Co-diffusion of social contagions

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Abstract

Prior social contagion models consider the spread of either one contagion on interdependent networks or multiple contagions on single layer networks, usually under assumptions of competition. We propose a new threshold model for the diffusion of multiple contagions. Individuals are placed on a multiplex network with a periodic lattice layer and a random-regular-graph layer. On these population structures, we study the interface between two key aspects of the diffusion process: the level of synergy between two contagions, and the rate at which individuals become dormant after adoption. Dormancy is defined as a looser form of immunity that limits active spreading but without conferring resistance. Monte Carlo simulations reveal lower synergy makes contagions more susceptible to percolation, especially those that diffuse on lattices. Faster diffusion of one contagion with dormancy probabilistically blocks the diffusion of the other, in a way similar to ring vaccination. We show that within a band of synergy, bimodal or trimodal branchings occur on the slower contagion on the lattice. We also show complimentary contagions can provide a synergistic boost to help spread contagions that have almost gone dormant.

1. Introduction

The term ‘social contagion’ implicitly captures two worlds: the world of social science and the world of epidemiology. Although the term was initially coined in 1895 by Gustave Le Bon [1] to describe undesirable collective behaviors in crowds, its definition has been stretched to encompass and explain types of collective behavior produced through social contact [2–6]. This broad definition, with advances in statistical physics [7], has led to Contagion Theory’s inclusion within many avenues of social science research [8], including marketing [9], innovation diffusion [10], medicine [11], health interventions [12], rumor and information spreading [13, 14], sociology [15, 16], and the spread of emotion [17–19].

In the same way that two contagions may influence each other’s infectious paths, related innovations such as ideas, behaviors, products or technologies influence each other as they diffuse. In the past, social contagion models considered the spread of either one contagion at a time on interdependent networks or multiple contagions on single layer networks, usually under assumptions of pure competition. There is a recent desire to understand the diffusion of multiple social contagions under synergistic assumptions, by modeling the mechanisms of their concurrent, interfering spread. The paper has three main objectives. First, drawing upon established models within epidemiology and pharmacology, we propose a model which quantifies the amount of synergy between two contagions. Secondly, we consider the effects of stochastic dormancy to model contagions that are simultaneously cooperative and competitive. Lastly, we contrast short-range connections of lattice graphs and the long-range connections of a random-regular-graph within the multiplex network.

The term ‘social contagion’ is an oxymoron. The word contagion denotes a spreading process in which agents are infected by proximity alone. On the other hand, complex and rational individuals have the agency to make decisions. By relaxing the definition of immunity, the spread of a contagion becomes a decision rather than a neutral phenomenon. A common goal in computational social sciences is modeling agential decisions explicitly.
rather than stretch our interpretation of existing physical diffusion models. The most common critique of any mathematical representation is that they are too reductive. To add complexity while keeping assumptions tractable, we study multiplex networks and complex diffusion mechanism.

We use the term social contagion broadly, pertaining to ideas, behaviors and a field of immediate application: technological and product diffusion. The difference between competition and complementary action becomes difficult to identify when competition may, in fact, help another innovation. A new brand can increase the entire market potential due to its promotion or compete for the same market potential and slow down diffusion [20]. Given the role of social contagions in the process of innovation [21], synergy quantification frames cooperation and competition in precise, numerical terms consider general purpose technologies (GPTs). GPTs are technologies that have an impact across sectors and spillover effects as network externalities with economic benefits [22]. GPTs give rise to competing products that share complementary standards. With the imminent arrival of the next GPT revolution with artificial intelligence and blockchain, finding means to quantify synergy and predict the diffusion of related innovations is timely and useful. Similarly, the distinction between competition and cooperation in information diffusion is not clearly defined [23]. Understanding its complex propagation through the scope of network models can thus help stymie the spread of misinformation [24, 25], particularly those with multiple channels of exposure.

1.1. Literature review

Guilbeaut et al [8] give three contemporary research directions. This study focuses on the first two: the ecology of contagions, which studies how multiple contagions diffuse across different network structures, and diffusion mechanism by modeling different threshold structures.

The Diffusion of Innovation, based on the work of Rogers [26] and Bass [27], is one of the first mathematical treatments of social contagion diffusion. Models in statistical physics [7] and epidemiology were then appropriated for diffusion studies. Of particular relevance are models of multiple infections. Nowak and May were the first to model super-infection, where they assumed only the strongest virus is active and the only one that spreads [28]. Afterward, they modeled co-infection with multiple active viruses [29]. Super-infection was then shown to be a limit of co-infection. Similar to technology diffusion models, competition is modeled as cross-immunity in networks [30].

These models were then extended to study social contagions, particularly interacting, antagonistic contagions [31–33], and their cascading behaviors on multiplex networks [34–37]. Shu et al [38] present the dynamics of social contagions on two interdependent two-dimensional lattices, and give examples of nodes in communication networks which are spatially embedded [39–41]. Li et al [42] use a similar set-up to study the spread of epidemics, where they first pair two interconnected lattices, then pair two Erdős–Rényi (ER) networks. Liu et al [43, 44] experimented with similar synergistic assumptions on double layer multiplex networks, specifically ER-ER, SF-SF, ER-SF, and RR-RR graphs. In contrast, this paper considers one lattice and one random-regular-graph, and investigates the interplay of spatial and long-range graphs. In regards to application Aleta and Moreno [45] give a comprehensive review of multilayer networks percolation and how this is applied to ecology, biology, transportation, economics, game theory, and transportation.

Another approach towards diffusion on networks is with evolutionary games [46, 47]. Jiang [48] treats the mechanism for actively spreading information using games on social networks. New information is treated as a mutant; there are recent efforts to extending this to multilayer networks [48–50]. Factors of importance within networks include noise-induced adoption and network topology [51], collective influence by degree [52], teaching activity [53], and stochasticity [54]. Perc’s treatment of noise is of particular relevance given the well-documented spontaneous adoption of behavior [55]. Szolnoki has identified sharing information leads to correlated strategy choice and reinforced cooperation on multilayer networks [56], which is even more pronounced if strategy and topology coevolve [57].

