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# Universality of Spreading Processes with Spontaneous Activity

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When one forwards a funny email or retweets a viral tweet, one participates in a spreading process unfolding on a social network. Spreading processes on networks are ubiquitous in both human and natural systems: power failures can cascade between electrical network substations, action potentials ripple through neural networks, radioisotope tracers traverse predation networks, and computer worms burrow through computer networks [1]. Even processes embedded in space, such as the percolation of a fluid through porous rock, is a type of spreading on a network.

Although it might be hard to imagine that such a disparate collection of processes might have any unifying structure governing their spread when the underlying systems are so distinct, a truly marvellous fact is that all of them exhibit a phase transition as the probability of transmission between nodes is increased. To use the language of epidemiology, when the probability of transmission is low outbreaks are almost always finite (see Fig. 1a and Fig. 1e "sub-critical"). However, when the reproduction number (i.e., the average number of new infections caused by an infected node) is one, it becomes possible for epidemics (outbreaks extensive with the system size) to occur (see Figs. 1b, 1d). Of course, being a well-behaved phase transition, the vicinity of this critical transmission probability is characterised by various scale-free phenomena governed by critical exponents that depend only on the dimensionality and topology of the underlying network, thereby defining a shared universality class [1].

Quite generically, the universality class for re-excitable nodes was understood to be that of directed percolation.

**Summary**

**Spontaneous activation in spreading processes changes the universality class from directed percolation to undirected percolation.**

Directed percolation is a broad universality class, encompassing the network spreading processes already mentioned, along with models of wildfires, catalytic chemical reactions, and Reggeon field theory (for a review, see [2]). It is characterised by local interactions and a transition from an active state to a unique absorbing state (see Fig. 1a). In the context of disease spreading, a unique absorbing state exists for the disease of small-pox: no humans are infected with the disease and it will never reappear. However, the requirement of a unique absorbing state is not strictly satisfied for many spreading processes.

For many spreading processes, random initiation drives recurring outbreaks which then lapse back into the "absorbing" quiescent state (see Fig. 1c). For instance, neurons cultured in the lab self-organize to a near-critical point, which we know from observing spontaneous scale-free neuronal avalanches. Initially, these were observed with directed percolation exponents [3]. However, the very fact that we see multiple avalanches indicates that these neurons cannot strictly belong to the universality class of directed percolation, and recent work has called the underlying universality class into question [4]. Even in the case of small-pox, one could envision an unenviable future in which it is released from research laboratories and the "absorbing" state is shown to be spongy. Therefore, directed percolation is only a good approximation for systems in which there is a good separation between the time-scale of initiation of *ex nihilo* activity and the spreading and cessation of that activity.

To study what happens when spontaneous activity is considered, we have introduced a discrete-time model that has both a spreading parameter and a probability of spontaneous activation baked in [5]. In each time-step, each

1. Daniel Korchinski received 1st place in the CAP Best Student Oral Presentation competition at the 2019 CAP Congress at Simon Fraser University in Burnaby, BC.

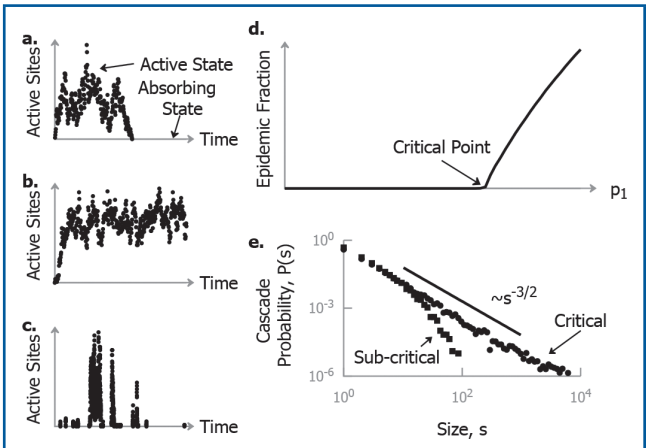


Fig. 1 The behaviour of the directed-percolation transition. **a.** The number of active sites starting from a single infected node below the critical point, exhibiting a fluctuating active phase that returns to a quiescent absorbing state. **b.** Above the critical point, a single infected node can lead to an eternal fluctuating steady state. **c.** Here, nodes are randomly reactivated, but a clear separation of time-scales continues to drive scale-free cascades, destroying the absorbing state. **d.** The fraction of nodes that belong to the largest cluster shows a clear phase transition as the spreading parameter $p_1$ is increased. **e.** The cascade distribution becomes scale-free at the critical point labelled in panel d.

