

Max-Planck-Institut  
für Mathematik  
in den Naturwissenschaften  
Leipzig

Self-organization of heterogeneous topology and  
symmetry breaking in networks with adaptive  
thresholds and rewiring

by

*Thimo Rohlf*

Preprint no.: 54

2008





---

# Self-organization of heterogeneous topology and symmetry breaking in networks with adaptive thresholds and rewiring

THIMO ROHLF

*Max-Planck-Institute for Mathematics in the Sciences, Inselstr. 22, D-04103 Leipzig, Germany*

PACS 05.45.-a –

PACS 05.65.+b –

PACS 89.75.-k –

**Abstract.** – We study an evolutionary algorithm that locally adapts thresholds and wiring in Random Threshold Networks, based on measurements of a dynamical order parameter. If a node is active, with probability  $p$  an existing link is deleted, with probability  $1 - p$  the node's threshold is increased, if it is frozen, with probability  $p$  it acquires a new link, with probability  $1 - p$  the node's threshold is decreased. For any  $p < 1$ , we find spontaneous symmetry breaking into a new class of self-organized networks, characterized by a much higher average connectivity  $\bar{K}_{evo}$  than networks without threshold adaptation ( $p = 1$ ). While  $\bar{K}_{evo}$  and evolved out-degree distributions are independent from  $p$  for  $p < 1$ , in-degree distributions become broader when  $p \rightarrow 1$ , indicating crossover to a power-law. In this limit, time scale separation between threshold adaptations and rewiring also leads to strong correlations between thresholds and in-degree. Finally, evidence is presented that networks converge to self-organized criticality for large  $N$ , and possible applications to problems in the context of the evolution of gene regulatory networks and development of neuronal networks are discussed.

---

**Introduction.** – Interaction networks in nature often exhibit highly inhomogeneous architectures. Examples are scale-free degree distributions in protein networks [1] and metabolic networks [2], mostly accompanied by intricate second order regularities as, for example, community structure [3]. The emergence of these properties often is explained by means of intuitive topology-based models, e.g. preferential attachment [4] or node duplications [5]. Real networks, however, are characterized not only by an evolving topology, but also by evolution of *function*, conveniently abstracted in terms of dynamics, i.e. the flow of information or matter on these networks. So far, only few studies explicitly consider the more general case of co-evolution between network dynamics and -topology [6–9].

One example is the question how networks may evolve topologies that optimize biologically relevant parameters, e.g. flexible adaptation with respect to changing environments, or insensitivity against random perturbations of topology or dynamics (robustness) [10]. In this context, Kauffman introduced random Boolean networks (RBN) to study the dynamics of gene regulatory networks from a global perspective [11, 12]. It was shown that RBN undergo a order-disorder transition at a critical wiring den-

sity (connectivity)  $K_c = 2$  [11–14]; similar results were established for random threshold networks (RTN), which constitute a sub-class of RBN [15–18]. It has been postulated that evolution should drive dynamical networks towards this 'edge of chaos' to optimize adaptive flexibility and robustness [11, 12, 19]. Interestingly, in recent years experimental evidence has accumulated that information processing networks in biological organisms indeed operate close to criticality. In particular, gene regulatory networks of several organisms have been shown to exhibit critical dynamics [20–22], similar results were established for neuronal networks in the brain [23, 24]. Since, in all these systems, there generally exists no central control instance that could continuously adjust global system parameters to poise dynamics at the critical state, we have to postulate that there are simple, *local* adaptive mechanisms present that are capable of driving *global* dynamics to a state of *self-organized criticality*. However, for many years no such mechanism able to generate critically connected networks could be provided.

To address this problem, a RTN-based model was proposed, linking rewiring of network nodes to local measurements of a dynamical order parameter, e.g. the aver-