For the diffusion mechanism, one common paradigm is the susceptible-infected-susceptible (SIS) model [58–60]. In the basic SIS model, individuals transition between states of susceptibility and infection, where recovered individuals are once more susceptible. Recovery from disease confers no immunity. Hill et al [55] characterize a SIS a model, in which they distinguish between spontaneous and contagious infection. Their study mentions many of the challenges this paper wishes to address: the probabilistic nature of the contagion and asymmetry within the contagions. Dodds and Watts [61] identify three basic classes of contagion models: epidemic threshold, vanishing critical mass, and critical mass. While the examples above occur on single layer networks, Liu et al [62] have modeled epidemic spread on interconnected small-world networks, where neighbors of a node are likely to be neighbors of other nodes. The typical distance L between two nodes chosen at random will grow proportionally to log N with N the number of nodes. For multiple infections, Chen et al [63] propose a model built on intrinsic properties of cooperative contagions A and B. Host individuals are in two
possible states: susceptible or infected. Susceptibles are equivalent to naïve agents and can be infected by either A or B, each associated with a cooperativity coefficient $\xi_A$ and $\xi_B$, respectively, to capture their mutual influence.

The susceptible–infection–recovered model, in contrast, confers a removed or recovered status to individuals, who are no longer susceptible to disease [64, 65]. Immunity is a parameter that has analogous application in social systems, such as resistance to rumors-spreading or belief-change. However, immunity is bidirectional. Gaining recovered status precludes the node from being both infected and from infecting others, a distinction of great import to our study. Centola notes that random links with distant nodes can either facilitate or impede diffusion when the propagation mechanism is complex [66]. Coupling implications of recovery with complex propagation forms the background of our study on diffusion mechanism.

2. Materials and methods

Without loss of generality, assume that we have two contagions: Contagion A and Contagion B. Every node in the experiment must take on one of four states: naïve ($\exists$), A, B, and AB, which correspond to uninfected/naïve individuals, adopters of Contagion A, adopters of Contagion B, and adopters of both Contagion A and B. Diffusion denotes the spread of a contagion across a network, which influences the particular status of a node. The diffusion simulation occurs on a multilayer network. The first layer is the periodic square lattice, the second is a random-regular-graphs with degree four. Thus, long-range connections versus spatially localized connections can be compared as different dynamical channels or modes within the diffusion process [37]. The threshold for A is determined by the neighbors on the lattice graph while the threshold for B is determined from the random-regular-graph. Furthermore, each node is either active or dormant, represented by the Boolean 1 or 0, respectively. The status for each node is expressed as the tuple (State, Activity). For instance, an active node infected with A would be represented by (A, 1). Updating is synchronous [67]. The algorithm is stated succinctly in algorithm 1 in the supplementary materials available online at stacks.iop.org/NJP/20/095001/mmedia.

At first, one node is randomly selected from all nodes, then seeded with Contagion A. Another one is seeded with Contagion B, with no repetition. For each node at each time step, its activity and status (denoted as Uninfected, A, B, or AB) is updated. Contagion A diffuses on a $80 \times 80$ periodic square lattice and Contagion B diffuses on a random-regular-graph with 6400 nodes and degree 4. The table 1 summarizes the fixed parameters.

2.1. Adoption probability kernel

Traditional Chinese medicines typically use mixtures of herbs to maximize their potency during the healing process. This notion has not escaped the contemporary field of pharmacology, and the last century has seen an increase in use of drug combinations. Theoretical research within the field quantify the concepts of synergy and antagonism, which go beyond the simple additive effect of using drugs individually.

The Hill function is the log-transform of the logistic function and is useful for modeling density-dependent growth. With roots in biochemistry, the Hill function is used to measure the rate of reaction between reactant concentration and substrate density. Researchers in pharmacology noticed that the effect of drugs on killing cells and bacteria could be modeled similarly, and extended the model to capture the effects of drug interaction. Since studies have shown that behavioral contagions depend on density rather than numbers [68], existing models within the pharmacology literature prove suitable for studying co-diffusion. Specifically, we develop our model with Loewe additivity [69], detailed in the supplementary materials in appendix A.2. This model gives us a general framework that is grounded in similar empirical observations between the fields of contagion diffusion and drug effectiveness. Instead of considering how two drugs work together to kill cells, we consider how complex contagions spread concurrently.

2.1.1. Probability kernel for the threshold

We treat an increase in the number of neighbors as a threshold lowering effect. As with the typical threshold model, each node is assigned a threshold $\mu_k$, where:

| Parameter     | Quantity |
|---------------|----------|
| Number of nodes | 6400     |
| Total timesteps | 700      |
| Number of initial seeds | 1        |
| Iterations per parameter set | 100      |

Table 1. Exogenous variables.
\[ \mu_i \in (0, 1) \quad \text{where } i \text{ is the coordinate of each unique node.} \]

We have assumed, without loss of generality, that we have two contagions A and B. We also assume that only one contagion can be adopted per time-step. Then there are two possible paths of adopting both A and B. Let \( i \) denote a node

\[
\begin{align*}
& i(\text{Naïve}) \rightarrow i(A) \rightarrow i(AB) \\
& i(\text{Naïve}) \rightarrow i(B) \rightarrow i(AB). 
\end{align*}
\]

(1)

Simply put, the naïve/uninfected individual must adopt A first or B first. This is known as inclusive adoption. Exclusive adoption in contrast denotes the case where the state \( \phi_i (AB) \) is not possible. Next, we denote an indicator function for the status where:

\[
S_A(i) = \begin{cases} 
1 & \text{if } i \text{ adopts A} \\
0 & \text{otherwise} 
\end{cases} \quad S_B(i) = \begin{cases} 
1 & \text{if } i \text{ adopts B} \\
0 & \text{otherwise} 
\end{cases} .
\]

