Complex Contagions and hybrid phase transitions in unclustered and clustered random networks

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A complex contagion is an infectious process in which an individual may require multiple transmissions. We typically think of individuals as beginning inactive and becoming active once they are contacted by sufficient numbers of active partners. These have been studied in a number of contexts, but the analytic models for dynamic spread of complex contagions are typically complex. Here we study the dynamics of a generalized Watts Threshold Model (gWTM). We first show that a wide range of other processes can be thought of as a special case of this gWTM. Then we adapt an “edge-based compartmental modeling” approach used for infectious diseases in networks to develop and analyze analytic models for the dynamics the gWTM in configuration model and a class of random clustered (triangle-based) networks. The resulting model is relatively simple and compact, and we use this model to gain insights into the dynamics. Under some conditions a cascade can happen with an arbitrarily small initial proportion active, and we derive conditions for this to happen. We also sometimes observe hybrid phase transitions when cascades are not possible for small initial conditions, but do occur for large enough initial conditions. We derive some simple new sufficient conditions for the hybrid phase transition to occur. We show that in many cases, above the hybrid phase transition, all individuals will eventually become active. Finally, we discuss the role clustering plays in facilitating or impeding the spread. This approach allows us to unify many existing disparate results and derive some new results.

INTRODUCTION

Many “infectious” processes spread in social contact networks. The most studied of these are susceptible-infected-susceptible (SIS) and susceptible-infected-recovered (SIR) diseases. For these processes, a single transmission suffices to infect. However, a range of behavioral patterns such as rumor spread or technology use are believed to spread as “complex contagions.” Here, an individual typically requires more than one transmission from “active” individuals to become “activated” [42]. These have received less study compared to infectious diseases.

We will investigate a generalized version of the Watts Threshold Model [42] (gWTM) spreading through a network. In this general model, each node is assigned a threshold \( r_u \) such that if at least \( r_u \) neighbors are active, then the node “activates”, becoming active. If \( r_u \leq 0 \), then \( u \) is active at the initial time. In frequently studied cases, \( r_u \) is the same for all nodes, or \( r_u = \alpha k_u \) represents the same fraction \( \alpha \) of neighbors for all nodes. By careful choice of the rule for assigning \( r_u \), we can arrive at a wide range of other percolation processes as special cases of the gWTM. We will briefly describe some of these processes.

We will study a continuous time version of the gWTM. Rather than using “synchronous” updating, we will adopt an asynchronous update rule. Active nodes “transmit” at rate \( \beta \) to their neighbors. We can think of this as a node transmitting infection or perhaps information that it has adopted some behavior. Often this is considered to be a “message” which is passed. In this paper we will use the term “transmit”. Once a node \( u \) has received \( r_u \) transmissions, it immediately switches to active. To study this, we will adapt a compartmental modeling approach focusing on the status of edges rather than nodes. This “Edge-based compartmental modeling” technique was originally developed for SIR disease spread and can be applied with a wide range of assumptions about the population or disease process [30, 33, 34]. It allows for a significant reduction in the number of equations used compared to other approaches, in some cases reducing an infinite (or even doubly infinite) system of differential equations to an equivalent system governed by a single differential equation [32]. We will see that the same approach can give a significant simplification here as well.

Because the gWTM contains many other percolation processes as special cases, this approach can be adapted to those as well.

It is generally believed that clustering enhances the spread of complex contagions [6, 14, 17], and experimental evidence supports this [5]. The basis for this argument is that clustering will tend to introduce correlations between the statuses of a node’s neighbors. Thus a node who sees one active neighbor is more likely to see others, and thus more likely to activate. In contrast it is fairly well established that clustering tends to reduce the spread of SIR diseases, even if only mildly [11, 22, 23, 28, 29, 41]. Work by [14] suggests that clustering may increase or decrease the final size of gWTM cascades. We will use our model to study how clustering alters the dynamics of gWTM cascades.

In our study, we find that hybrid phase transitions appear to be common in the gWTM. We discuss some of the examples we encounter. Although these do not appear to have been seen in the gWTM, they have been seen in several types of percolation that we show to be a special case of the gWTM [3, 7, 8], so this result is not surpris-
ing of its own right. We are able to find some sufficient conditions for the hybrid phase transition to occur. In particular, there is a hybrid phase transition if \( r \leq k - 1 \) for all nodes and neither the \( r = 1 \) nodes nor the \( r = k - 1 \) nodes form a giant component. Furthermore in this scenario we anticipate that above this threshold all nodes eventually activate, but we do not have a rigorous proof.

The remainder of this paper is set out as follows. We first show that many percolation processes can be thought of as special cases of the gWTM. We then adapt the recently developed techniques for SIR disease to the modeling of complex contagions in both unclustered and clustered networks. To do this, we first develop an analytic model of the dynamics in Configuration Model networks. We observe an initially surprising hybrid phase transition. We derive conditions for this hybrid phase transition to occur. We then develop equations for a specialized class of random clustered networks. We can study this system analytically, but it comes at a cost that these random clustered networks have a special form that limits the role of clustering. So the insights we gain may not be universal.

**THE GWTM MODEL AS A GENERALIZATION OF OTHER MODELS**

The gWTM can be thought of as a generalization of a wide range of other percolation models. So results derived for the gWTM are immediately applicable to these other models. In this section we show how site percolation, bond percolation, \( k \)-core percolation, and bootstrap percolation arise as special cases of the gWTM. The first three of these can alternately be shown following the “response function” formalism of [9]. We also show that if transmission is modified such that the probability an active node \( v \) eventually transmits to any given neighbor \( u \) is \( p < 1 \) independently of all other neighbors, then by an appropriate change of \( r_u \), we can capture this using the gWTM model.

