slower diffusion contagion. The faster diffusing contagion’s diffusion depth depends on both dormancy rates, shown by the diagonal, whereas the slower contagion depends more on its own dormancy rate (more sensitivity across the y-axis). Bi-modal branching is observed in the diffusion curves, although the variance of the modes depends on the topology.

Specifically PLG-LAT and ERG-LAT pairings. Figure 5 shows the heat-maps of the long-range layer (first column) and the lattice layer (second column), then the diffusion curves on the right.

In contrast to the long-long-range diffusion heat maps, The long-range layer only depends on its own dormancy. This makes intuitive sense, as the spatially constrained lattice graph diffuses much slower than the PLG or ERG. On the contrary, the diffusion of the lattice layer depends heavily on the long-range layer. First, there is a steep transition between $\tau_A = 0$ and $\tau_A = 0.01$. Since the long-range contagion diffuses much faster, even low rates of dormancy induction produce a big drop in diffusion height. However, as dormancy rate increases for the long-range contagion, the long-range contagion slows in its own diffusion, eventually being overtaken by the lattice contagion. This can simultaneously between heat maps— as the PLG turns dark vertically on the left, the LAT heat-map grows brighter above the long-range phase transition.

Note the strong diagonal line on the lattice heat map. This implies as long as $\tau_{LAT} < \tau_A$ for $A \in \{RRG, ERG, PLG\}$, then the lattice contagion will diffuse.
Figure 5: Long-short range pairings with LAT-ERG on the top, LAT-PLG on the bottom. Long-range graphs (left) demonstrate vertical sensitivity and little sensitivity to the short-range dormancy. The lattice graphs show sensitivity to both dormancy values, and diffuses only when the constraint $\tau_{LAT} > \tau_A$ is met, as shown by the diagonal. Note, as the PLG and ERG graphs transition to black, thus in general as $\tau_A$ increases, the deeper the lattice contagion diffuses, particularly above the long-range phase transition constrained by $\tau_{LAT} < \tau_A$.

4 Discussion and Conclusion

The purpose of this paper is to understand the general properties of multilayer diffusion for network layers of fixed degree, and results show the relationship between synergy, dormancy, and topology is very intricate. First, we establish facts about diffusion primacy. The faster diffusing contagion, if containing non-zero dormancy, will induce branching on the slower contagion. We showed this by varying the rewiring probability of small-world graphs, and observed as the network radius decreases and the primacy relation between contagion changes, branch induction behavior switches between contagions. Second, synergistic contagions are found to have a more generous diffusion window— their diffusion depths are less sensitive to increases in dormancy compared to antagonistically additive contagions.

Third, we investigate the interface of dormancy rates and topology. In long-range graph pairings, higher variance and right-skew in degree distribution cause marginal, but important decreases in diffusion rate. The relative order in diffusion rate for long-range graphs is found to be regular-random graphs, ER-random graphs, and scale-free power law graphs. The faster contagion depends on both dormancy rates, and the slower one on its own. This relationship is flipped, however, when a long-range graph is paired with a spatially constrained lattice graph. The long-range graph depends mostly on its own dormancy rate, where dormancy and diffusion depth are inversely related. For the lattice contagion, when the long-range dormancy is zero, then it only depends on its own dormancy rate. Its diffusion depth drops drastically when the long-range dormancy increases from 0 to a small value,
but as the long-range dormancy continues to grow, the lattice contagion can penetrate deeper. The lattice contagion’s diffusion rate is thus constrained by the faster contagion’s dormancy from below, and from above by its own, as shown in Figure 5.

Before extending this model to interpret real data, analysis of a few more properties is required. Real networks typically contain a subset of all nodes, so analysis of how these properties generalize to fractional coverage is required. Additionally, investigating the gradual shift in sensitivity using lag regression would yield quantitative insight regarding the precise relationship between dormancy and topology-specific diffusion rate.

**Competing interests**
The authors declare that they have no competing interests.

**Author’s contributions**
Both authors contributed equally to the manuscript.

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**Author details**
1 School of Informatics, University of Edinburgh, Edinburgh, UK. 2 Department of Mathematics, Dartmouth College, 03755 Hanover, USA.

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