Thus, the inclusive adoption probability of any state can be expressed using this general formula:

\[
P(i) = \frac{(1 - S_A(i))\left(\frac{|A|}{K_A}\right)^\alpha + (1 - S_B(i))\left(\frac{|B|}{K_B}\right)^\alpha}{1 + (1 - S_A(i))\left(\frac{|A|}{K_A}\right)^\alpha + (1 - S_B(i))\left(\frac{|B|}{K_B}\right)^\alpha},
\]

(2)

where \( K_A \) and \( K_B \) controls the attractiveness of each social contagion. The smaller the value, the more attractive it is to the population since it controls for the time step of the inflection point. \( |A| \) denotes the density of neighbor nodes that have already adopted contagion A. Specifically, let \( T \) be the total number of neighbors, then:

\[ |A| = \frac{\text{No. of } A}{T}. \]

(3)

The assumption is that \( \alpha \) and \( K \) are known and can be fit based on past data. For the purpose of this study we assume \( K_A = K_B = 2.0 \), where the choice of this parameter suits the simulation time scale. Next, we break down the sub-cases to clarify equation (2). For the naïve individual \( i \) the values of indicator function \( S_A(i) \) and \( S_B(i) \) are both zero. Hence the adoption rate of either A or B can be characterized by:

\[
P(i \leftarrow A \text{ or } B) = \frac{\left(\frac{|A|}{K_A}\right)^\alpha + \left(\frac{|B|}{K_B}\right)^\alpha}{1 + \left(\frac{|A|}{K_A}\right)^\alpha + \left(\frac{|B|}{K_B}\right)^\alpha}.
\]

(4)

The node is first activated with this probability. Then it will choose one of A and B based on their relative proportions. That is

\[
\Pr(i \leftarrow A) = \frac{\left(\frac{|A|}{K_A}\right)^\alpha}{\left(\frac{|A|}{K_A}\right)^\alpha + \left(\frac{|B|}{K_B}\right)^\alpha} \quad \Pr(i \leftarrow B) = \frac{\left(\frac{|B|}{K_B}\right)^\alpha}{\left(\frac{|A|}{K_A}\right)^\alpha + \left(\frac{|B|}{K_B}\right)^\alpha}.
\]

(5)

The notation \( \Pr(i \leftarrow A) \) denotes the probability of node \( i \) adopting Contagion A, and it is the analogous case for B. The adoption probability is shown as a surface in figure 1. In lieu of Loewe Additivity described in appendix 2.1.2, these surfaces indicate the different relationships of synergistic and antagonistic additivity. A curve that is concave downwards indicates that the effect of their sum is more than their parts and thus synergistic. When \( \alpha > 1.0 \) as with the case in the bottom right, the relationship is concave upwards which indicates antagonism. This corresponds to the formulation given in the isobologram analysis of [69].

Now without loss of generality suppose \( i \) has already adopted A. Then the probability of adopting B is given by:

\[
P(i \leftarrow B) = \frac{\left(\frac{|B|}{K_B}\right)^\alpha}{1 + \left(\frac{|B|}{K_B}\right)^\alpha}.
\]

In the experiment we set the thresholds as a function of this adoption probability. The threshold \( \mu_i \) is given in equation (6):

\[ \mu_i = 1 - P(i \leftarrow B). \]

(6)

2.1.2. Exclusive adoption

While not explicitly studied in this paper, exclusive adoption is a useful contrast to our case above. The adoption pathways can be represented as:
Naive Immunity against Naive Immunity against. 7

The expression for adoption is thus:

$$i(\text{Naive}) \rightarrow i(A) \rightarrow \text{Immunity against } B$$  
$$i(\text{Naive}) \rightarrow i(B) \rightarrow \text{Immunity against } A.$$ (7)

The expression for adoption is thus:

$$P(i \leftarrow A \ or \ B) = (1 - S_A(i))(1 - S_B(i)) \frac{(\frac{|A|}{K_A})^\alpha + (\frac{|B|}{K_B})^\alpha}{1 + (\frac{|A|}{K_A})^\alpha + (\frac{|B|}{K_B})^\alpha}.$$ (8)

The node then chooses A or B with the coin-flip expressed in equation (5).

2.2. Stochastic dormancy

The prior two parameters $\alpha$ and $K$ model the shape of diffusion. As time approaches infinity, the diffusion process will always attain maximal value. This is not the case in reality, as the penetration is limited by exogenous factors. We model these by introducing stochastic dormancy to every node on the graph, such that nodes are not active in perpetuity.

To do this, we attach a constant $\tau_A$ and $\tau_B$ to Contagions A and B, respectively. $\tau_A$ denotes the probability that a node infected with A will become dormant at any given time step. When a node is considering adoption, if a neighbor is dormant then that neighbor is discounted from the numerator of the density, and is not included in the threshold lowering effect. To be numerically precise

$$[A] = \frac{\text{No. of Active } A}{T}.$$ (9)

The same holds for Contagion B. Another way of interpreting $\tau$ is that $\tau_A$ represents the expected proportion of nodes infected by A that switch off at each time-step. For nodes infected with both A and B, $\tau_{AB}$ is the arithmetic average, or $\tau_{AB} = \frac{\tau_A + \tau_B}{2}$. Different conditions for convexity is another line of inquiry.

It is important to distinguish between immunity/recovery/resistance with dormancy. In epidemiology, immunity implies two conditions—a recovered individual can no longer be infected nor can it infect other nodes. For the purpose of studying social contagions, we relax the first condition. In other words, while inactive individuals no longer affect other nodes, they themselves can still be infected by other contagions. They do not gain immunity but cease to participate in diffusion. This distinction models agents playing an active role in decision making during the diffusion process.