We begin by analyzing site percolation. In classical site percolation, nodes are either present with probability \( p \) or absent with probability \( 1 - p \). Typically one studies the connected components of present nodes. We can find one of these components by starting with a single present node and exploring the network by following present edges. In the context of the gWTM, if the probability of a given \( r \) is \( p(1-p)^{r-1} \) then the probability that it would be the \( m \)-th active neighbor to push \( u \) over its threshold is \( p(1-p)^{m-1} \). With this assignment, each time a neighbor of \( u \) becomes active, the probability that \( u \) becomes active is \( p \). This is identical to the probability that a node would be added to a connected component in site percolation. So the distribution of bond percolation clusters is identical to the distribution of the nodes activated through the gWTM with this assignment for \( r_u \).

We now consider \( k \)-core percolation. This is sometimes referred to as “bootstrap percolation” [7, 12] but we reserve “bootstrap percolation” for another process described below. In \( k \)-core percolation, nodes with degree less than some fixed value \( k_0 \) are removed from the network. This may result in other nodes’ degrees going below \( k_0 \), and these in turn are removed. This is repeated until a network remains in which all nodes have degree at least \( k_0 \). We can model this using the gWTM as follows. Once \( k_0 \) is chosen, for each node \( u \), we set \( r_u = k_u - k_0 - 1 \) with the understanding that if \( r_u \leq 0 \) then \( u \) is initially active. As these nodes transmit to their neighbors, other nodes begin to activate. The process continues until no further activations or transmissions occur. The nodes that are still quiescent are identical to the \( k_0 \)-core of the network. This approach easily generalizes to the heterogeneous \( k \)-core [3] in which each node has its own threshold.

We now move to bootstrap percolation. Bootstrap percolation is defined as follows: We set \( r = r_0 \) for every node, and then activate a randomly chosen collection of nodes. More nodes begin to activate, and the final set of active nodes is the set of interest for bootstrap percolation. If we analogously define the heterogeneous bootstrap percolation model such that each node is assigned a threshold and activated if its number of active neighbors exceeds the threshold, then we arrive at the gWTM described in the introduction. However, in this context, it becomes easy to see that in fact this is the dual of heterogeneous \( k \)-core percolation. To see this, we set our
thresholds as in the heterogeneous $k$-core case, and take those with $r \leq 0$ to be the initially active nodes. The heterogeneous $k$-core is exactly those nodes that remain quiescent, while the result of the heterogeneous bootstrap percolation is exactly those nodes that eventually become active. This simple observation explains some of the commonalities in the transitions seen in $k$-core and bootstrap percolation [3].

We finally consider the **gWTM with transmission probability** $p < 1$. This can occur if, for example, active nodes remain active for a fixed finite period of time. If $r = 1$ for all nodes, this corresponds to the Reed–Frost model of SIR disease spread [1]. It is well-known that this model is equivalent to bond percolation [1, 13, 16, 21, 26, 35, 40], which we have seen above is a special case of the gWTM. We now consider the case where $r \neq 1$. Consider a node $u$ with degree $k_u$ and threshold $r_u$. The probability that the $m$-th activated neighbor would cause $u$ to reach its threshold $r_u$ is $p^{(m-1)}(1-p)^{r_u-1}$, that is $p$ times the probability that the first $m-1$ activated neighbors yielded $r_u - 1$ transmissions to $u$. We consider the usual gWTM on an identical network, but set the threshold of $u$ to be $m$ with probability $p^{(m-1)}(1-p)^{r_u}$. If we imagine that we know $r_u$ but that the actual assigned value $m$ is hidden from us, then each time a neighbor of $u$ is activated, the probability $u$ becomes active will be the same as in the model with $p < 1$. Perhaps surprisingly this shows that the class of problems we investigate is not increased by relaxing the assumption that active nodes eventually transmit (so long as $p$ is uniform). Thus without loss of generality we can take transmission probability to be 1.

Note that if activation duration is not constant, we would expect some nodes to be more likely to transmit than others. In this case the behavior is not a special case of the gWTM. This applies to any effect which would heterogeneously alter the probability a node transmits to its neighbors such that some transmit with higher probability than others. In Configuration Model networks, we would expect this to alter the probability of a large-scale cascade, but not the final proportion activated [16, 21, 26, 40, 43]. In contrast, if there is heterogeneity in probability of receiving transmission, this will alter the final size, but not the probability of a large-scale cascade. For networks with short cycles, either heterogeneity will alter both size and probability [27, 40].

Further note that although we find the final states to be the same whether we take $p < 1$ or take it to be 1 and change the assignment for $r$, the intermediate dynamics may be different.

**PRELIMINARIES FOR DERIVING THE EQUATIONS**

We now develop equations which predict the deterministic dynamics of the gWTM in Configuration Model networks. We begin by finding an equivalent question which we can investigate more directly. We make the assumption that the population size and initial condition are large and the population-scale dynamics as deterministic. This implies that the probability a random node has a particular state is the proportion of the population that has that state. In turn, we note that until a node is active, it has no impact on the state of its neighbors, and once active its neighbors have no impact on its state. Thus we arrive at three equivalent questions:

1. Given the initial conditions, what proportion of the population is quiescent or active at time $t$?
2. Given the initial conditions, what is the probability a random node $u$ is quiescent or active at time $t$?
3. If we choose a random node $u$ and prevent it from transmitting once active, given the initial conditions what is the probability it is quiescent or active at time $t$?

The first two are equivalent due to the assumption that the spread is deterministic [44]. The last two are equivalent due to the fact that until it is active the change to $u$ has no effect, and once it is active, the change has no effect on its own status. Consequently by answering the third question which involves an alteration of a single node, we arrive at the solution to the first question which does not involve any alteration. This third question is amenable to probability-based techniques.

This is the basis for the “test node” assumption [33]. We define a test node to be a randomly chosen node $u$ which is prevented from transmitting to others [45]. We can calculate the probability $u$ has a given state, and this in turn will tell us the proportion of the population in that state. This is equivalent to the “cavity state” used by [20]. By making the test node assumption we can ignore correlations that otherwise occur between two neighbors of $u$ due to transmissions passing through $u$.