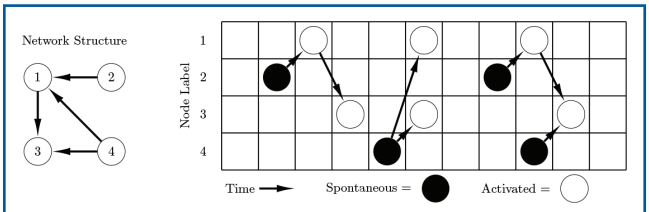


Fig. 2 An illustration of a spreading process on a simple network with spontaneous activation. (Left) A directed network consisting of four nodes and four edges. (Right) A sample time series of activations. Spontaneous activations can be identified as those nodes that activate without any of their parents being active in the preceding time-step. When assuming a separation of time-scales, the first two cascades might be falsely labelled a single cascade because they are contiguous. Knowledge of the network structure allows us to disentangle independent cascades. Additionally, when there is no separation of time-scales, initially-independent streams of activity can merge, leading to cascades with multiple roots, as occurs in the third cascade.

node $i$ activates with a probability that depends only on the number of their parents $m_{i,t}$ (we consider networks with directed edges) that were active in the preceding time-step:

$$P_{i,t+1} = 1 - (1 - p_0)(1 - p_1)^{m_{i,t}} \qquad (1)$$

In the case that the spontaneous activation parameter $p_0 = 0$, we have a simple branching process, which falls into the universality class of directed percolation, while for $p_1 = 0$, we simply have dynamical percolation, belonging to the distinct universality class of undirected (isotropic) percolation (for a comparison, see [6]). Finding a well-defined critical point for $p_0 > 0$ is challenging. In directed percolation, there are several measures that could be used to define the critical point: it could be identified with a unity reproduction number, or with a divergence in the susceptibility of the active fraction, or as the point at which epidemics appear. However, when $p_0 > 0$, it's not so clear which measure to use. Since cascades merge and over-lap, nodes can have more than one parent (see Fig. 2), meaning the reproduction number, $\sigma$, (defined by the mean number of active daughters to an active node) is not simply $\sigma = p_1 \times$ (average number of outgoing connections) as it is in directed percolation. Further, the susceptibility no longer diverges, but instead attains a maximum on a Widom line [7]. Even defining cascades in the presence of spontaneous activity is challenging, because initially-independent cascades can merge together (see Fig. 2). It is necessary to use the network structure to separate cascades that overlap in time but not in space. However, if one considers cascades defined by causal clusters, as has been proposed in the neuroscience context [8], epidemics can be readily defined and observed for sufficiently large $p_1$. We found that all three of these measures, the reproduction number equalling one, the Widom line, and the appearance of epidemics, all disagree when $p_0 > 0$ (see Fig. 3a).

To determine which of these phase-lines correctly generalized the directed-percolation critical point we conducted numerical and analytical investigations of cascade statistics on all three phase-lines. We found that only the critical line associated with the appearance of epidemics produced the expected scale-free behaviour in the cascade distribution [5]. Additionally, for simple network structures, that line can independently be derived by identifying the set of points that cause the correlation length or average cluster size to diverge. However, in our investigation we also found something quite surprising on the critical line: a cross-over between two distinct sets of critical exponents (see Fig. 3b). Small cascades obey statistics $P(s) \sim s^{-1.5}$ that would correspond to the universality class of mean-field directed percolation; however, large cascades obey statistics corresponding to undirected percolation, $P(s) \sim s^{-2.5}$.