Figure 1. Adoption probability. The kernel surface of the multivariate Hill function (equation (5)) describes the probability of adopting either A or B, depending on the densities of A and B. Diagram (a) denotes constant adoption, (b) denotes synergistic additivity, (c) denotes near linear additivity, and (d) denotes antagonistic additivity. We assume $K_A = K_B = 2.0$ such that the parametrization is symmetric between Contagion A and Contagion B, though the parameter can be adjusted for asymmetry. This adoption kernel is used to study diffusion on a multiplex graph of 6400 nodes, with a periodic lattice layer and a random regular graph of degree 4.
We begin the analysis by considering the diffusion curves directly. In figure 2, Multiplex diffusion curves while varying $\tau_B$, fixing $\alpha = 1.2$ and $\tau_A = 0$. Introducing dormancy through $\tau_B$ produces branching in Contagion A diffusion curves. Low levels of $\tau_B$ introduce dense lower branch for Contagion A as shown in (b). As $\tau_B$ is increased to 0.10, Contagion B undergoes greater percolation and the branches of Contagion A are distributed more evenly. At high levels of $\tau_B$ in (c) and (f), Contagion B rarely diffuses first and Contagion A returns to its original shape of diffusion.

2.3. Experimental and measured variables

The primary variable of interest is the penetration depth, which we term ceiling as shorthand. This is the equilibrium value at the end of diffusion, calculated with the final 20% of period values. Secondly, the inflection point is used as a proxy for rate, as it reflects the time-step where the curve attains exactly half of its ceiling. If this point is pushed right, diffusion takes a longer time and implies a lower rate. Once more, only active members on each contagion’s respective graphs are considered. The experimental variables are as follows: synergy $\alpha \in [0, 1.3]$ and dormancy $\tau_A, \tau_B \in [0, 0.15]$.

3. Results

We begin the analysis by considering the diffusion curves directly. In figure 2(a), Contagion B shown in yellow diffuses much faster than Contagion A. Given that ‘attractiveness’ parameterized by $K_A$ and $K_B$ are equal, this can be attributed to two advantages of random long-range connections. First, as the diffusion process starts the uninfected nodes that Contagion B is in contact with is much greater than that of Contagion A. The uninfected nodes that Contagion A affects are always restricted to some physical front, like the propagation of a wave. Secondly, the long-range connections reduce the effect of percolation produced by $\tau_B$. In contrast, restricting diffusion to von Neumann neighborhood encourages local percolation since many of the naïve individuals share neighbors. This increases the chance that a mutual neighbor no longer participates in diffusion and thus decreasing the density-based adoption probability on the overall front.

The central topic of this study is how two contagions can spread synergistically, and how their intrinsic properties affect one another. Of great interest is the branching of Contagion A shown in blue in figure 2, where we fix the values of $\alpha = 1.2$ and $\tau_A = 0$ while varying $TB$. When Contagion B’s dormancy rate is increased marginally from $\tau_B = 0$ to $\tau_B = 0.02$, a large degree of branching in Contagion A is observed, shown in figure 2(b). In other words, the intrinsic dormancy of Contagion B greatly influences the diffusion of A. As $\tau_B$ is increased from 0.02 to 0.07 the upper branch of blue (Contagion A) grows denser. Notice that the upper branch does not contain any red. This implies when Contagion A diffuses fully, Contagion B had not diffused much if at all. We observe that as $\tau_B$ grows, the upper yellow branch diminishes, and a greater degree of blue curves converges maximally. Naturally, we suspect that $\tau_B$ induces a bimodal split on Contagion B, which is confirmed by kernel density estimates in the supplementary materials (figure A4).

To understand this instability, first consider the effect of synergy on the rate of diffusion. Figure 3 shows an increase in $\alpha$ slows down the rate of diffusion. Furthermore, there is a sharp rise at $\alpha = 0.8$, where the adoption kernel shown by equation (2) and figure 1 transitions from synergistically additive to antagonistically additive. Additionally, note that as $\tau_A$ increases, the rate of diffusion slows. This implies the dormancy of the slower diffusing contagion produces percolation.

Figure 4 shows two possible outcomes produced from the branching, though not the only two. Figure 4(a) occurs when Contagion B is stopped by its own dormancy rate, thus allowing Contagion A to fully diffuse to its von Neumann neighbors and across the entire lattice. Figure 4(b), on the other hand, occurs when Contagion B cascades and diffuses first, and over time introduces dormancy into the population. Notice that figure 4(b)
converges to equilibrium much faster, which is reflected in the inflection points of figure 2. This gives a deep result. Ring vaccination is shown in figure 4(b), where dormancy in the surrounding neighbors percolates the diffusion of Contagion A, only requires a uni-directional definition of dormancy. In other words, resistance is un-necessary for ring vaccination given that agents do not actively spread the contagion.

In comparison, varying $t_A$ under the same parametrization does little to influence Contagion B, as Contagion B diffuses much more quickly (shown in figure A7). We observe an average rightwards shift to the curves of Contagion A resulting from the higher values of $t_A$. Asymmetric parametrization of equation (2) is discussed later in the conclusion as a potential improvement to comparing diffusion on different graphs.

From these facts and figure A4 we infer that Contagion B’s branching produces three outcomes for blue. If Contagion B does not diffuse, then Contagion A diffuses fully. If Contagion B diffuses fully, then Contagion A diffuses partially or not at all. As the value of $t_B$ increases and these modes of Contagion B shifts, so too does the convergence of Contagion A.