age activity (magnetization) [7]. It was shown that this simple, *local* adaptive mechanism leads to a *global* self-organized critical state in the limit of large system sizes  $N$ . Subsequently, this principle was generalized to networks of noisy neurons [8] and to RBN with evolvable logical functions [9]. Interestingly, finite size networks in these models evolve a broadly distributed heterogeneous in-degree connectivity [9, 25]. Still, these topological heterogeneities are smaller than those observed in real-world networks, presumably because dynamical elements were assumed to be homogeneous with respect to their dynamical behavior. While this assumption leads to elegant models, it is quite unrealistic, as it becomes apparent e.g. in the frequent occurrence of canalizing functions in gene regulatory networks, with strong impact on dynamics in RBN models [26]. Recent studies have shown that partial canalization completely dominates the space of all possible Boolean functions, in particular, for higher  $k$  [27]; this can implicitly result in a rewiring in models that evolve the functions of the nodes [28, 29]. Considering the accumulating experimental evidence of both close-to criticality and heterogeneous architecture of information processing networks in nature, for instance in gene regulatory networks [20–22, 30, 31] and neuronal networks in the nervous system [23, 24, 32], it is fascinating to speculate about a mechanism that might explain both observations: coevolution of *local* structural-dynamical heterogeneity and *global* homeostasis by local dynamical rules. For this purpose,

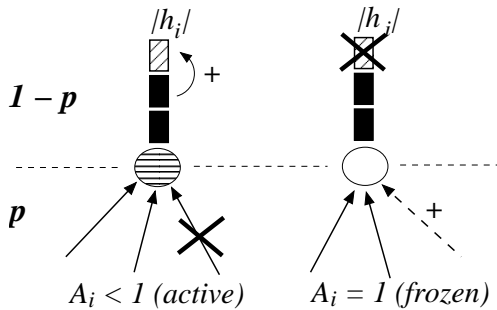


Fig. 1: *Left*: with probability  $p$ , active nodes lose one of their inputs, with probability  $1 - p$  they increase their (absolute) threshold  $|h_i|$ . *Right*: with probability  $p$ , frozen nodes acquire a new input from a randomly chosen site  $j$ , changing  $c_{ij} = 0$  to  $c_{ij} = \pm 1$ ,  $+1$  or  $-1$  with equal probability. With probability  $1 - p$ , the (absolute) threshold  $|h_i|$  is decremented instead.

we introduce a minimal model linking regulation of activation thresholds and rewiring of network nodes in RTN to local measurements of a dynamical order parameter. A new control parameter  $p \in [0, 1]$  determines the probability of rewiring vs. threshold adaptations: If a node is active, with probability  $p$  an existing link is deleted, with probability  $1 - p$  the node's threshold is increased, if it is frozen, with probability  $p$  it acquires a new link, with probability  $1 - p$  the node's threshold is decreased. We show that the symmetry of the evolutionary attractor for

$p = 1$  (no threshold adaptations, rewiring only) is broken spontaneously for any  $p < 1$ . This new dynamical fixed point, that potentially constitutes a new universality class of self-organized networks, exhibits a much higher average connectivity  $\bar{K}_{evo}$ , compared to  $p = 1$  networks, however, with a value  $\bar{K}_{evo}$  that is *insensitive* to  $p$ . In-degree distributions become very broad, suggesting a crossover to a distribution with a power-law tail  $\sim k_{in}^{-3/4}$  for  $p \rightarrow 1$ . Further, we establish the emergence of strong correlations between in-degree and thresholds in this limit, while for small  $p$ , correlations are weak. This indicates that an adaptive time-scale separation, with rare events of dynamical diversification and frequent rewiring, can lead to emergence of highly inhomogeneous topologies, without the need for network growth (as, for example, in preferential attachment models). Evidence is presented that networks with  $p < 1$  converge to a critical state for large  $N$ , however, with a finite size scaling significantly different from the one found for the case  $p = 1$ , and problems associated to the identification of criticality in this new class of densely wired coevolutionary adaptive networks are discussed. Finally, we discuss how the adaptive principles working in this model might apply to gene regulatory networks in living organisms and neuronal networks in the nervous system.

**Dynamics.** – We consider a network of  $N$  randomly interconnected binary elements with states  $\sigma_i = \pm 1$ . For each site  $i$ , its state at time  $t + 1$  is a function of the inputs it receives from other elements at time  $t$  (synchronous updates):

$$\sigma_i(t + 1) = \begin{cases} +1 & \text{if } f_i(t) > 0 \\ -1 & \text{else} \end{cases} \quad (1)$$

with

$$f_i(t) = \sum_{j=1}^N c_{ij} \sigma_j(t) + h_i. \quad (2)$$

The interaction weights  $c_{ij}$  take discrete values  $c_{ij} = \pm 1$ , with  $c_{ij} = 0$  if site  $i$  does not receive any input from element  $j$ . Thresholds  $h_i$  may vary from node to node, taking integer values  $h_i \leq 0$ <sup>1</sup>. In the following discussion, adaptive changes will be applied to the absolute value  $|h_i|$ , keeping in mind that the sign of  $h_i$  is always negative.