**GWTM IN CONFIGURATION MODEL NETWORKS**

A Configuration Model network is generated by assigning each node a degree $k$ independently of the others and giving it $k$ stubs. Stubs are paired to create the edges. We use $P(k, r)$ to be the probability a node has a given $k$ and $r$.

A consequence of the formation process is that a random neighbor (neighbor) of a node has a given degree $k$ and threshold $r$ with probability $P_n(k, r) =...
Continuous time

We first consider a continuous time model, allowing transmission to occur at rate $\beta$ along any quiescent–active edge. Once the number of transmissions a node has received equals its threshold, it immediately activates. Following activation, it can immediately transmit, but does so as a Poisson process with rate $\beta$. The rate $\beta$ may be arbitrarily set to 1 because we have no other time scales. We set it to 1 in simulations but leave it in our equations.

We let $u$ be a test node and $v$ a random neighbor of $u$. We define $\theta(t)$ to be the probability $v$ has not yet transmitted to $u$. We break $\theta(t)$ into two parts, $\phi_Q(t)$, the probability $v$ has not yet transmitted and is quiescent, and $\phi_A(t)$, the probability $v$ has not yet transmitted but is active. We demonstrate the model graphically in figure 1. The probability $u$ is still quiescent is the probability $m < r$ neighbors of $u$ have transmitted to it.

$$Q(t) = \sum_k \sum_{r=0}^{k-1} \sum_{m=0}^{r-1} P(k,r) \binom{k}{m} \theta(t)^{k-m} (1 - \theta(t))^m$$

where $\psi_{r,CM}(x) = \sum_k P(k,r)x^k$. Later when we consider networks with triangles, there will be a related function $\psi_{r,\Delta}$. Note that the inner sum is the first $r$ terms of the Taylor Series of $\psi_{r,CM}(1)$ expanded at $\theta(t)$. We take advantage of this to reduce the summation to

$$Q(t) = \sum_{r>0} TS(\psi_{r,CM}, 1, \theta(t), r-1),$$

where $TS(g,x,x_0,n) = \sum_{j=0}^{n} g^{(j)}(x_0)(x - x_0)^j/j!$ represents the Taylor series for $g(x)$ centered at $x_0$ truncated at order $n$.

To complete this, we need an expression for $\theta(t)$. To find this, we adapt the test node assumption to $v$. We allow $v$ to transmit to $u$, but prevent it from transmitting to any others. The probability $v$ is quiescent is

$$\phi_Q(t) = \sum_k \sum_{r=0}^{k-1} \sum_{m=0}^{r-1} kP(k,r) \binom{k-1}{m} \theta(t)^{k-1-m} (1 - \theta(t))^m$$

$$= 1 - (\frac{1}{K}) \sum_{r>0} \sum_{m=0}^{r-1} \frac{\psi_{r,CM}^{(m+1)}(\theta(t))}{m!} (1 - \theta(t))^m$$

$$= 1 - (\frac{1}{K}) \sum_{r>0} TS(\psi_{r,CM}', 1, \theta(t), r-1),$$

with $\phi_A(t) = \theta(t) - \phi_Q(t)$. To find $\theta(t)$, we simply observe that

$$\dot{\theta} = -\beta \phi_A.$$

Substituting for $\phi_A$ in terms of $\theta$, our full system of equations is

$$\dot{\theta} = -\beta \left( \theta - \frac{1}{\langle K \rangle} \sum_{r>0} TS(\psi_{r,CM}', 1, \theta(t), r-1) \right)$$

$$Q = \sum_{r>0} TS(\psi_{r,CM}, 1, \theta(t), r-1)$$

$$A = 1 - Q.$$
phase transition threshold, correlation lengths diverge (in $k$-core percolation for $N \to \infty$) \cite{12}. This long correlation magnifies stochastic effects, so close to this threshold the simulations diverge somewhat from the predictions.

**Discrete time**

We now consider a discrete time model, with synchronous updating. That is, at each time step, all nodes with at least as many active neighbors as their threshold simultaneously become active, and the process repeats for the next time step. We use $t = 0, 1, \ldots$ to denote the discrete time and we find that the expressions for $Q(t)$, $\phi_Q(t)$, and $\phi_A(t)$ in terms of $\theta(t)$ remain the same as in the continuous time model. However, the rule for how $\theta$ updates must change:

$$
\theta(t) = \phi_Q(t - 1) = \frac{1}{(K)} \sum_{r > 0} TS(\psi_{r,\text{CM}}, 1, \theta(t - 1), r - 1),
$$

for $t = 1, 2, \ldots$ and $\theta(0) = 1$. We will be interested in how the results change as $\rho$ changes. To simplify our notation, we define $f(x)$ to be the right hand side of this

$$
f(x) = \frac{1}{(K)} \sum_{r > 0} TS(\psi_{r,\text{CM}}, 1, x, r - 1).
$$

This has a dependence on $\rho$ through $\psi_{r,\text{CM}}$. When we want to make the dependence on $\rho$ explicit, we write $f(x|\rho)$. Assuming that the initial active nodes are chosen randomly, we have $f(x|\rho) = (1 - \rho)f(x|0)$.

Note that the final size relation derived in the continuous dynamics case can be expressed as $\theta = f(\theta)$. The final value $\theta(\infty)$ will be the same for this model and the continuous model. This is because in both models any node that becomes active will eventually transmit to all of its neighbors. The timing of those transmissions is irrelevant to their effect on the recipient. So the final state is the same.

The dynamics resulting from equation (1) can be understood through the cobweb diagrams in figure 3. Here we plot $f(\theta)$ and the diagonal line $\theta(t + 1) = \theta(t)$. The cobweb diagram allows us to graphically iterate $f$ to find successive values of $\theta$.