We now summarize the interaction of $t_A$ and $t_B$ using heatmaps, beginning with Contagion A, in figures 5 and 6. When $\alpha = 0$ the diffusion probability is uniformly 0.5 as shown in the probability function (figure 1(a)). Diffusion is both constant and rapid, which forces Contagion A to converge maximally, similar to the case of single layer diffusion. As $\alpha$ increases and diffusion slows, $t_B$ has a pronounced effect on the ceiling. Consider the case when $\alpha = 0.2$ and $t_B$ jumps from 0.02 to 0.03, shown as a horizontal white line. The mean ceiling diminishes rapidly shown by the vertical change of red to white to blue. As $\alpha$ increases the decrease in ceiling mean is even more pronounced, which produces the very noticeable color jump between $t_B = 0$ and $t_B = 0.01$. This rapid drop in the ceiling is a testament to how much faster $t_B$ diffuses, where even low values of $t_B$ have sufficient time to inject dormancy into the populace. Consider the last row of the grid where $\alpha = 1.1, 1.2$ and 1.3. When $t_B$ is constant we observe a gradient effect left to right from increasing $t_A$, most pronounced when $t_B = 0$. We conclude that if $\alpha > 1.0$, then $t_A$ has a significant effect on the ceiling of Contagion A.

The previous two observations on $t_B$ and $t_A$, respectively, allow us to conclude that increasing $\alpha$ compresses the graph from the right and from the top. The two red horizontal rows in the $\alpha = 0.2$ grid are eventually compressed into one row from above. The horizontal gradient of $t_B = 0$ and $\alpha = 1.1, 1.2$ and 1.3 (bottom row

Figure 3. Increasing $\alpha$ slows diffusion of nodes with both contagions. An increase in $\alpha$ decreases the synergy between two contagions, hence decreases the rate of diffusion. Higher levels of dormancy, shown by $\tau_A$, produces a greater amount of branching.

Figure 4. Two branched outcomes from the same parametrization. On the left, Contagion A diffuses completely as Contagion B has stopped diffusion due to high percolation resultant of $t_B = 0.1$. On the right, Contagion B diffuses fully and produces ring vaccination against A. Contagion B in the blue cluster are not necessarily neighbors. Not shown is a sub-case of the left, where Contagion B diffuses fully with a ‘synergistic boost’ from Contagion A. Note, the two contain different RRG topologies.
of the grid) is compressed towards the left. Increasing $\alpha$ decreases the synergy, slows diffusion, and thus gives $\tau_A$ and $\tau_B$ more time to produce dormancy although they demonstrate different sensitivities towards $\alpha$.

When $\tau_B$ is high the ceiling of $A$ actually increases. This phenomenon is due to $\tau_B$ slowing the diffusion of Contagion $B$, which produces more branched ‘upper’ curves of Contagion $A$. This is observable by comparing figures 2(b) and (d) (with $\tau_B = 0.02$ and 0.1, respectively). This branching can also be shown with the standard deviation heatmap. The strongest nonlinearities occur when $\tau_A \gg \tau_B$ or $\tau_B \gg \tau_A$, shown in figure 6. This corresponds to the white regions in figure 6 (upper left and bottom right corner when $\alpha > 0.8$), which reflect high levels of standard deviation as result of the branching. Further testing on single layer lattices (shown in the figures A2 and A3 in the supplementary materials) confirms that the greatest branching occurs when the difference between $\tau_A$ and $\tau_B$ is large, which can branch into three clusters. However, $\tau_B$ influences the diffusion variance of $A$ for many low values of $\tau_A$, shown by the top-left white region when $\alpha > 0.8$. In comparison, the effect of $\tau_A$ is most pronounced when $\tau_A = 0$. The dormancy variable of Contagion $B$ influences the ceiling of Contagion $A$ greatly because of its diffusion primacy. Also, note the bottom right corner has standard deviation always at 0 when $\tau_B = 0$, since the contagions diffuse maximally.

Contagion $B$ produces a very different looking heat map in figure 7. Given its fast diffusion, Contagion $B$ penetrates fully or close to fully for $\alpha < 0.8$. Then, a diagonal white line moves from the top right downwards and compresses towards the bottom left. The slope of this white line and its spread can be interpreted as
Contagion B’s sensitivity to $\tau_A$. In other words, it quantifies how much the dormancy constant of Contagion A affects its own diffusion. In a similar vein, the prior heat maps are also sensitivity analyses and also show the dominance of $\tau_B$. The effects of $\tau_A$ only come in play with sufficiently high $\alpha$ to slow the diffusion rate.

We are interested in pinpointing the specific conditions that induce branching. Instabilities can be inferred from high levels of standard deviation shown in figure 8. The region of highest instability, shown in red, overlaps with the white line in figure 7. This is particularly evident in the last row where $\alpha = 1.4$–1.6. The region of the line becomes compressed and is sandwiched between two blue areas. The blue zone in the bottom left denotes the cases where Contagion B fully diffuses, the blue zone above denotes the case where Contagion B does not diffuse at all due to percolation produced by the high value of $\tau_B$. Increasing $\alpha$ produces the compression effect towards $\tau_A = \tau_B = 0$ from above as timescale increases.

One implication of Contagion B’s variance profile is that, unlike the lattice diffusion experiment, instabilities for Contagion B not only occur when $\tau_A \gg \tau_B$ or $\tau_B \gg \tau_A$, but on any point of the white line. For instance, with each parameter set denoted with the tuple $(\alpha, \tau_A, \tau_B)$, the outcome of $(1.6, 0.00, 0.04)$ is equal to $(1.6, 0.10, 0.02)$ by expectation, although there is greater instability in the latter case when $\tau_A = 0.10$ shown by the red in figure 8. In any case, while $\tau_B$ induces branching to a greater effect than $\tau_A$, $\tau_A$ influences instability as well. Since we know Contagion B diffuses bimodally, the red regions in figure 8 produce the most even branches for B. Once more, we observe compression towards $\tau_A = \tau_B = 0$ from increasing $\alpha$. 

Figure 7. Multiplex ceiling mean of Contagion B, varying $\alpha$. The white line indicates a linear relationship for $\tau_A$ and $\tau_B$ to Contagion B’s ceiling sensitivity.