As a *dynamical order parameter*, we define the average activity  $A(i)$  of a site  $i$

$$A(i) = \frac{1}{\tau + 1} \sum_{t=T_1}^{T_1 + \tau} \sigma_i(t). \quad (3)$$

Notice that a *frozen* site, i.e. a site that does not change its state, has  $|A(i)| = 1$ , whereas an *active* site has  $|A(i)| < 1$ .

<sup>1</sup>We chose  $h_i \leq 0$  to ensure that thresholds make *activation*, i.e.  $\sigma_i = +1$ , more difficult.

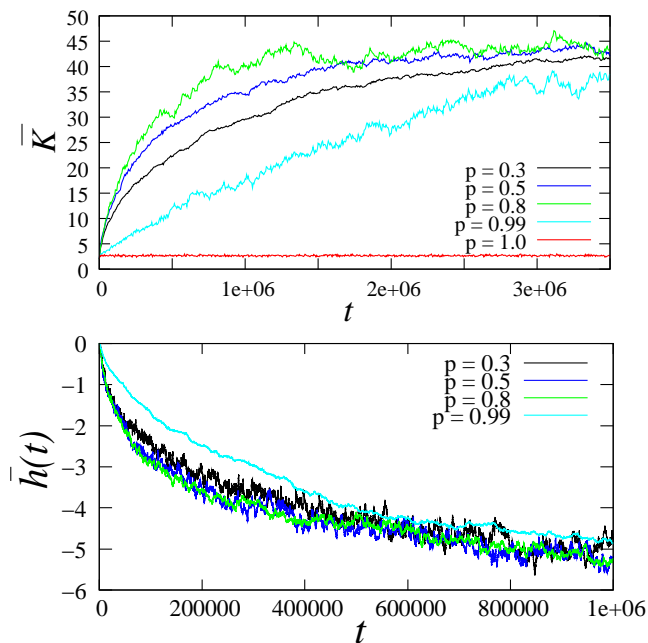


Fig. 2: *Upper panel:* Evolution of the average connectivity  $\bar{K}$  of threshold networks, using the adaptive algorithm (cf. Fig. 1), for  $N = 512$  and initial connectivity  $\bar{K}_{ini} = 1$ . Time series for five different values of  $p$  are shown. *Lower panel:* The same for the average threshold  $\bar{h}$ .

**Topology evolution.** – Let us now discuss a particular evolutionary scheme that couples local adaptations of both the number of inputs and of thresholds to a site’s average activity. Since the switching dynamics of nodes is governed by the deterministic rule Eq. (1), it can be modified only by adaptations of  $c_{ij}$  or  $h_i$  in Eq. (2), by either changing the values of the existing non-zero weights  $c_{ij}$ , by setting a weight that zero previously to  $c_{ij} = \pm 1$ , or by incrementing/decrementing  $h_i$ . If node  $i$  is frozen, it can increase the probability to change its state by either setting a zero weight to  $c_{ij} = \pm 1$ , thereby increasing its number of inputs  $k_i \rightarrow k_i + 1$ , or by making its threshold  $h_i \leq 0$  less negative, i.e.  $|h_i| \rightarrow |h_i| - 1$ . If  $i$  is active, it can reduce its activity by adapting either setting one of its existing non-zero inputs to  $c_{ij} = 0$ , i.e.  $k_i \rightarrow k_i - 1$ , or by increasing its threshold  $|h_i| \rightarrow |h_i| + 1$ . This adaptive scheme is realized in the following algorithm (see also Fig. 1):

1. Create a random network with average connectivity  $\bar{K}_{ini} > 0$  and average threshold  $\bar{h}_{ini} = 0$ . Each of the  $N \cdot \bar{K}_{ini}$  interaction weights is randomly initialized to  $c_{ij} = +1$  or  $c_{ij} = -1$  with equal probability.
2. Select a random initial state  $\vec{\sigma}_{ini} = (\sigma_1, \dots, \sigma_N)$ .
3. Iterate network dynamics for  $T$  timesteps.
4. Select a network site  $i$  at random and measure its average activity  $A_i$  over the last  $\tau = T/2$  updates.