This cobweb diagram gives insight into the phase transition. We can use geometric arguments to show that as $\rho$ approaches the critical value $\rho_c$ from below, the resulting value of $\theta(\infty)$ scales like $\theta(\infty) - \theta_c \sim (\rho_c - \rho)^{1/2}$. We can rotate and flip the coordinates such that the diagonal $\theta(t + 1) = \theta(t)$ is the horizontal axis and then $f(\theta)$ becomes locally a parabola that crosses the (new) horizontal axis very close to its local minimum. By appropriately scaling and choosing $\theta$ in the new coordinates, we can represent this parabola as $x^2 - c$. The parabola

FIG. 2: The gWTM in a network having $P(2) = P(4) = P(6) = 1/3$. A random fraction $p$ is initially active. The other nodes either have fixed $r = 2$ (left) or fixed proportion $r = k/2$ (right). Solid curves are simulations in a population of 400000, dashed curves are theoretical predictions. Note the discontinuous jump in final sizes at a critical value of $p$. Close to the critical value, the dynamics are sensitive to stochastic effects, but elsewhere the predictions match well.
We can alternately interpret the discrete model in terms of transmission chains of given lengths. We choose a node $u$ and a random neighbor $v$. We prevent $u$ from transmitting to $v$ and $v$ from transmitting to its neighbors other than $u$. We allow that the possibility a random neighbor of $v$ transmits to $v$ might be different from the probability $v$ transmits to $u$ (ultimately we will see that the values are the same, but for now we allow this possibility). If $\theta_u$ is the probability $v$ does not transmit to $u$ then in fact it is the probability $v$ remains quiescent (otherwise it would eventually transmit). If $\theta_v$ is the probability a random other neighbor of $v$ does not transmit to $v$, then we get

$$\theta_u = \frac{1}{\langle K \rangle} \sum_{r>0} TS(\psi'_{r,CM}, 1, \theta_v, r-1).$$

We see that if $r_v = k_v$, even if all neighbors other than $u$ transmit to $v$, that might not be enough to push $v$ over its threshold, so $v$ will not transmit to $u$. Note that this implies that if every node has a threshold at least 1 less than its degree, then in the cobweb diagrams $f(\theta)$ goes through 0 at $\theta = 0$ [46].

We can derive an equivalent expression to equation (2) for $\theta_c$ in terms of the neighbors of $v$, and for those neighbors in terms of their neighbors. Continuing this, we can define $\theta(t)$ to be the probability that a random neighbor of $u$ will eventually transmit to $u$ if we discard all nodes of distance greater than $t$ from $u$. Then $\theta(t)$ follows the same recurrence seen before

$$\theta(t) = \frac{1}{\langle K \rangle} \sum_{r>0} TS(\psi'_{r,CM}, 1, \theta(t-1), r-1),$$

reproducing equation (1).

Threshold condition

We now derive the threshold condition for an arbitrarily small value of $\rho$ initiating a cascade. Consider the cobweb diagram of figure 3. In the limit $\rho \rightarrow 0$, we find $f = 1$ at $\theta = 1$. A global cascade is possible in this limit only if $f'(1) > 1$. Note that $TS(\psi'_{r,CM}, 1, x, r-1) = \psi'_{r,CM}(1) + O((1-x)^r)$ because it is the first $r-1$ terms of the Taylor Series for $\psi'_{r,CM}(1)$ centered at $x$. It follows that only the $r = 1$ term in equation (1) gives a nonzero contribution to the slope of $f(\theta)$ at $\theta = 1$ for $\rho \rightarrow 0$. The leading order term of $f'(1)$ in the $\rho \rightarrow 0$ limit is $\psi''_{1,CM}(1)/\langle K \rangle$. So only when $\psi''_{1,CM}(1)/\langle K \rangle > 1$ can a global cascade begin from an arbitrarily small randomly chosen initial fraction $\rho$. Because $\psi''_{1,CM}(1)/\langle K \rangle > 1$ can a global cascade begin from an
arbitrarily small randomly chosen initial condition then the \( r = 1 \) nodes form a giant connected component of the population.

We can also derive this threshold through a physical interpretation. If the initial \( \rho \) is small, then the condition for a cascade is the same as the condition that the “infection” travels infinitely far from an initial source. Each active node will transmit to all of its neighbors, but until the cascade grows, we may assume that the network is tree-like. So the transmission leads to activation iff the neighbor has \( r = 1 \). Thus the disease explores the largest component made up of \( r = 1 \) nodes around the initial node. If the \( r = 1 \) nodes percolate the network, then there is a cascade for arbitrarily small \( \rho \). Otherwise, there is not. This generalizes theorem 5 of \cite{2} where the same result was proven under the assumption that the threshold \( r_u \) is a function of the degree \( k_u \) of \( u \) (although the proof here has been less rigorous).

**Sufficient condition for the hybrid phase transition**

We now consider the alternate question of under what condition can there be a hybrid phase transition? In general this appears to be a difficult problem. However, if we assume that every node’s threshold \( r_u \) satisfies \( r_u \leq k_u - 1 \), we can make some progress. In this case, for \( \rho = 0 \), there is a fixed point at 0 and at 1. The slope of \( f(\theta|0) \) at \( \theta = 0 \) is given by

\[
\frac{1}{\langle K \rangle} \sum_{r>0} \left[ \frac{d}{d\theta} \left( \psi_{r,CM}^{(1)}(0,1,\theta, r-1) \right) \right]_{\theta=0} = \frac{1}{\langle K \rangle} \sum_{r>0} \sum_{m=0}^{r-1} \left( \frac{d}{d\theta} \left( \psi_{r,CM}^{(m+1)}(0) \right) (1 - \theta(0))^m \right)_{\theta=0} = \frac{1}{\langle K \rangle} \sum_{r>0} \left( \psi_{r,CM}^{(r+1)}(0) \right) / (r-1)! = \frac{1}{\langle K \rangle} \sum_{r>0} P(k = r + 1, r)(r + 1)k
\]

If \( \sum P(k, r - 1)k(k - 1) < \langle K \rangle \), then the slope at \( \theta = 0 \) is less than 1. That is, if the nodes with \( r = k - 1 \) do not form a giant component the slope at \( \theta = 0 \) is less than 1 \cite{17}. If additionally there is no cascade for arbitrarily small \( \rho \), then the derivative of the curve is less than 1 at \( \theta = 1 \). So if \( r_u \leq k_u - 1 \) for every node \( u \) and neither the \( r = 1 \) nodes nor the \( r = k - 1 \) nodes form a giant component, then for \( \theta \) close to 0, the curve is below the diagonal, while for \( \theta \) close to 1 it is above the diagonal \cite{48}. This forces the existence of at least one additional fixed point strictly between 0 and 1. Take the largest of these. As \( \rho \) increases, this fixed point moves right, while the fixed point at 1 moves left. Eventually these two meet and annihilate in a saddle-node bifurcation, resulting in a hybrid phase transition. We see this in figure 3 for which \( k = 4 \) and \( r = 2 \) for all nodes.

