# On the dangers of Boolean networks:
# Activity dependent criticality and threshold networks not faithful to biology

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Spin models of neural networks and genetic networks are considered elegant as they are accessible to statistical mechanics tools for spin glasses and magnetic systems. However, the conventional choice of variables in spin systems may cause problems in some models when parameter choices are unrealistic from the biological perspective. Obviously, this may limit the role of a model as a template model for biological systems. Perhaps less obviously, also ensembles of random networks are affected and may exhibit different critical properties. We here consider a prototypical network model that is biologically plausible in its local mechanisms. We study a discrete dynamical network with two characteristic properties: Nodes with binary states 0 and 1, and a modified threshold function with $\Theta_0(0) = 0$. We explore the critical properties of random networks of such nodes and find a critical connectivity $K_c = 2.0$ with activity vanishing at the critical point.

We currently experience a revived interest in dynamical networks of nodes with binary states, driven by two active fields of research: modeling of molecular information processing networks (as, e.g., genetic networks or protein networks) [1], as well as modeling of adaptive networks [2]. These network models with binary states are reminiscent of artificial neural networks as studied in the statistical mechanics community about two decades ago.

An early motivation of networks with binary node states $\sigma_i \in \{0, 1\}$ was given by McCulloch and Pitts in 1943 [3] as a model for neural information processing. A model for associative memory constructed from such nodes by Hopfield in 1982 [4] attracted considerable interest among physicists as it is conveniently accessible to equilibrium statistical mechanics methods [5–7]. A simple redefinition of weights and thresholds maps the model onto the mathematical representation of a spin glass with states $\sigma_i \in \{-1, 1\}$, which has become the usual form of the Hopfield model in the physics literature. The corresponding redefinition of weights and thresholds does not affect the functioning of the model, as its mechanism of an associative memory works on the redefined weights as well.

In some circumstances, however, when faithful representation of certain biological details is important, the exact definition matters. In the spin version of a neural network model, for example, a node with negative spin state $\sigma_j = -1$ will transmit non-zero signals through its outgoing weights $c_{ij}$, despite representing an inactive (!) biological node. In the model, such signals arrive at target nodes $i$, e.g., as a sum of incoming signals $h_i = \sum_{j=1}^{N} c_{ij}\sigma_j$. However, biological nodes, as genes or neurons, usually do not transmit signals when inactive. In biochemical network models each node represents whether a specific chemical component is present ($\sigma = 1$) or absent ($\sigma = 0$). Thus the network itself is mostly in a state of being partially absent as, e.g., in a protein network where for every absent protein all of its outgoing links are absent as well [25]. In the spin state convention, this fact is not faithfully represented.

Another example for an inaccurate detail is the common practice to use the standard convention of the Heaviside step function as an activation function in discrete dynamical networks (or the sign function in the spin model context). The convention $\Theta(0) = 1$ is not a careful representation of biological circumstances. Both, for genes and neurons, a silent input frequently maps to a silent output. Therefore, we use a redefined threshold function defined as

$$\Theta_0(x) = \begin{cases} 1, & x > 0 \\ 0, & x \le 0. \end{cases} \qquad (1)$$

When studying statistical properties of ensembles of threshold networks with random links, these details have a considerable influence on the network's dynamics and critical properties. When simulating ensembles via networks of spins $\sigma_i \in \{-1, 1\}$, care should be taken to properly renormalize weights and activation thresholds to ensure faithful implementation of the original model with states $\sigma_i \in \{0, 1\}$. However, this is frequently omitted, resulting in the statistics of a system of limited biological plausibility [8–12].

Another example where normalization and the definition of the nodes' thresholds matters are adaptive networks, currently discussed in the context of neural networks [2, 13–16]. When defining local adaptive mechanisms, it is particularly important to base it on biologically plausible definitions of nodes and circuits. While these mechanisms work also for spin type networks [13], such an implementation is not realizable in a biological context, as it would require signals over links which are in fact silent, due to the inactivity of their source nodes. An adaptive algorithm based on such correlations of non-activity is therefore not plausible.

In this paper, we first define a binary threshold network

that does not include explicitly forbidden states in the context of biological examples. Then we study its critical properties which we find to be distinctly different from those of random Boolean networks [17–21] and random threshold networks [8, 22, 23]. In particular, activity of the network now influences criticality in a non-trivial way, as recently observed for random threshold networks with bistable nodes [24].