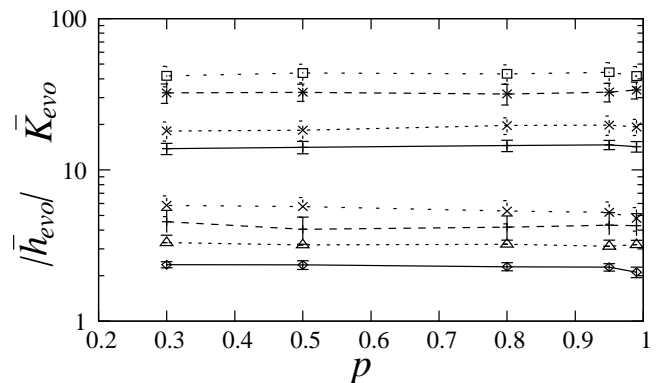


Fig. 3: *Upper four curves:* Evolutionary mean values  $\bar{K}_{evo}$  of the average connectivity, as a function of  $p$ ; system sizes from top to bottom:  $N = 512$ ,  $N = 256$ ,  $N = 128$  and  $N = 64$ . *Lower four curves:* The same for the evolutionary mean values  $|\bar{h}_{evo}|$  of the average absolute threshold. Statistics was taken over  $10^6$  evolutionary steps, after a transient of  $4 \cdot 10^6$  steps.

5. Adapt input number  $k_i$  and threshold  $h_i$  in the following way:
  - If  $|A_i| < 1$ , then with probability  $p$  remove one input  $c_{ij}$  randomly selected from the  $k_i$  inputs, i.e. set  $c_{ij} = 0$ . With probability  $1 - p$ , adapt  $|h_i| \rightarrow |h_i| + 1$  instead.
  - If  $|A_i| = 1$ , then with probability  $p$  add a new input  $c_{ij}$  from a randomly selected site  $j$ , assigning  $c_{ij} = +1$  or  $c_{ij} = -1$  with equal probability. With probability  $1 - p$ , adapt  $|h_i| \rightarrow |h_i| - 1$  instead. If  $h_i = 0$ , let its value unchanged.
6. Go back to step 3.

If the control parameter  $p$  takes values  $p > 1/2$ , rewiring of nodes is favored, whereas for  $p < 1/2$  threshold adaptations are more likely. Notice that the model introduced in [7] is contained as the limiting case  $p = 1$  (rewiring only and  $h_i = \text{const.} = 0$  for all sites). Notice that adaptation of interaction weights  $c_{ij}$  in step 5 conserves the initial symmetry of the weight distribution, i.e.  $c_{ij}$  takes the values  $+1$  and  $-1$  with equal probability  $p = 1/2$ . The number of dynamical updates  $T$  in step 3 of the algorithm was set to  $T = 200$  in simulations, i.e. the average activity was measured over the last  $T/2 = 100$  dynamical updates, after a transient of 100 updates. The transient before activity measurement ensures a proper decoupling from initial conditions, while averaging of  $A(i)$  over 100 updates is enough to provide the typical switching behavior of  $i$  and a decent time scale separation between fast switching dynamics and slow topology adaptation<sup>2</sup>. However, simulations show that the results are not very sensitive to the choice of  $T$ , only for very small values  $T < 10$  we see differences, e.g. convergence to a slightly lower average connectivity.

<sup>2</sup>Since in each evolutionary step one node is rewired, this time scale separation is at the order of  $N \cdot T$ .