In fact, if both of these conditions hold, then it is likely that after the hybrid phase transition, all nodes become active. The fixed point at 0 remains at 0 for all \( \rho \). Unless there are additional fixed points below \( \theta_c \) when the bifurcation occurs, the system will immediately move to the only remaining fixed point, \( \theta = 0 \). In this case, all edges eventually transmit, and all nodes eventually become active.

More generally, as we see in figure 3 we can have a cascade even when \( r_u \geq k_u \) for some nodes. If there is no cascade from arbitrarily small \( \rho \) and there is any value of \( \rho \) for which \( f(\theta|\rho) \) goes below and then above the diagonal, then at a larger value of \( \rho \) this bifurcation will occur. This is what we see in figure 3. In this case, the cascade fails to reach the entire population.

We can generalize our sufficient condition somewhat. If we remove the \( r \geq k - 1 \) nodes from the network, we are left with a new configuration model network. Some nodes may now have a threshold \( r \) greater than or equal to their new \( k - 1 \). If we repeat this process until no more nodes are removed, and the resulting core network has a giant component, then we will satisfy the condition for a hybrid phase transition.
We now show that the hybrid phase transition requires that \( r > 1 \) for at least some nodes. Physically this makes sense because if \( r = 1 \) for all nodes, then any node in a connected component containing an initially active individual will eventually activate. We then find another necessary condition on \( f \) which is harder to translate back into \( r \).

When the saddle-node bifurcation occurs, the derivative of \( f(\theta|\rho) \) must decrease through 1 at the bifurcation point. This means that it must have a negative second derivative. Since \( f(\theta|\rho) = (1 - \rho)f(\theta|0) \), this means that \( f(\theta|0) \) must have negative second derivative somewhere. Taking the second derivative of \( f(\theta|0) \) gives

\[
\sum_{r>0} \sum_{m=0}^{r-1} \frac{1}{m!} \left( \psi_{r,CM}^{(m+3)}(\theta)(1 - \theta)^m - m\psi_{r,CM}^{(m+2)}(\theta)(1 - \theta)^{m-1} \right.
\]

\[
+ m(m-1)\psi_{r,CM}^{(m+3)}(\theta)(1 - \theta)^{m-2} \bigg) .
\]

Because \( \psi \) is defined as a summation with positive coefficients, each derivative of \( \psi \) is non-negative. In order for the expression above to be negative, the middle term, \( m\psi_{r,CM}^{(m+2)}(\theta)(1 - \theta)^{m-1} \) must be nonzero for at least one value of \( m \). If \( r \leq 1 \) for all nodes, then when \( r < 1 \), the sum is empty and when \( r = 1 \) the only term in the sum over \( m \) has \( m = 0 \), so the term is zero. So a hybrid phase transition in a configuration model network requires \( r > 1 \) for some nodes. With minor modifications, this shows that site percolation (on configuration model networks) cannot exhibit this phase transition.

Note that we can also show that if having a cascade requires \( \rho \) above some positive threshold, then \( f''(\theta|0) \) must change sign between 0 and 1. This is because just before the bifurcation happens, there must be (at least) three solutions to \( \theta = f(\theta|\rho) \). The top of these three has \( f' < 1 \), the middle has \( f' > 1 \) and the bottom has \( f' < 1 \). Since the average second derivative must be positive in one interval and negative in the other, it must have a sign change somewhere. It is not clear if this leads to any simple statements about necessary conditions for a hybrid phase transition.

**TRIANGLE-BASED NETWORKS**

In [28, 37], Newman and Miller independently introduced a model of random clustered networks. In these networks, nodes are assigned two degrees, an independent degree \( k_l \) and a triangle degree \( k_\Delta \) with probability \( P(k_l, k_\Delta) \). A node is then given \( k_l \) regular stubs and \( k_\Delta \) triangle stubs. Regular stubs are joined into pairs to define edges, while triangle stubs are joined into triples to form triangles, and so a node will have degree \( k_l + 2k_\Delta \).

We again take \( u \) to be a test node. We consider a triangle involving \( u, v, \) and \( w \). Taking \( \xi(t), \xi_* (t), \) and \( \xi**, (t) \) to be the probabilities that the neighbors in the triangle have combined to transmit 0, 1, or 2 times to \( u \) we have

\[
Q(t) = \sum_{r>0} \sum_{m=0}^{r-1} \left( \frac{\psi_{r,CM}^{(m)}(\xi)}{m!} \sum_{d=0}^{r-m-1} \left( \frac{m}{d} \right) \xi_*^{m-d} \xi_**^d \right) .
\]

To interpret this equation, note that the sum over \( r > 0 \) considers all thresholds for nodes which are not initially
the probability that \( w \) is quiescent given that \( u \) and \( v \) are prevented from transmitting to \( w \), and \( \delta_1 \), the probability that \( w \) would still be quiescent even if \( v \) has transmitted to it (and \( u \) has not). These are

\[
\delta_0 = \frac{1}{(K_\Delta)} \sum_{r \geq 0} \sum_{m = 0}^{r-1} \left( \frac{\psi_{r,\Delta}^{(m+1)}(\xi)}{m!} \right) \sum_{d = 0}^{r-m-1} \left( \frac{m}{d} \right) \xi^{m-d} \xi_d^{(*)} \\
\delta_1 = \frac{1}{(K_\Delta)} \sum_{r \geq 0} \sum_{m = 0}^{r-2} \left( \frac{\psi_{r,\Delta}^{(m+1)}(\xi)}{m!} \right) \sum_{d = 0}^{r-m-2} \left( \frac{m}{d} \right) \xi^{m-d} \xi_d^{(*)} .
\]