Let us consider randomly wired threshold networks of $N$ nodes $\sigma_i \in \{0, 1\}$. At each discrete time step, all nodes are updated in parallel according to

$$\sigma_i(t + 1) = \Theta_0(f_i(t)) \tag{2}$$

using the input function

$$f_i(t) = \sum_{j=1}^{N} c_{ij}\sigma_j(t) + \theta_i. \tag{3}$$

In particular we choose $\Theta_0(0) := 0$ for plausibility reasons (zero input signal will produce zero output). While the weights take discrete values $c_{ij} = \pm 1$ with equal probability for connected nodes, we select the thresholds $\theta_i = 0$ for the following discussion. For any node $i$, the number of incoming links $c_{ij} \neq 0$ is called the in-degree $k_i$ of that specific node. $K$ denotes the average connectivity of the whole network. With randomly placed links, the probability for each node to actually have $k_i = k$ incoming links follows a Poissonian distribution:

$$p(k_i = k) = \frac{K^k}{k!} \cdot \exp(-K). \tag{4}$$

To analytically derive the critical connectivity of this type of network model, we first study damage spreading on a local basis and calculate the probability $p_s(k)$ for a single node to propagate a small perturbation, i.e. to change its output from 0 to 1 or vice versa after changing a single input state. The calculation can be done closely following the derivation for spin-type threshold networks in ref. [23], but one has to account for the possible occurrence of '0' input signals also via non-zero links. The combinatorial approach yields a result that directly corresponds to the spin-type network calculation via $p_s^{\text{bool}}(k) = p_s^{\text{spin}}(2k)$. However, this approach does not hold true for our Boolean model in combination with the defined Theta function $\Theta_0(0) := 0$ as it assumes a statistically equal distribution of all possible input configurations for a single node. In the Boolean model, this would involve an average node activity of $b = 0.5$ over the whole network. Instead we find (Fig. 1) that the average activity on the network is significantly below 0.5. At $K = 4$ (which will turn out to be already far in the supercritical regime), less than 30 percent of all nodes are active on average. Around $K \approx 2$ (where we usually expect the critical connectivity for such networks), the average activity is in fact below 10 percent. Thus,
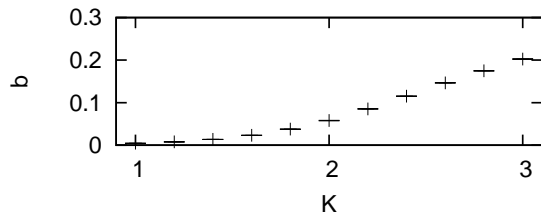


FIG. 1: Average node activity $b$ as function of connectivity $K$ measured on attractors of 10000 sample networks each, 200 nodes.

random input configurations will more likely consist of a higher number of '0' signal contributions than of $\pm 1$ inputs.

Therefore, when counting input configurations for the combinatorial derivation of $p_s(k)$, we need to weight all relevant configurations according to their realization probability as given by the average activity $b$. For the first $k = 1, 2, 3\ldots$ this yields

$$p_s(1) = \frac{1}{2}$$

$$p_s(2) = \frac{1}{2} - b\left(\frac{1}{4}\right)$$

$$p_s(3) = \frac{1}{2} - b\left(\frac{1}{2} - \frac{1}{4}b\right)$$

$$p_s(4) = \frac{1}{2} - b\left(\frac{3}{4} - \frac{3}{4}b + \frac{5}{16}b^2\right)$$

$$p_s(5) = \ldots$$

which generalizes to

$$p_s(k) = \frac{1}{2} + \left(\sum_{i=1}^{k} (-1)^i \binom{k-1}{i} X_i b^i\right) \tag{5}$$

using $X_i = X_{i-1} \cdot \left(\frac{2i-1}{i+1}\right)$ and $X_1 = \frac{1}{4}$.

As the in-degree $k$ is not equal for all nodes, the expectation value of $p_s(K)$ is essential to determine the critical connectivity of the whole network. This will yield the average probability for damage spreading for a certain average connectivity $K$.

Consider a network of size $N - 1$. For large $N$, the problem studied in the above section is equivalent to connecting a new node $j$ with state $\sigma_j$ to an arbitrary node $i$, increasing $k_i$ to $k_i + 1$. If now $\sigma_j$ is changed, this will result in a state change of node $i$ with a probability $p_s(k + 1)$. As mentioned above, the link distribution follows a Poissonian; thus we can calculate the expectation value $\langle p_s \rangle(K)$ for the thermodynamical limit $N \longrightarrow \infty$ as