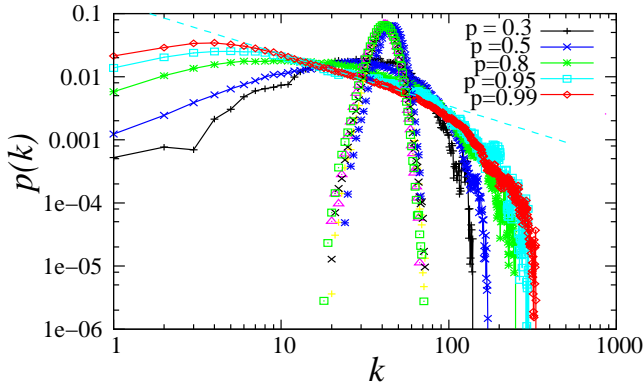


Fig. 4: *Line-pointed curves*: in-degree distributions of evolved networks, *data points only*: the corresponding out-degree distributions ( $\triangle$ )  $p = 0.3$ , (+)  $p = 0.5$ , (x)  $p = 0.8$ , (\*)  $p = 0.95$ , ( $\square$ )  $p = 0.99$ ). Statistics was gathered over  $10^6$  evolutionary steps, after a transient of  $4 \cdot 10^6$  steps. Networks had size  $N = 512$ . The dashed line has slope  $-3/4$ .

**Results.** – After a large number of adaptive cycles, networks self-organize into a *global* evolutionary steady state. An example is shown in Figure 2 for networks with  $N = 512$ : starting from an initial value  $\bar{K}_{ini} = 1$ , the networks’ average connectivity  $\bar{K}$  first increases, and then saturates around a stationary mean value  $\bar{K}_{evo}$ ; similar observations are made for the average threshold  $\bar{h}$  (Fig. 2, lower panel). The non-equilibrium nature of the system manifests itself in limited fluctuations of both  $\bar{K}$  and  $\bar{h}$  around  $\bar{K}_{evo}$  and  $\bar{h}_{evo}$ . Regarding the dependence of  $\bar{K}$  with respect to  $p$ , we make the interesting observation that it changes non-monotonically. Two cases can be distinguished: when  $p = 1$ ,  $\bar{K}$  stabilizes at a very sparse mean value  $\bar{K}_{evo}$ , e.g. for  $N = 512$  at  $\bar{K}_{evo} = 2.664 \pm 0.005$ . When  $p < 1$ , the symmetry of this evolutionary steady state is broken. Now,  $\bar{K}$  converges to a much higher mean value  $\bar{K}_{evo} \approx 43.5 \pm 0.3$  (for  $N = 512$ ), however, the particular value which is finally reached is *independent of  $p$* . The latter observation is made rigorous from measurements of  $\bar{K}_{evo}$  for different  $N$  over  $10^6$  evolutionary steps, after systems have reached the steady state. While  $\bar{K}_{evo}$  obviously depends on the system size  $N$ , curves are very flat with respect to  $p$  (Fig. 3, upper four curves); the same holds for  $|\bar{h}_{evo}|$  (Fig. 3, lower four curves). On the other hand, *convergence times*  $T_{con}$  needed to reach the steady state are strongly influenced by  $p$ :  $T_{con}(p)$  diverges when  $p$  approaches 1 (compare Fig. 2 for  $p = 0.99$ ). We conclude that  $p$  determines the *adaptive time scale*. This is also reflected by the stationary in-degree distributions  $p(k_{in})$  that vary considerably with  $p$  (Fig. 4); when  $p \rightarrow 1$ , these distributions become very broad. The numerical data suggest that a power law

$$\lim_{p \rightarrow 1} p(k_{in}) \propto k_{in}^{-\gamma} \quad (4)$$

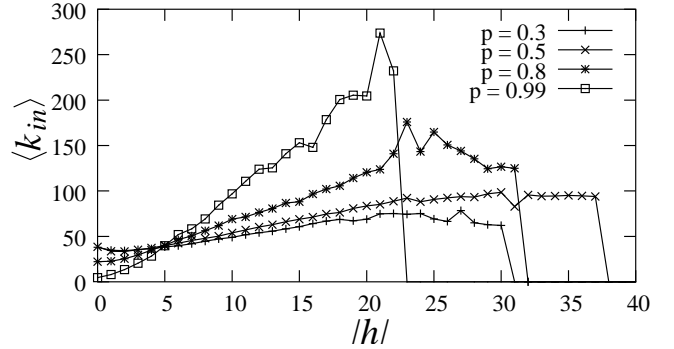


Fig. 5: Average number  $\langle k_{in} \rangle$  of inputs for a given node in evolving networks, as a function of the respective nodes (absolute) threshold  $|h|$ . Statistics was taken over  $10^6$  rewiring steps, after a transient of  $4 \cdot 10^6$  steps. For all values  $p < 1$ , a clear positive correlation between  $\bar{k}_{in}$  and  $|h|$  is found.

with  $\gamma \approx 3/4 \pm 0.03$  is approached in this limit (cf. Fig. 4, dashed line). At the same time, it is interesting to notice that the evolved out-degree distributions are much narrower and completely insensitive to  $p$  (Fig. 4, data points without lines).