We now introduce some auxiliary variables which help to find the \( \xi \) variables. We assume \( u \) and \( v \) are prevented from transmitting to \( w \). We define \( \zeta_Q \) to be the probability that \( w \) is quiescent, \( \zeta_A \) the probability it is active but has not transmitted to \( u \) or \( v \), \( \zeta_{A,u} \) the probability it has transmitted to \( u \) but not \( v \), \( \zeta_{A,v} \) the probability it has transmitted to \( v \) but not \( u \), and \( \zeta_{A,uv} \) the probability it has transmitted to both. Figure 7 demonstrates the flow of these variables. We have

\[
\zeta_Q = \delta_0 \\
\zeta_{A,u} = \beta \zeta_A - \beta \zeta_{A,u} \\
\zeta_{A,v} = \beta \zeta_A - \beta \zeta_{A,v} \\
\zeta_{A,uv} = \beta (\zeta_{A,u} + \zeta_{A,v}) \\
\zeta_A = 1 - \zeta_s - \zeta_{A,u} - \zeta_{A,v} - \zeta_{u,v} .
\]

We now introduce our final set of variables. We define \( \phi_{AB} \) to be the probability of having one neighbor of status \( A \) and the other of status \( B \). We use \( Q^* \) in the subscript of \( \phi \) to denote a quiescent neighbor that has received transmission from the other neighbor. We use \( * \) or \( ** \) in the superscript of \( \phi \) to denote that \( u \) has received 1 or 2 transmissions from the neighbors in the triangle. We can find most of these variables in terms of the \( \zeta \) variables. The others (\( \phi_{AA}, \phi_{AA}', \) and \( \phi_{AA}'' \)) can be found in terms of the \( \zeta \) variables, following figure 6.

\[
\phi_{QQ}(t) = \delta_0^2 = \zeta_Q^2 \\
\phi_{QA}(t) = 2\delta_0 \zeta_A \\
\phi_{QA'}(t) = 2\delta_1 \zeta_{A,u} \\
\phi_{QA''}(t) = 2\delta_0 \zeta_{A,u} \\
\phi_{A}(t) = \zeta - \phi_{QQ} - \phi_{QA} - \phi_{QA'} \\
\phi_{A'}(t) = \zeta_s - \phi_{QA} - \phi_{QA'} \\
\phi_{A''}(t) = \zeta_s .
\]

We can now write down our final differential equations for the derivatives of the \( \xi \) variables

\[
\dot{\xi} = -\beta (\phi_{QA} + \phi_{QA'} + 2\phi_{AA}) \\
\dot{\xi}_s = \beta (\phi_{QA} + \phi_{QA'} + 2\phi_{AA}) - \beta \phi_{AA}' \\
\dot{\xi}_s = \beta \phi_{AA} .
\]
We now have a closed system of equations. We could replace the $\xi^*$ equation with $\xi^* = 1 - \xi - \xi_*$. The generalization of our approach to nonzero $k_I$ would be straightforward but technical.

**Discrete time**

We now consider the discrete time version of the spread in these triangle-based networks. The dynamics are simplified because we do not need to consider cases where a node has transmitted to some, but not all, of its neighbors.

The non-ODE equations remain the same except that $\zeta_{A,v} = \xi_{A,v} = 0$, which implies $\phi_{Q,A} = 0$. The ODEs change. We have $\zeta_A(t) = 1 - \delta_0(t) - \zeta_{A,u,v}(t)$ and

$$
\begin{align*}
\xi(t+1) &= \xi(t) - \phi_{Q,A}(t) - \phi_{AA}(t) \\
\xi_*(t+1) &= \xi_*(t) + \phi_{QA}(t) - \phi_{AA}^* \\
\zeta_{A,u,v}(t+1) &= 1 - \delta_0(t).
\end{align*}
$$

Once we have $\zeta_{A,u,v}(t+1), \xi(t+1),$ and $\xi_*(t+1)$ we can build up all of the other $\phi$ variables at time $t+1$. Then we can update for the next time.

**Threshold**

To understand the threshold, the initial analytic method presented for the configuration model case becomes more complicated, so we consider just the second method, calculating the probability that the initial node manages to activate others arbitrarily far from it.

Consider a triangle containing a single active node $u$ early in the spread and determine the probability that the activation results in 1 or 2 further transmissions to others $v$ and $w$ in the triangle. It will transmit to both of them directly, but it will only directly cause activation if $r = 1$. A neighbor has $r = 1$ with probability $q_1 = \sum_{k_\Delta} k_\Delta^2 P(k_\Delta,1)/\langle K_\Delta \rangle = \psi_{1,\Delta}(1)/\langle K_\Delta \rangle$. This results in an additional $k(k-1)P(k,1)\sum kP(k,1) = \psi_{1,\Delta}(1)/\psi_{1,\Delta}(1)$ triangles with disease introduced for each $r = 1$ neighbor. So from the two other nodes in the triangle, there are an expected $2q_1\psi_{1,\Delta}(1)/\langle K \rangle$ new triangles with a single introduced activation because of the nodes having $r = 1$.