$$\langle p_s \rangle(K) = \exp(-K) \sum_{k=0}^{\infty} \frac{K^k}{k!} p_s(k + 1) \tag{6}$$
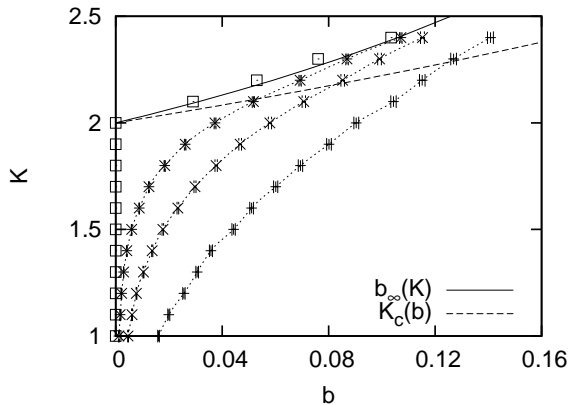
FIG. 2: Average activity $b$ on attractors of different network sizes (right to left: $N = 50, 200, 800$, ensemble averages were taken over 10000 networks each). Squares indicate activity on infinite system determined by finite size scaling, which is in good agreement with the analytic result (solid line). The dashed line shows the analytic result for $K_c(b)$ from eq. (7). The intersections represent the value of $K_c$ for the given network size.

| $N$ | 50 | 100 | 200 | 400 | 800 | $\to \infty$ |
|---|---|---|---|---|---|---|
| $K_c(\pm 0.005)$ | 2.285 | 2.225 | 2.180 | 2.145 | 2.115 | 2.000 |

TABLE I: Critical Connectivity $K_c$ for different sizes as determined from curve intersections in Figure 2.

which can be computed numerically using $p_s(k)$ as given in eq. (5).

The actual calculation of the critical connectivity $K_c$ for the network model presented above is now done by applying the annealed approximation as introduced by Derrida and Pomeau [18]. The application of this method on threshold networks is discussed in detail in [23], which can be directly transferred on the network model discussed in the present work. The critical connectivity can thus be obtained by solving

$$\langle p_s \rangle (K_c) \cdot K_c = 1. \qquad (7)$$

However, $K_c$ now depends on the average network activity, which in turn is a function of the average connectivity $K$ itself as shown in Fig. 1. From the combined plot in Fig. 2 we find that both curves intersect at a point where the network dynamics – due to the current connectivity $K$ – exhibit an average activity which in turn yields a critical connectivity $K_c$ that exactly matches the given connectivity. This intersection thus corresponds to the critical connectivity of the present network model.

However, the average activity still varies with different network sizes, which is obvious from Figure 2. Therefore, also the critical connectivity is a function of $N$. Table I lists results for different values of $N$. For an analytic approach to the infinite size limit, we can now calculate the

probability for a node at given in-degree $k$ and average network activity $b_t$ at time $t$ to exhibit output state 1. This probability equals the average activity for the next time step $b_{t+1}$. By examining all relevant input configurations, we find that for given constant $k$ this generalizes to

$$b_{t+1}(k) = \sum_{i=1}^{k} (-1)^{i+1} \frac{1}{2^i} \binom{2i-1}{i-1} \binom{k}{i} b_t^i. \qquad (8)$$

Again, we have to account for the Poissonian distribution of links in our network model, so the average evolution of network activity is obtained by

$$\langle b_{t+1} \rangle (K) = \exp(-K) \sum_{k=1}^{\infty} \frac{K^k}{k!} b_{t+1}(k). \qquad (9)$$

It is now possible to distinguish between the different dynamical regimes by solving $\langle b_{t+1} \rangle = b_t(K)$ for the critical line. The solid line in Figure 2 depicts the evolved activity in the long time limit. We find that for infinite system size, the critical connectivity is at

$$K_c(N \to \infty) = 2.000 \pm 0.001$$

while up to this value all network activity vanishes in the long time limit ($b_\infty = 0$). For any average connectivity $K > 2$, a certain fraction of nodes remains active. In finite size systems, both network activity evolution and damage propagation probabilities are subject to finite size effects, thus increasing $K_c$ to a higher value.

As a numerical verification, we can also derive the critical connectivity for infinite system size $K_c(N \to \infty)$ using finite size scaling of the above simulation results (Table I). The optimum fit is shown in Figure 3 and yields

$$K_c(N \to \infty) = 2.00 \pm 0.01$$

which perfectly supports the analytical calculation. The same consideration is also possible to obtain the average activity $b(N)$ for increasing network size. This can be done both at constant values of $K$ as well as along the critical line in Figure 2 using the values of $K_c(N)$ from Table I. For the critical line, we indeed find vanishing network activity (inset of Figure 3):

$$b_c(N \to \infty) = 0.00 \pm 0.01.$$

This does also hold true for any connectivity below the critical line. For supercritical networks at $K > K_c$, the numerical simulation yields a non-zero fraction of nodes which remains active on average in good agreement with the analytic computation (see squares in Figure 2).