How can one understand the emergence of broad in-degree distributions for with increasing  $p$ ? Evidently, life times of both low thresholds  $|h_i| \approx 0$  and high thresholds  $|h_i| \gg 0$  become long for  $p \rightarrow 1$ . Since sites with low thresholds tend to be active and hence, on average, loose links, while sites with high thresholds tend to freeze and hence, on average, acquire new links, we would indeed expect that  $p(k_{in})$  is broadened for  $p \rightarrow 1$ . On the other hand, for  $p \rightarrow 0$ , frequent adaptive changes of thresholds prevent long sequences of both frozen or highly active states, and hence make emergence of strong local wiring heterogeneities less probable. If this idea is correct, we would expect that, in the limit  $p \rightarrow 1$ , the in-degree of sites should exhibit a strong positive correlation to their thresholds, while for  $p \rightarrow 0$  these correlations should be less pronounced. This is indeed exactly what we observe. For  $p = 0.99$ , the average in-degree  $\langle k_{in} \rangle$  of a given node, as a function of its threshold  $|h|$ , shows a step increase, while the corresponding curve is relatively flat for  $p = 0.3$  (Fig. 5).

An interesting question is whether the networks with  $p < 1$  still approach a self-organized critical state for large  $N$ , as it was found for the case  $p = 1$  [7]. Since networks now evolve more densely wired, non-trivial topologies, this question has to be answered by application of a *dynamical* criterion. For this purpose, we studied *damage spreading*: after each adaptive step, dynamics was run from an initial system state  $\vec{\sigma}$  and again from a direct neighbor state  $\vec{\sigma}'$  differing in one bit; after  $t = 200$  updates, the Hamming distance  $d$  between both trajectories was measured and the average fraction of damaged nodes  $\bar{y}(t) =: d/N$  was determined. Figure 6 shows  $\bar{y}$ , averaged over  $10^6$  evolutionary steps, as a function of  $N$ . We find that the finite

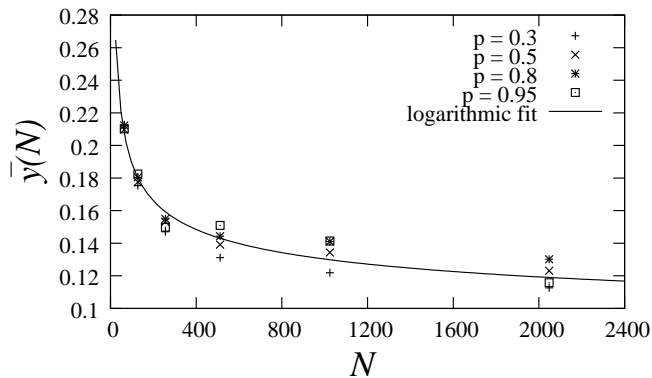


Fig. 6: Average fraction  $\bar{y}(N)$  of damaged nodes, 200 updates after a one-bit perturbation at time  $t = 0$ , for different  $p$ , as a function of system size  $N$ . The lined curve is a fit of the average scaling behavior.

networks investigated here are all supercritical, however,  $\bar{y}$  decreases monotonically with  $N$ . The average scaling behavior can be fit by

$$y(N) \approx a \cdot [\ln(N)]^{-\beta} \quad (5)$$

with  $a = 0.77 \pm 0.02$  and  $\beta = 0.917 \pm 0.01$ . This dependence indicates that  $\bar{y} = 0$ , i.e. the critical transition from chaotic to frozen dynamics, is approached for large  $N$ . Notice, however, that convergence is logarithmic, whereas for  $p = 1$  power laws were found [7, 9]. Again, this indicates that  $p < 1$  networks evolve to a new dynamical fixed point different from the case  $p = 1$ , and hence may constitute a new universality class.

