Critical percolation in self-organized media:
A case study on random directed networks

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A minimal model for self-organized critical percolation on directed graphs with activating and
de-activating links is studied. Unlike classical self-organized criticality, the variables that determine
criticality are separated from the dynamical variables of the system and evolve on a slower timescale,
resulting in robust criticality. While activity of nodes percolates across the network, the network
self-organizes through local adjustment of links according to the criterion that a link’s adjacent
nodes’ average activities become similar. As a result, the network self-organizes to the percolation
transition with activity avalanches propagating marginally across the graph. No fine-tuning of
parameters is needed.

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One astonishing property of many complex natural systems is their extreme robustness and their stability in
the face of a changing environment. Complex information processing in biological systems, as neural networks,
molecular signaling of the cell, or the immune system, seems to avoid regimes of chaotic dynamics, which easily
could occur wherever a large number of interacting regulators or switches are connected. Mechanisms that in
principle can stabilize complex interacting systems have been studied recently in model systems of theoretical
physics. One mechanism that allows a system to self-organize into a specific global state on the basis of loc-
cally acting forces is Self-Organized Criticality (SOC), enabling dissipative systems to drive themselves into crit-
cical states without any parameter tuning. Examples are sandpile models [1] or models of evolving species rel-
ationships in biological evolution [2]. Similar systems that self-organize to specific critical states are forest fire mod-
els [3], and earthquake models [4]. It is an interesting question whether similarly simple principles of self-organization are at work in biological information processing systems. In such systems, often
organized as networks of cells or molecules, the architecture of interactions may contribute to the overall rob-
ustness. Classical models of self-organized criticality, in contrast, are defined on a Euclidean neighborhood and
any information about robustness of the system is contained in the excitations of the system. Thus the dynam-
ical variables fulfill two purposes: On the one hand they act as the dynamical variables of the system, however,
in addition they store the memory of the self-organized state, for example in terms of a particular spatial pattern of excitations. One may question, whether this form of a

memory provides a sufficient basis for robustness as some minimal long term stability is required. Indeed, in some
natural information processing systems one observes that robustness is stored in topology and details of the local
interaction structure [5]. In this paper we will consider a toy model for self-organization of communication net-
works that separates the two time scales of dynamics and adaptation of the system and also the associated vari-
ables. Models of network evolution have been considered earlier from the perspective of self-organization [6]. Self-
organization through coupling of locally adapting links to an order parameter of the global dynamical phase has
shown to be robust [7]. Here we wish to address the question of self-organizing networks for an even simpler
system: percolation on a graph with adaptive links.

Percolation has been a valuable concept for questions of signal propagation through structures [8]. Critical prop-
erties of a dynamical system can often be conveniently accessed by observing percolation of damage or pertur-
bations through the network [9]. One sign of criticality often observed in dynamical systems is marginal damage
spreading across the system [10, 11]. We will study here how marginal damage spreading can be the result of a dy-
namical (adaptation) process itself. For this purpose let us consider percolation on a directed graph with the addi-
tional property of two types of links, such that an active site can activate or de-activate a neighbor site, depending
on the specific link. This percolation process is closely related to the contact process [12] and the susceptible-
infected-susceptible (SIS) model [13]. In the model studied here, however, de-activation of active sites does not
occur as a decay process with some given rate, but instead is an interactive process with an active neighbor site over a de-activating link. Both, activation and de-
activation of a site thus are regulatory processes. This process exhibits a percolation transition where the con-
rol parameter is the fraction of activating links $\vartheta$. To

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introduce an adaptive process in this system, the excitatory or inhibitory characteristics of links are allowed to adapt on a time scale that is slow in comparison to the time scale of activity propagation on the network. We will show that percolation at the critical threshold occurs naturally in this system, when sites tend to equalize their own average activity with that of their neighbors by locally adjusting their incoming links.