Figure 8. Multiplex ceiling SD of Contagion B, varying $\alpha$. The SD is greatest along the line in figure 8. The branching is linearly dependent on both $\tau_A$ and $\tau_B$. 
is highly nonlinear and probabilistic. Diffusion of contagion contagions interacting on a multiplex network. Heat maps reveal how the parameters broadly in

The objective of this study was to investigate the properties of a diffusion model by simulating two complex

4. Discussion

Lastly, we consider when both values of $\tau$ are non-zero. Figure 9 shows the interaction of $\tau_A$ and $\tau_B$. When both $\tau_A$ and $\tau_B$ are low, Contagion A converges to the lower cluster. When $\tau_A$ is high in figures 9(a) and (b), an already congested process percolates further and Contagion A barely diffuses. More interesting is when $\tau_A$ is low and $\tau_B$ is gradually increased. For Contagion A, the branching effect is most pronounced when $\tau_A$ is low and $\tau_B$ is high in figures 9(e) and (f). The inverse happens to Contagion B, which loses its upper branches as $\tau_B$ is increased, illustrated by the density of yellow. However, note the overlap of yellow and red lines in figures 9(e) and (f). Once Contagion A has fully diffused, it produces synergistic effects that help boost the diffusion of Contagion B.

To summarize this interaction, the ratio of $\alpha$ and $\tau_B$ determines the distribution of branching. The case is similar to when $\tau_A$ was fixed to zero. When Contagion B diffuses faster than Contagion A, then $B$ sets the ceiling either maximally or to the partial branch. If Contagion B does not diffuse then Contagion A diffuses near maximally, shown by the blue upper branches without red in figures 9(e) and (f). When $\tau_B$ is large, the latter case where Contagion $B$ fails to diffuse is probabilistically more likely, but the number of curves that diffuse after Contagion A due to synergistic effects increases. It does not diffuse partially since it is not affected by local percolation, and is supported by the critical mass that Contagion A provides. Although $\tau_A$ negatively influences Contagion B, in the cases where Contagion A diffuses first and $\tau_A$ influences $A$ more negatively than $B$, overall, an increase of $\tau_A$ may boost the diffusion of $B$.

4. Discussion

The objective of this study was to investigate the properties of a diffusion model by simulating two complex contagions interacting on a multiplex network. Heat maps reveal how the parameters broadly influence the level of diffusion. Increasing $\alpha$ decreases the synergy and decreases the rate of diffusion. The stochastic dormancy of Contagion B lowers the penetration depth of both Contagion B and Contagion A. The parameter set ($\alpha$, $\tau_A$, $\tau_B$) is highly nonlinear and probabilistic. Diffusion of Contagion B on the RRG based on the value of $\tau_B$, then Contagion A branches into two or three clusters based on the ratio $\tau_A$ to $\tau_B$ for a set range of $\alpha$. This branching on the lattice occurs when the difference between $\tau_A$ and $\tau_B$ is large within the region of $0.8 < \alpha < 1.3$.

Contagion B diffuses much faster than Contagion A, due to two reasons. First, the random-regular-graph has a topologically larger diffusion front compared to the lattice's, which is constrained physically. Second, it is diffusion is not constrained by local percolation. We conclude that primacy is important in this model of co-diffusion. Furthermore, if Contagion B faces percolation from its own dormancy on the RRG, then Contagion A can attain maximal penetration and help Contagion B diffuse with a synergistic boost. If Contagion A diffuses more slowly than Contagion B, then it is subject to partial diffusion due to ring vaccination and the dormancy introduced by the faster diffusion contagion. This implies that dormancy, the looser and one directional definition of immunity, is a sufficient condition for ring vaccination.

One critique of agent-based modeling is that reductivism diminishes utility and applicability. Here, we offer some potential ways to interpret these results. In particular, we draw on the notions of synergy and dormancy.
with GPTs mentioned in the literature review in section 1.1. Blockchain technology has recently been considered as a potential general purpose technology [70–72]. The application of blockchain to cryptocurrencies, or Bitcoin, spurned a large wave of interest and investment in 2017 [73]. Given the speculation surrounding cryptocurrency, this investment behavior can be categorized as a social contagion [74]. While cryptocurrencies theoretically serve as means of transactions, investors also treat them as a commodity or asset, rather than as liquid money. The adoption of cryptocurrency is not dissimilar to a firm’s adoption of a GPT to increase output and returns. Thus, if you are an investor with a finite portfolio, there are two questions that naturally arise. First, do you adopt the technology and invest in Bitcoin? Second, which cryptocurrency do you choose to invest in?

The first question corresponds to equation (4), where all cryptocurrencies synergistically contribute to the diffusion of the adoption behavior. The second question refers to the coin-flip in equation (5), which frames individual cryptocurrencies as competitors. However, this does not preclude the possibility of adopting the other cryptocurrency at a future time. The diffusion mechanism of this paper captures both cooperative and quasi-competitive aspects of the technology as stated by Chandrasekaran [20].

Future pathways of inquiry include shape parametrization, seeding, the diffusion mechanism, and graph topologies. In this study, we assume the shape characterization of contagion adoption to be the same; where $K_A = K_B$. Introducing asymmetry can control precisely how much faster one contagion diffuses relatively to the other. For $\tau_A$ to restrain the diffusion of $\tau_B$, $B$ must diffuse slowly. Hence, varying $K_B$ would be meaningful for interdependent networks, to curb diffusion on random-regular-graph. The timing of entry poses an interesting question. Given that Contagion $A$ diffuses first with a high value of $\tau_A$, a late entry by Contagion $B$ would most likely affect its penetration depth. Quantifying this result would be useful for benchmarking the benefits or risks of late adaptation.

We have shown a difference in the diffusion of spatial and long-range graphs. For a model to accurately describe information dissemination, realistic networks would have to be implemented [16] to capture different modes and dynamical channels. For instance, in the case of social media, power-law graphs and lattice graph may be suitable. Applying clustering algorithms to the ceilings may help us understand the modality of convergent behavior. In lieu of current research directions [8], we can also consider structural diversity and the effects of homophily. Lastly, modeling exclusive adoption as we have outlined in section 2.1.2. Such a probability kernel for diffusion would be useful for modeling products where mutual adoption is impossible. Instead, a network coordination model could be used to produce a more complicated, but still tractable diffusion mechanism.