**Discussion.** – To summarize, we studied a model of network evolution that couples both rewiring of inputs and adaptation of activation thresholds to local measurements of a dynamical order parameter. A control parameter  $p$  determines the probability of threshold adaptations vs. link rewiring. While for  $p = 1$  (rewiring only, no threshold adaptation) networks evolve a self-organized critical state with a sparse average connectivity  $\bar{K}_{evo} \approx 2$ , for any  $p < 1$  (both rewiring and threshold adaptation) networks evolve a significantly more dense wiring, with broad heterogeneous in-degree distributions exhibiting a crossover to a power-law  $\sim k_{in}^{-3/4}$  for  $p \rightarrow 1$ . In this limit, time scale separation between rare threshold adaptations and frequent rewiring leads to emergence of strong correlations between thresholds and in-degree. Hence, a new dynamical fixed point of adaptive network evolution has been found that is fundamentally different from networks without threshold adaptation ( $p = 1$ ) and may correspond to a new universality class. We presented evidence that, in the limit of large  $N$ , networks logarithmically approach a self-organized critical state. A detailed characterization of the critical state for this new class of more densely wired, self-organized networks in the limit  $N \rightarrow \infty$  is difficult and remains to be done in future work.

Our model presents a novel mechanism leading to co-evolution of topological *and* dynamical heterogeneity with robust homeostatic regulation, the latter reflected e.g. by the insensitivity of the evolved average connectivity with respect to  $p$ . This combination of properties is rather remarkable and may play a decisive role in information processing networks in nature for maintaining both a close-to critical state and diversity of structure and dynamics; hence, it is interesting to speculate that similar mechanisms might be at work in the evolution of biological networks. Let us briefly discuss this for gene regulatory networks and neuronal networks.

Recent experimental results provide evidence that gene regulatory networks of Eukaryotic cells operate close to criticality [20–22], exhibiting a stunning degree of structural diversity [30] and interactions that can change in response to diverse stimuli [31]. The paradigm of activity-dependent rewiring, that can be paraphrased in this context as *frozen genes acquire new inputs (functions), active (chaotic) genes lose inputs*, provides a simple coevolutionary adaptive scheme [33] for the evolution of a self-organized critical state in regulatory networks. While this model correctly captures topological properties of regulatory networks in simple organisms, e.g. Bacteria [25, 34], it does not reproduce the structural diversity observed in regulatory networks of Eukaryotes. In this paper, we showed that coevolution of dynamical diversity, abstracted in terms of threshold adaptations, with dynamical rewiring can lead to emergence of such non-trivial topologies, reflected e.g. in the observed crossover to power-law distributed connectivities for  $p \rightarrow 1$ . It is interesting to note that diversity of dynamical mechanisms in gene regulation has primarily evolved in Eukaryotes, for example in the context of RNA-based regulation [35] that complements protein-based circuits, and epigenetic reprogramming through DNA methylation [36].

Indications for critical behavior were found also in neuronal networks in the brain [23, 24]. Activity-dependent neural development [32, 37, 38] is a candidate mechanism for emergence of criticality from local dynamical rules capable to regulate crucial global parameters, e.g. the average wiring density, without global knowledge about the system state. Coevolution of global homeostasis with structure-dynamics diversity is found, for example, in the distribution of dendritic spines, with an almost constant average density despite a broad life time distribution of individual spines [32]. Interestingly, the activity-dependent adaptation of wiring and thresholds in our model leads to a similar emergence of both local heterogeneity and global homeostasis. While the current model certainly strongly simplifies the complexity of real dynamical networks, these two examples demonstrate that its evolutionary principles might well apply to different classes of adaptive systems in nature.