However, there is an additional case to consider. If one neighbor has $r = 1$ and the other has $r = 2$, then the $r = 2$ node will become active. Early in the spreading, an $r = 2$ neighbor will eventually become active from transmissions within the triangle iff the other neighbor has $r = 1$. Thus the probability that one node has $r = 2$ and becomes active is the probability that one has $r = 2$ and the other has $r = 1$. That is, $2q_1q_2$, where $q_2 = \sum k_\Delta kP(k_\Delta,2)/\langle K_\Delta \rangle$. The resulting number of triangles from the $r = 2$ node is $\sum_{k_\Delta} k_\Delta(k_\Delta - 1)P(k_\Delta,2)/\langle K_\Delta \rangle$. So the expected number of new triangles coming through $r = 2$ nodes is $2q_1\psi_{2,\Delta}(1)/\langle K_\Delta \rangle = 2\psi_{1,\Delta}(1)\psi_{2,\Delta}(1)/\langle \langle K_\Delta \rangle \rangle^2$. 

FIG. 8: Comparison of simulations (solid) and predictions (dashed) for different sized initial conditions in a network with $P_\Delta(1,2) = P_\Delta(2,2) = P_\Delta(3,2) = 1/3$ (left) and $P_\Delta(1,1) = P_\Delta(2,2) = P_\Delta(3,3) = 1/3$ (right) in populations of 400000 nodes. Note that the degree of a node is twice its number of neighbors.

FIG. 9: The flow diagram for the discrete time (synchronous update) random clustered network model. At each time step any active node transmits to all of its neighbors. The diagram is somewhat simpler than in the continuous time (asynchronous update) case.
So the epidemic threshold occurs when
\[
2 \frac{\psi''_{1,\triangle}(1)}{\langle K_\triangle \rangle} + 2 \frac{\psi'_{1,\triangle}(1)\psi''_{2,\triangle}(1)}{\langle K_\triangle \rangle^2} = 1,
\]
with a population-scale cascade possible if the left hand side is larger. If \( r \) values are assigned independently of \( k \), this reduces to
\[
2P(r = 1)(1 + P(r = 2)) \frac{\langle K_\triangle^2 - K_\triangle \rangle}{\langle K_\triangle \rangle} = 1.
\]
This condition is consistent with that found by [14].

**Condition for hybrid phase transition**

Because of the added variables, it is much more difficult to prove the existence of an “interior” fixed point and the saddle-node bifurcation. We will not attempt to prove as strong of a result here. We anticipate that the conditions for the hybrid phase transition will be similar.

We assume that there is no cascade in the \( \rho \to 0 \) limit. We consider a configuration model network for which \( k \) has the same distribution as \( k_\triangle \). The active proportion in the configuration model is less than that of the corresponding triangle network. This is because being joined to two nodes in a triangle increases the probability that at least one transmission has come, and it also opens the possibility of two transmissions. If this configuration model network has a hybrid phase transition, then the triangle-based network must as well. This is a fairly crude bound.

**IMPACT OF CLUSTERING**

To investigate the role of clustering, we want to compare the spread in triangle-based networks and configuration model networks with the same degree distribution. Note that in a triangle-based network, a node with a given \( k_\triangle \) has degree \( 2k_\triangle \), so in the configuration model networks, \( k \) has the same distribution as \( 2k_\triangle \) in the triangle-based networks. We find \( \psi_{r,CM}(x) = \psi_{r,\triangle}(x^2) \).

The condition for a cascade from arbitrarily small \( r \) in a configuration model network is that \( \psi''_{1,CM}(1)/\langle K \rangle > 1 \). In this case, we have a cascade in the Configuration Model network if
\[
1 < \frac{\psi''_{1,CM}(1)}{\langle K \rangle}
= \frac{2\psi'_{1,\triangle}(1) + 4\psi''_{1,\triangle}(1)}{2\langle K_\triangle \rangle}
= \frac{\psi'_{1,\triangle}(1) + 2\psi''_{1,\triangle}(1)}{\langle K_\triangle \rangle}.
\]

If the initially active nodes are randomly chosen, \( \psi_{1,\triangle}(1) = P(r = 1) \langle K_\triangle \rangle \) and \( \psi_{1,\triangle}(1) = P(r = 1) \langle K_\triangle^2 - K_\triangle \rangle \). So this becomes
\[
1 < 2P(r = 1) \frac{\langle K_\triangle^2 - K_\triangle \rangle}{\langle K_\triangle \rangle} + P(r = 1).
\]

If we instead consider the threshold for a corresponding triangle-based network, it is
\[
1 < 2P(r = 1) \frac{\langle K_\triangle^2 - K_\triangle \rangle}{\langle K_\triangle \rangle} + 2P(r = 1)P(r = 2) \frac{\langle K_\triangle^2 - K_\triangle \rangle}{\langle K_\triangle \rangle}.
\]

If \( P(r = 2) = 0 \), then cascades from arbitrarily small initial conditions are inhibited in the triangle-based network compared to the configuration model. However, if \( 2P(r = 2) \langle K_\triangle^2 - K_\triangle \rangle / \langle K_\triangle \rangle > 1 \), cascades from arbitrarily small initial conditions are enhanced in the triangle-based network in the sense that the threshold is reduced.

We can interpret this result physically by considering an \( r = 1 \) node \( u \) which receives transmission from a neighbor \( v \) early in the spread. We assume \( u \) and \( v \) share a common neighbor \( w \). In the absence of \( r = 2 \) nodes, \( u \) cannot effectively transmit to \( w \) because \( w \) is either already activated by \( v \) (if \( r_w = 1 \)) or still requires multiple transmissions to activate (if \( r_w > 2 \)). In contrast in an unclustered network, if \( w \) has the same degree, then it will have one more neighbor it can transmit to, which may have \( r = 1 \).