The topology chosen in this paper is motivated from immunology; however, the mechanism is more general and can easily be applied to systems with different topologies and other motivations. Here we consider a graph motivated by idiotypic interactions of the immune system [4, 13, 16]. The graph is spanned by a binary sequence space, where each sequence is a node representing a possible motif of an immune cell receptor. Interactions are represented as links: Each motif interacts with its complement and the nearest neighbors of its complement. Topological properties of and percolation on this so-called one-mismatch graph structure has been discussed in [17]. A number of cellular automata models to study possible dynamical and functional roles of such networks in the immune system have been performed (for reviews see [4] and [15]). While from an experimental standpoint idiotypic networks are an open issue today, (neither their large scale structure is known nor a possible functional role confirmed), from a theoretical view it can be inspiring to nevertheless speculate about possible functions. We here consider a possible role in the regulation of activity levels of autoreactive lymphocytes which are present in healthy individuals [13] but usually do not lead to auto-immune diseases as long as adequate regulation takes place [21, 22]. At several stages, the immune system shows a preference for lymphocytes with medium activity [20], a state that appears to be actively regulated. One observes that lymphocytes, on the one hand, need some minimal stimulus for proliferation, but on the other hand may die under chronic stimulation by self-antigens [20]. Such local interactions between motifs in the immune system are candidates for systemwide regulatory mechanisms, however, how a globally stable state of cells with medium average activities is reached in detail is still an open question. In the following we ask how a network of general dynamical states may self-organize to intermediate activity levels of its nodes. Preference for medium activity should not externally be introduced but result from local dynamics. We study a basic mechanism where each node tends to adjust its own average activity to that of its neighbors. An active site that on average is more (less) active than its neighbors in the network experiences more inhibition (activation). As we will see, this results in marginal percolation of activity across the network corresponding to a critical state of the system with stable, intermediate average activity of every node.

Consider a sequence space of binary strings of length \( n \). Each string codes for a specific immune receptor. Interaction is defined between complementary sequences and their one-bit-mutants [17]. Each site in sequence space is assigned a Boolean variable which determines whether the site is active or inactive. Each active sequence can affect its complementary sequence and the associated one-bit-mutants in terms of activation or inhibition. This interaction is represented by directed links between the involved nodes. The system is initialized with a fraction \( \vartheta \) of activating links. Starting from an initial condition of sites being mostly inactive and a small number of activated sites, the following dynamical rules are iterated.

1. **Choose a random site.**

2. **If this site is active** evaluate the influence of its \( n + 1 \) outlinks as follows:
   - If the outlink is activating, the target sequence gets activated.
   - If the outlink is inhibiting, the target sequence gets de-activated.

As can be seen from figure 1, the system shows a percolation transition under variation of \( \vartheta \). The percolation condition is given by

\[
\vartheta > \frac{1}{n+1}.
\]

This corresponds to the condition that each site on average activates one other site. Above the percolation threshold, the system’s equilibrium state is insensitive to the fraction of active sites at initialization. The system’s evolution can be described by the master equations

\[
\frac{dp_0}{dt} = w_{01}p_1 - w_{10}p_0, \\
\frac{dp_1}{dt} = w_{10}p_0 - w_{01}p_1 = w_{10} - (w_{10} + w_{01})p_1,
\]

where \( p_1 \) (\( p_0 = 1 - p_1 \)) denotes the probability to find an activated (inactivated) site and \( w_{01} \) and \( w_{10} \) represent the respective transition probabilities. In the mean field limit we can approximate

\[
\frac{dp_1}{dt} \sim p_1 \vartheta (1 - p_1) - p_1 (1 - \vartheta) p_1 = p_1 \vartheta - p_1^2.
\]

![FIG. 1: Average fraction of active sites in equilibrium vs. fraction of activating links \( \vartheta \), \( n = 15, \vartheta^c = \frac{1}{16} = 0.0625 \)](image)
Assuming to have reached equilibrium, we demand $\frac{dp}{dt} = 0$ and get

$$p^*_i = p^{\text{supercrit.}}(\infty) = \vartheta. \quad (2)$$

Sufficiently far above the percolation threshold the fraction of active sites after equilibration of the system is identical to the fraction of activating links. Below the percolation threshold the fraction of active sites vanishes for infinite system size. For finite systems, the number of activated sites in equilibrium may also depend on the initial set of active sites (cp. figure 1).

Next let us add a slow, local adaptation of links to the model by which nodes adjust their respective average activity to each other. We will see that this may drive the system to and stabilize it at the critical percolation threshold. Define the mean activity $\langle a_i \rangle_T$ of the site $i$ over some time interval $T$ as

$$\langle a_i \rangle_T = \frac{1}{T} \sum_{j=1}^{T} a_i(j), \quad (3)$$

with $a_i(j) \in \{0,1\}$ denoting the activity status of site $i$ at time $j$. The mean activity of the nearest neighbors of a site $i$ is given by

$$\langle a_i \rangle^{nn}_T = \frac{1}{n+1} \sum_{j \in nn} \langle a_j \rangle_T. \quad (4)$$

The self-organization of the network takes place by adjusting the average activity of active sites towards their neighbors’ mean activity, according to the following rules:

1. Choose a random site $i$.
2. Spread the activity as in the topologically static model.
3. If the site is active adjust activating/inhibiting inlinks as follows:
   - if $\langle a_i \rangle_T > \langle a_i \rangle^{nn}_T$ and if the site $i$ has activating inlinks, then reset a random one of them to be inhibiting with probability $p$.
   - if $\langle a_i \rangle_T \leq \langle a_i \rangle^{nn}_T$ and if the site $i$ has inhibiting inlinks, then reset a random one of them to be activating with probability $p$.