## REFERENCES

- [1] MASLOV S. and SNEPPEN K., *Science* , **296** (2002) 910.
- [2] JEONG H., TOMBOR B., ALBERT R., OLTVAI Z. N. and BARABÁSI A. L., *Nature* , **407** (2000) 651.
- [3] GIRVAN M. and NEWMAN M. E. J., *Proc. Natl. Acad. Sci.* , **99** (2002) 7821.
- [4] BARABÁSI A. and ALBERT R., *Science* , **286** (1999) 509.
- [5] BEBEK G., BERENBRINK P., COOPER C., FRIEDETZKY T. and NADEAU J. E. A., *Theor. Comp. Sci.* , **369** (2006) 239.
- [6] BORNHOLDT S. and SNEPPEN K., *Phys. Rev. Lett.* , **81** (1998) 236.
- [7] BORNHOLDT S. and ROHLF T., *Phys. Rev. Lett.* , **84** (2000) 6114.
- [8] BORNHOLDT S. and RÖHL T., *Phys. Rev. E* , **67** (2003) 066118.
- [9] LIU M. and BASSLER K. E., *Phys. Rev. E* , **74** (2006) 041910.
- [10] SAVAGEAU M. A., *Nature* , **229** (1971) 542.
- [11] KAUFFMAN S., *J. Theor. Biol.* , **22** (1969) 437.
- [12] KAUFFMAN S., *The Origins of Order: Self-Organization and Selection in Evolution* (Oxford University Press) 1993.
- [13] DERRIDA B. and POMEAU Y., *Europhys. Lett.* , **1** (1986) 45.
- [14] LUQUE B. and SOLE R. V., *Phys. Rev. E* , **55** (1996) 257.
- [15] KÜRTEK K., *Phys. Lett. A* , **129** (1988) 156.
- [16] KÜRTEK K., *J. Phys. A* , **21** (1988b) L615.
- [17] ROHLF T. and BORNHOLDT S., *Physica A* , **310** (2002) 245.
- [18] ROHLF T., *Critical line in random threshold networks with inhomogeneous thresholds* Submitted, preprint: [arxiv.org/abs/0707.3621](http://arxiv.org/abs/0707.3621) (2007).
- [19] LANGTON C., *Physica D* , **42** (1990) 12.
- [20] SHMULEVICH I., KAUFFMAN S. A. and ALDANA M., *Proc. Natl. Acad. Sci. USA* , **102** (2005) 13439.
- [21] P. R., KESSELI J. and YLI-HARJA O., *J. Theor. Biol.* , **242** (2006) 164.
- [22] NYKTER M., PRICE N. D., ALDANA M., RAMSEY S. A., KAUFFMAN S. A., HOOD L. E., YLI-HARJA O. and SHMULEVICH I., *Proc. Natl. Acad. Sci. USA* , **105** (2008) 1897.
- [23] LINKENKAER-HANSEN K., NIKOULINE V. V., PALVA J. M., and ILMONIEMI R. J., *J. Neurosci.* , **21** (2001) 1370.
- [24] BEGGS J. M., *Phil. Trans. Roy. Soc. A* , **366** (2008) 329.
- [25] ROHLF T. and BORNHOLDT S., *Gene regulatory networks: A discrete model of dynamics and topological evolution* presented at *Function and regulation of cellular systems: experiments and models*, edited by DEUTSCH A., HOWARD J., FALCKE M. and ZIMMERMANN W., (Birkhäuser Basel) 2004.
- [26] MOREIRA A. A. and AMARAL L. A. N., *Phys. Rev. Lett.* , **94** (2005) 218702.
- [27] REICHHARDT C. J. O. and BASSLER K. E., *J. Phys. A* , **40** (2007) 4339.
- [28] BASSLER K. E., LEE C. and LEE Y., *Phys. Rev. Lett.* , **93** (2004) .
- [29] SZEJKA A. and DROSSEL B., *Eur. Phys. J. B* , **56** (2007) 373.
- [30] TONG A. H. Y., LESAGE G. and BADER G. D., *Science* , **303** (2004) 808.
- [31] LUSCOMBE N. M. and ET AL. M. M. B., *Nature* , **431** (2004) 308.
- [32] TRACHTENBERG J. T., CHEN B. E., KNOTT G. W., FENG G., SANES J. R., WALKER E. and SVOBODA K., *Nature* , **420** (2002) 788.
- [33] GROSS T. and BLASIUS B., *J. Roy. Soc. Interface* , **5** (2008) 259.
- [34] THIEFFRY D., HUERTA A., PEREZ-RUEDA E. and COLLADO-VIDES J., *BioEssays* , **20** (1998) 433.
- [35] AMARAL P. P., DINGER M. E., MERCER T. R. and MATTICK J. S., *Science* , **319** (2008) 1787.
- [36] REIK W., DEAN W. and WALTER J., *Science* , **293** (2001) 1089.
- [37] ENGERT F. and BONHOEFFER T., *Nature* , **399** (1999) 66.
- [38] VAN OUYEN A., *Network: Computation in Neural Systems* , **12** (2001) R1 .