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References

[1] Le Bon G 2017 The Crowd (New York: Transaction Press)
[2] Bond R M et al 2012 A 61-million-person experiment in social influence and political mobilization Nature 489 295
[3] Wang W, Tang M, Shu P and Wang Z 2016 Dynamics of social contagions with heterogeneous adoption thresholds: crossover phenomena in phase transition New J. Phys. 18 013029
[4] Wang W, Shu P, Zhu Y X, Tang M and Zhang Y C 2015 Dynamics of social contagions with limited contact capacity. Chaos: an Interdisciplinary J. Nonlinear Sci. 25 103102
[5] Ruan Z, Iniguez G, Karsai M and Kertész J 2015 Kinetics of social contagion Phys. Rev. Lett. 115 218702
[6] Cozzo E, Banos R A, Meloni S and Moreno Y 2013 Contact-based social contagion in multiplex networks Phys. Rev. E 88 050801
[7] Wang Z et al 2016 Statistical physics of vaccination Phys. Rep. 664 1–112
[8] Guilleaudeau D, Becker J and Centola D 2018 Complex contagions: a decade in review Complex Spreading Phenomena in Social Systems (Berlin: Springer) pp 3–25
[9] Van den Bulte C and Stremersch S 2004 Social contagion and income heterogeneity in new product diffusion: a meta-analytic test Mark. Sci. 23 530–44
[10] Iyengar R, Van den Bulte C and Valente T W 2011 Opinion leadership and social contagion in new product diffusion Mark. Sci. 30 195–212

[11] Van den Bulte C and Lilien G L 2001 Medical innovation revisited: social contagion versus marketing effort Am. J. Sociol. 106 1409–35

[12] Wang Z, Moreno Y, Boccacci S and Perc M 2017 Vaccination and Epidemics in Networked Populations—An Introduction (Amsterdam: Elsevier) (https://doi.org/10.1016/j.chaos.2017.06.004)

[13] Moreno Y, Nekovee M and Pacheco A F 2004 Dynamics of rumor spreading in complex networks Phys. Rev. E 69 066130

[14] Jalili M and Perc M 2017 Information cascades in complex networks J. Complex Netw. 5 665–93

[15] Burt R S 1987 Social contagion and innovation: obsession versus structural equivalence Am. J. Sociol. 92 1287–335

[16] Gallos L K, Rybiki D, Liljeros F, Havlin S and Makse H A 2012 How people interact in evolving online affiliation networks Phys. Rev. X 2 031014

[17] Fowler J H and Christakis N A 2008 Dynamic spread of happiness in a large social network: longitudinal analysis over 20 years in the Framingham heart study BMJ 337 a2338

[18] Caccioppo J T, Fowler J H and Christakis N A 2009 Alone in the crowd: the structure and spread of loneliness in a large social network J. Personality Soc. Psychol. 97 977

[19] Hill A L, Rand D G, Nowak M A and Christakis N A 2010 Emotions as infectious diseases in a large social network: the SIS a model Proc. R. Soc. B 277 3827–35

[20] Chandrasekaran D and Tellis G J 2007 A critical review of marketing research on diffusion of new products Review of Marketing Research (Bingley: Emerald Group) pp 39–80

[21] Langley D J, Bijmolt T H, Ortt J R and Pals N 2012 Determinants of social contagion during new product adoption J. Prod. Innov. Manage. 29 623–38

[22] Lisper G R, Carlaw K I and Bekar C T 2005 Economic Transformations: General Purpose Technologies and Long-Term Economic Growth (Oxford: Oxford University Press)

[23] Myers S A and Leskovec J 2012 Clash of the contagions: cooperation and competition in information diffusion 2012 IEEE 12th Int. Conf. on Data Mining (ICDM) (Piscataway, NJ: IEEE) pp 539–48

[24] Budak C, Agrawal D and El Abbadi A 2011 Limiting the spread of misinformation in social networks Proc. 20th Int. Conf. World Wide Web (New York: ACM) pp 665–74

[25] Weng L, Flammini A, Vespignani A and Menczer F 2012 Competition among memes in a world with limited attention Sci. Rep. 2 3355

[26] Rogers E M 2010 Diffusion of Innovations (New York: Simon and Schuster)

[27] Bass F M 1969 A new product growth for model consumer durables Manage. Sci. 15 215–27

[28] Nowak M A and May R M 1994 Superinfection and the evolution of parasite virulence Proc. R. Soc. B 355 81–9

[29] May R M and Nowak M A 1995 Coinfection and the evolution of parasite virulence Proc. R. Soc. B 261 209–15

[30] van de Bovenkamp R, Kuipers F and Van Mieghem P 2014 Domination–time dynamics in susceptible–infected–susceptible virus competition on networks Phys. Rev. E 89 042818

[31] Czaplicka A, Toral R and San Miguel M 2016 Competition of simple and complex adoption on interdependent networks Phys. Rev. E 94 062301

[32] Velásquez-Rojas F and Vazquez F 2017 Interacting opinion and disease dynamics in multiplex networks: discontinuous phase transition and nonmonotonic consensus times Phys. Rev. E 95 052315

[33] Zhao K and Bianconi G 2013 Percolation on interdependent networks with a fraction of antagonistic interactions J. Stat. Phys. 152 1069–83

[34] Brummitt C D, Lee K M and Goh K I 2012 Multiplexity–facilitated cascades in networks Phys. Rev. E 85 045102

[35] Yagan O and Gilgior V 2012 Analysis of complex contagions in random multiplex networks Phys. Rev. E 86 036103

[36] Lee K M, Brummitt C D and Goh K I 2014 Threshold cascades with response heterogeneity in multiplex networks Phys. Rev. E 90 062816