These rules are then iterated. For a full run, one first initializes the system with a supercritical fraction of activating links and sets all sites to be activated. One then lets the system evolve as in the static model until it reaches its equilibrium fraction of active sites $p^*_i \approx \vartheta$. At that point, also topological evolution as defined by the above rules is switched on. We observe that the system equilibrates at a fraction of activating links that is near the critical value, independent from the initial fraction of activating links $\vartheta$. No fine tuning is needed to reach this point. In particular, we observe that the time scale of equilibration $T$ can be varied in a wide parameter range without major effects on the limit fraction of activating links. Furthermore, the convergence is robust against changes in the probability of local adjustment $p$ which only affects the time scale of equilibration. To estimate the effect of noise on equilibration, we studied a variant where, at any iteration, we flip the status of a random link with some fixed probability $p_{\text{noise}}$. One observes that low noise levels help avoiding suboptimal freezing (sometimes occurring in small systems) and help approaching the critical fraction of activating links. Further increasing noise levels, the system equilibrates at higher than critical levels of activating links.

An interesting observable of a system at criticality is the size of dynamical avalanches following small perturbations of the system. In order to obtain the distribution of avalanches in a system that has been equilibrated following the above dynamical rules, all sites are reset to be inactive and then a single random site is activated and the subsequent activity avalanche is measured. A statistics of measured avalanche sizes is shown in figure 2.

![Figure 2: Distribution of avalanche sizes in the self-organized network, corresponding to the time averaged number of active sites after equilibration (•) and the number of sites that have ever been activated during the spread of an avalanche (○).](image)

Avalanche sizes are shown as time-averaged number of active sites in an avalanche (•) and as number of sites that have been active at some point during an avalanche (○). We observe power law scaling with exponents $-1.43$ and $-1.33$, respectively. The small peak at the right end of the distribution is a contribution from the giant component of the network, indicating that the network of this simulation is slightly overcritical. The activating links still form a system-spanning subnetwork here, allowing for broad spreading of activity. Finally, let us study whether the critical point is approached in the thermodynamic limit using finite size scaling arguments. Figure 3 shows the deviation of the equilibrium fraction of activating links $\vartheta_{eq}(N)$ from the critical value $\vartheta^c$ with increasing system size $N = 2^n$. One finds a finite size scaling relationship for $\vartheta_{eq}(N)$ given by

$$\vartheta_{eq}(N) - \vartheta^c = \vartheta_{eq}(2^n) - \frac{1}{n+1} = \alpha N^{-\beta}.$$
Thus we see that the fraction of activating links $\vartheta_{eq}(N)$ of the equilibrated evolving network converges with increasing system size $N = 2^n$ (as computationally accessible) towards its critical value.

In this paper we studied percolation on a directed graph with activating and de-activating links, exhibiting a percolation transition at a critical fraction of activating links. In an evolution version of this model, links are allowed to evolve (change their character between activating and de-activating) on a slow timescale in a way that the average activities of the two nodes adjacent to the link become more similar to each other. One finds that this locally and extremely simplistic rule suffices to self-organize the network to the percolation transition with high precision yet without any need for fine tuning. From a damage spreading perspective, the condition for critical percolation is that activity spreads marginally across the system $\vartheta = \frac{1}{n + 1}$ with a scaling law $N^{-\beta}$, $\beta = 0.23$.

The particular graph structure used here is motivated by idiotypic immune networks and the need for balancing the activity of immune cells to an intermediate level for a functioning immune system. While today one can at most speculate about possible mechanisms for global immune regulation, toy models can give important information about dynamical phenomena that may occur in large complex dynamical systems. In particular, emergent phenomena as the one studied in this paper based on simple regulative processes on the microscale can generate unexpected global behavior that could manifest itself for example as powerful global regulation of the system. The model studied here is not restricted to the particular graph we used and it would be interesting to study networks with different topologies as, for example, random graphs or directed networks with a scale-free degree distribution.

Further, it is not difficult to conceive of other models where the basic principle discussed here, local activity adjustment between neighbors, could be at work in different contexts. Possible extensions include neural networks or more complicated cellular automaton models as used, e.g., in models of social systems. Also extensions of classical SOC models are possible, such that key variables for self-organization are stored in separate, more robust variables (as weights or couplings, for example).

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\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig3.png}
\caption{Finite size scaling of the fraction of activating links $\vartheta_{eq}(N)$, $\vartheta_{eq}(N) - \frac{1}{n+1}$ vs. system size $N = 2^n$, $\vartheta_{eq}(N)$ approaches $\vartheta^* = \frac{1}{n+1}$ with a scaling law $N^{-\beta}$, $\beta = 0.23$.}
\end{figure}

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