Nonetheless, all outbreaks on the critical line exhibit the same universal scaling $p(s) \sim s^{-3/2}\, g(s/s_m)$ with $s_m \sim p_0^{-2/3}$, for a cross-over scaling function $g(x)$ (see Fig. 3c). The transition between these regimes is set by the scale at which the merging of cascades occurs (see Fig. 3d). For systems with a lower rate of spontaneous activation, encountering another cascade is unlikely, and so the directed percolation exponents survive to larger sizes. The eventual dominance of the undirected percolation exponents is guaranteed because the isotropic correlation length diverges on

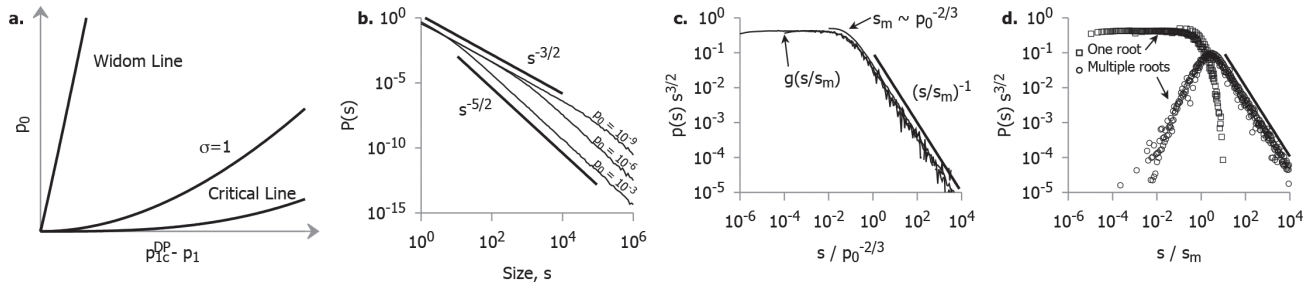


Fig. 3 Simulations of outbreaks on the critical line for a simple 10-regular network. **a.** The phase diagram for the spreading process with reactivation in the vicinity of the directed percolation critical point $p_{1c}^{DP}$. $\sigma$ denotes the reproduction number (i.e., average number of daughters). **b.** The outbreak distribution on the critical line is asymptotically scale-free, and exhibits a transition in the scaling exponent. **c.** The data in **b**, rescaled to exhibit a universal curve collapse, with $p(s) \sim s^{-3/2} g(s/s_m)$. The curve collapse explicitly gives the functional form of the cross-over function $g(s/s_m)$, showing that $g(x << 1) \sim x^0$ and that $g(x >> 1) \sim x^{-1}$. **d.** The data of **c**, partitioned into outbreaks with and without merging of activity, reveals that the exponent transition at $s_m$ is precisely when the merging of avalanches dominates.

the critical line, meaning that the anisotropy associated with the time-direction of the spreading process (i.e., the "directed" part of the associated percolation problem) must vanish. More simply, one cascade can merge with another one that was initiated either before or after it, restoring a time-symmetry that is not present in directed percolation, where activations always follow from their parent. This additional symmetry removes the absorbing state that defines directed percolation, lifting the universality class from directed to undirected percolation. We have tested the robustness of our findings on a variety of network topologies, including brain-connectome analogues, fat-tailed, and small-world networks, finding in each case numerical results that exhibit a transition between universality classes [5].

## CONCLUSION

The universality class of directed percolation appears in myriad places, but only when one assumes a separation of time-scales between the initiation and propagation of activity. When those time-scales mix, the cooperation of initiation and spreading changes the underlying universality class to one of undirected percolation. Characteristics of directed percolation survive only on the smallest scales, with exponents related to undirected percolation dominating asymptotically. Our work provides a bridge between two fundamental universality classes of non-equilibrium statistical physics and implies that spreading processes with spontaneous activity are fundamentally distinct from ordinary spreading processes.

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