In additional numerical simulations using the standard step function, we obtain a critical connectivity of $K_c \approx 3.7$, which is analytically supported by a combinatorial calculation following ref. [23] where we find
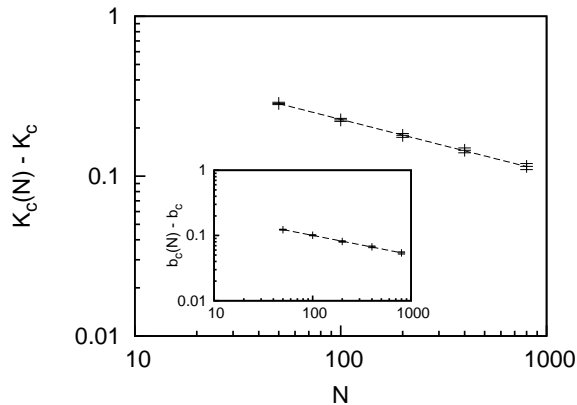
FIG. 3: Finite size scaling of $K_c(N)$, optimum fit shown here for $K_c(N \to \infty) = 2.00$. Inset: Finite size scaling of $b(N)$ along the critical line, the optimum fit is obtained for vanishing activity $b_c(N \to \infty) = 0$.
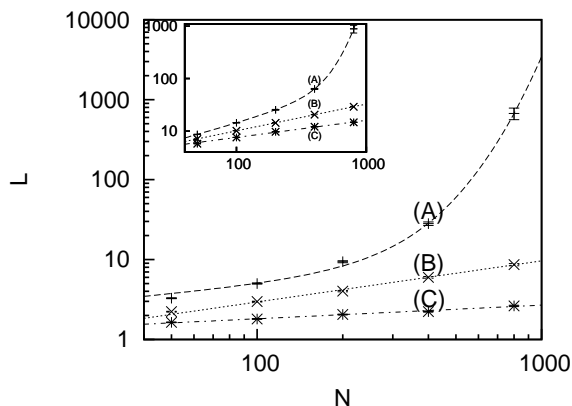


FIG. 4: Average attractor length at different network sizes. Ensemble averages were taken over 10000 networks each at (A) $K = 2.4$, (B) $K = 2.0$, (C) $K = 1.6$. Inset figure shows the scaling behavior of the corresponding transient lengths.

$p_s^{\text{bool}}(k) = p_s^{\text{spin}}(2k)$. The networks exhibit significantly higher average activity, while most of the active nodes are frozen in the active state. On a side note, if we chose to calculate the critical connectivity based on an assumed average activity of 0.5, the activity-dependent calculation via (5) and (7) would effectively reproduce the same result ($K_c \approx 3.7$) as the original combinatiorial approach from [23] would yield for the new Boolean model. As the assumption does not hold true here, this result can only be viewed as an additional plausibility check for the correspondence between both approaches. In passing we note that also for the spin type threshold network studied in [23] we find activity levels different from 0.5, such that conditions for a valid combinatorial approach might not be met in that case, as well.

Finally, let us have a closer look on the average length of attractor cycles and transients. As shown in Fig. 4, the

behavior is strongly dependent of the dynamical regime of the network. As expected and in accordance with early works on random threshold networks [8] as well as random Boolean networks [20], we find an exponential increase of the average attractor lengths with network size $N$ in the chaotic regime ($K > K_c$), whereas we can observe a sub-linear increase in the frozen phase ($K < K_c$). We find similar behavior for the scaling of transient lengths (inset of Figure 4).

In summary we studied threshold networks with Boolean node states that are biologically more plausible than current Boolean and threshold networks and which are simpler than the recently introduced networks with bistable threshold nodes [26, 27]. A major observation is that activity of the nodes depends on connectivity which also renders critical properties of the networks activity-dependent, as found earlier for random threshold networks with bistable nodes [24]. We extend the annealed approximation to correct for these effects and find connectivity $K_c = 2.0$ and vanishing activity at the critical point in the thermodynamic limit.

Going beyond the statistics of random network ensembles, also real biological circuits can be implemented with great ease using the threshold networks defined here. We successfully reproduced the dynamical trajectory of the budding yeast cell cycle network as implemented with bistable threshold functions in [26], as well as for the corresponding network in fission yeast [27]. [28]

To conclude, let us remind ourselves of the original idea of using random Boolean networks for characterizing typical properties of biological networks [17]. In 1969, using random Boolean networks as a null model for genetic networks was a logical approach, given our complete ignorance of the circuitry of genetic networks at that time. Thus the best guess was to treat all possible Boolean rules as equally probable. Today, however, we have much more details knowledge about certain properties of genetic networks and, therefore, about more realistic ensembles of random networks. A biologically motivated and carefully defined threshold network, as attempted in this paper, may provide a more suited null model for the particular properties of biological networks than random Boolean networks with equally distributed Boolean functions.

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[28] For this we replace self-degrading nodes with regular nodes of our model, represent the bistable nodes by a regular node plus a positive self-interaction, and set the thresholds accordingly.