[37] Kivelä M, Arenas A, Barthelemy M, Gleeson J P, Moreno Y and Porter M A 2014 Multilayer networks J. Complex Netw. 2 203–71

[38] Shu P, Gao L, Zhao P, Wang W and Stanley H E 2017 Social contagions on interdependent lattice networks Sci. Rep. 7 44669

[39] Barthelemy M 2011 Spatial networks Phys. Rep. 499 1–101

[40] Daqing L, Kosmidis K, Bunde A and Havlin S 2011 Dimension of spatially embedded networks Nat. Phys. 7 481

[41] Boccacci S et al 2014 The structure and dynamics of multilayer networks Phys. Rep. 544 1–122

[42] Li D, Qin P, Wang H, Liu C and Jiang Y 2014 Epidemics on interconnected lattices Europhys. Lett. 105 68004

[43] Liu Q H, Wang W, Cai S M, Tang M and Lai Y C 2018 Synergistic interactions promote behavior spreading and alter phase transitions on multiplex networks Phys. Rev. E 97 022311

[44] Liu Q H, Zhong L F, Wang W, Zhou T and Eugene Stanley H 2018 Interactive social contagions and co-infections on complex networks Chaos 28 013120

[45] Aleta A and Moreno Y 2018 Multilayer networks in a nutshell arXiv:180403488

[46] Zinoviev D and Duong V 2011 A game theoretical approach to broadcast information diffusion in social networks Proc. 44th Annual Simulation Symp. (Society for Computer Simulation International) pp 47–52

[47] Qiu W, Wang Y and Yu J 2012 A game theoretical model of information dissemination in social networks 2012 Int. Conf. on Complex Systems (ICCS) (Piscataway, NJ: IEEE) pp 1–6

[48] Jiang C, Chen Y and Liu K R 2014 Graphical evolutionary game for information diffusion over social networks IEEE J. Sel. Top. Sig. Process. 8 524–36

[49] Wang Z, Wang L, Szolnoki A and Perc M 2015 Evolutionary games on multilayer networks: a colloquium Eur. Phys. J. B 88 124

[50] Perc M, Gómez-Gardeñes J, Szolnoki A, Floría L M and Moreno Y 2013 Evolutionary dynamics of group interactions on structured populations: a review J. R. Soc. Interface 10 201102997

[51] Perc M 2006 Double resonance in cooperation induced by noise and network variation for an evolutionary Prisoner’s dilemma New J. Phys. 8 183

[52] Szolnoki A and Perc M 2016 Collective influence in evolutionary social dilemmas Europhys. Lett. 113 58004

[53] Szolnoki A and Perc M 2008 Coevolution of teaching activity promotes cooperation New J. Phys. 10 043036

[54] Perc M and Marhl M 2006 Evolutionary and dynamical coherence resonances in the pair approximated Prisoner’s dilemma game New J. Phys. 8 142

[55] Hill A L, Rand D G, Nowak M A and Christakis N A 2010 Infectious disease modeling of social contagion in networks Proc. R. Soc. B 277 3827–35

[56] Szolnoki A and Perc M 2013 Information sharing promotes prosocial behaviour New J. Phys. 15 053010
[57] Wang Z, Szolnoki A and Perc M 2014 Self-organization towards optimally interdependent networks by means of coevolution. New J. Phys. 16 033041

[58] Ball F, Mollison D and Scalia-Tomba G 1997 Epidemics with two levels of mixing Ann. Appl. Probab. 7 46–89

[59] Watts D J and Strogatz S H 1998 Collective dynamics of small-world networks Nature 393 440

[60] Keeling M J 1999 The effects of local spatial structure on epidemiological invasions Proc. R. Soc. B 266 859–67

[61] Dodds P S and Watts D J 2005 A generalized model of social and biological contagion J. Theor. Biol. 232 587–604

[62] Liu M, Li D, Qin P, Liu C, Wang H and Wang F 2015 Epidemics in interconnected small-world networks PLoS One 10 e0120701

[63] Chen L, Ghantabarnejad F and Brockmann D 2017 Fundamental properties of cooperative contagion processes New J. Phys. 19 103041

[64] Nowak M A 2006 Evolutionary Dynamics (Cambridge, MA: Harvard University Press)

[65] Anderson R M and May R M 1992 Infectious Diseases of Humans: Dynamics and Control (Oxford: Oxford University Press)

[66] Centola D, Eguíluz V M and Macy M W 2007 Cascade dynamics of complex propagation Physica A 374 449–56

[67] Schönfisch B and de Roos A 1999 Synchronous and asynchronous updating in cellular automata BioSystems 51 123–43

[68] Freedman J L, Birsky J and Cavoukian A 1980 Environmental determinants of behavioral contagion: density and number Basic Appl. Soc. Psychol. 1 135–61

[69] Foucquier J and Guedj M 2015 Analysis of drug combinations: current methodological landscape Pharmacol. Res. Perspect. 3 3

[70] Catalini C and Gans J S 2016 Some simple Economics of the Blockchain (Washington, DC: National Bureau of Economic Research) (https://doi.org/10.2139/ssrn.2932585)

[71] Kane E 2017 Is Blockchain a General Purpose Technology? (Rochester, NY: Social Science Research Network) (https://doi.org/10.2139/ssrn.2874598)

[72] Davidson S, De Filippi P and Potts J 2016 Economics of Blockchain (Rochester, NY: Social Science Research Network) (https://doi.org/10.2139/ssrn.2744751)

[73] Carrick J 2016 Bitcoin as a complement to emerging market currencies Emerg. Markets Financ. Trade 52 2321–34

[74] Baek Ch and Elbeck M 2015 Bitcoins as an investment or speculative vehicle? A first look Appl. Econ. Lett. 22 30–4

Bariviera A F, José Baigull M, Hasperué W and Naïouf M 2017 Some stylized facts of the Bitcoin market Physica A 484 82–90