* A simple example where clustering inhibits spread

We can find a simple case where clustering inhibits cascade spread. If we set \( r_u = k_u - 2 \) for every node, then no triangle can be invaded. More generally if a network has a \( k_0 \)-clique and every node in this clique has \( r_u > k_u - k_0 \), then the clique cannot be invaded. Thus if each node's \( r_u \) is set to

**DISCUSSION**

We have investigated the spread of complex contagions through static networks, focusing on a generalized version of the Watts Threshold Model (gWTM). We have unified many disparate results about this and related models under a common framework, and derived some new results. We see that a wide range of percolation processes (site percolation, bond percolation, \( k \)-core percolation, bootstrap percolation, and even a version of the gWTM in which transmission occurs with fixed probability \( p < 1 \)) can be thought of as a special case of the gWTM.
We have adapted the edge-based compartmental modeling approach from infectious disease modeling to study cascades in a generalized Watts Threshold Model. We find that the approach leads to a compact system of equations which correctly predict the dynamic spread of a complex contagion within Configuration Model and triangle-based networks (of the variety introduced by Newman [37] and Miller [28]).

In Configuration Model networks We find that activation of an arbitrarily small initial proportion can lead to activation of a large fraction of the population iff those nodes who require only a single transmission to become active (i.e., \( r = 1 \)) form a giant component. In triangle-based networks \( r = 2 \) nodes can contribute to cascades from a small initial active proportion.

For configuration model networks, we have found a sufficient condition for a hybrid phase transition to occur when there is a sufficiently large initially active population. If

1. No node has an activation threshold \( r_u \geq k_u \)
2. \( r = 1 \) nodes do not form a giant component, and
3. \( r = k - 1 \) nodes do not form a giant component,

then there is a threshold concentration of initially active individuals which will lead to a cascade.

Limitations

The triangle-based networks we study allow for analytic methods. However, it comes at a price that the structure of the networks is restricted. Triangles that share edges are very rare. If we consider \( k \)-clique percolation in other clustered networks and find a large component and \( r \leq k - 1 \) for all nodes in this component then if one \( k \)-clique in the component becomes fully active, the process will spread throughout the component. The “cluster-seeding” which is often used in simulations where a single node and all of its neighbors are activated for the initial condition will lead to this sort of behavior. This process will not be captured by our random clustered network model. In essence, some of the behavior of complex contagions in more general clustered networks is believed to be a consequence of “wide” bridges. This is missing in our clustered networks.

Possible extensions

It is easy to adapt this model to the case where initial active nodes are targeted by degree. This simply involves modifying the choice of \( P(k, r) \), noting that \( r = 0 \) corresponds to the initially active nodes. This appears as a change in \( \psi_{\text{CM}}(x) \), but we then cannot assume that \( \psi \) is proportional to \( 1 - \rho \).

We can also adapt this to account for biased mixing, where the degree of a node provides information about the degree of its neighbors. We assume we know \( P_n(k'|k) \), that is the probability a neighbor has degree \( k' \) given that a node has degree \( k \). We define \( \theta_k \) to be the probability that a degree \( k \) node’s neighbor has not yet transmitted to it. Our function \( \psi \) would then become a summation of \( P(k)\theta^u_k \) than a function of a single variable \( \theta \). Closely related models have been studied by [24].

We could also adapt this to account for a network which changes in time. Assuming for example that existing edges end at some rate \( \eta_1 \), and then the freed stubs seek out new neighbors at another rate \( \eta_2 \), the methods of [33] lead us to the new model equations. A particularly interesting limit of this would have \( \eta_2 \rightarrow \infty \) such that nodes immediately find new neighbors when their old edges end.

It would be straightforward to add a recovered class to this model, much as has been done previously in SIR disease models. Some of the relevant calculations have already been performed by [39]. We could also consider the possibility of nodes transmitting multiple times. This would involve subdividing \( 1 - \theta \) into more components based on the number of transmissions that have occurred, much like the \( \xi_* \) and \( \xi_{**} \) components that occurred in the triangle-based network model.

We can adapt this approach to consider multiple competing processes spreading as done by [18, 31, 36]. May be interesting for political opinion. For understanding the formation of political groups, it would be interesting to consider 4 beliefs spreading: \( A_1, A_2, B_1, \) and \( B_2 \) where the two \( A_i \) are incompatible and the two \( B_i \) are incompatible, while the \( A \) and \( B \) processes are independent, with the population rewiring and preferentially selecting neighbors with at least one common belief.

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If the initial conditions were sufficiently small that stochastic effects could alter the dynamics, then the probability that \( u \) is activated is the average probability. In contrast, the probability that \( u \) transmits to \( v \) is not independent, thus to calculate the probability of a cascade initialized by \( u \) we must account for these dependencies.

If a cascade occurs, the probability that two neighbors \( v \) and \( w \) transmit to \( u \) are iid variables, so all that will matter to the calculation of the probability \( u \) is activated is the average probability. In contrast, the probability that \( u \) transmits to \( v \) and \( w \) are not independent, thus to calculate the probability of a cascade initialized by \( u \) we must account for these dependencies.

If the initial conditions were sufficiently small that stochastic effects could alter the dynamics, then the probability that \( u \) has become active by time \( t \) given the initial conditions depends not just on the proportion active at time \( t \) in a given realization, but also the distribution of sizes of different realizations.

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dynamics because when we assume that the dynamics are deterministic at the population scale, a single node’s random status cannot be important.

This interpretation explains a potentially puzzling feature of cobweb diagrams in figure 3, namely that, for example, if we take $\theta(t - 1) = 0$, we find $\theta(t) > 0$. Previously the interpretation of this statement would be that if the probability a neighbor has transmitted prior to time $t - 1$ was 1, then the probability it has transmitted prior to the later time $t$ is less than 1, which of course is nonsensical since the probability an event has not happened can only decrease in time. This does not present a problem for the previous analysis because given an initial value of $\theta(0) = 1$, the system never reaches this scenario.

Note that in figure 3, the slope of $f(\theta)$ at $\theta = 0$ is 0 because $P(k, k - 1) = 0$. The value of $f$ at $\theta = 0$ is nonzero because $r_u$ is not less than $k_u$ for all nodes.

Technically we require that the concentration of $r = 1$ or $r = k - 1$ nodes must be strictly away from the threshold to form a giant component such that the inequalities are strict. Otherwise $k = 2,\ r = 1$ for all nodes provides a counterexample to this result.