

Neuropercolation: A Random Cellular Automata Approach to Spatio-Temporal Neurodynamics ^{*}

Robert Kozma¹, Marko Puljic¹, Paul Balister¹, Bela Bollobas¹,
and Walter J. Freeman²

¹ Department of Mathematical Sciences, University of Memphis,
Memphis, TN 38152-3240, USA

rkozma@memphis.edu, URL: <http://cnd.memphis.edu>

² Division of Neurobiology, University of California at Berkeley
Berkeley, CA 94720-3200, USA

wfreeman@socrates.berkeley.edu, URL:<http://sulcus.berkeley.edu>

Abstract. We outline the basic principles of neuropercolation, a generalized percolation model motivated by the dynamical properties of the neuropil, the densely interconnected neural tissue structure in the cortex. We apply the mathematical theory of percolation in lattices to analyze chaotic dynamical memories and their related phase transitions. This approach has several advantages, including the natural introduction of noise that is necessary for system stability, a greater degree of biological plausibility, a more uniform and simpler model description, and a more solid theoretical foundation for neural modeling. Critical phenomena and scaling properties of a class of random cellular automata (RCA) are studied on the lattice \mathbb{Z}^2 . In addition to RCA, we study phase transitions in mean-field models, as well as in models with axonal, non-local interactions. Relationship to the Ising universality class and to Toom cellular automata is thoroughly analyzed.

1 Introduction

Information encoding in the form of oscillatory patterns has advantage compared to fixed-point (static) memories. A number of researchers acknowledged the limitation of static approaches, and have worked around it. Grossberg has proposed adaptive resonance theory network which is a sophisticated topological arrangement of components [1]. Hopfield proposed his famous associative memory network, which is a recurrent system [2]. Elman uses processing elements with feedback as memory devices [3]. Cellular nonlinear neural network (CNN) represent another paradigm showing great promise by implementing cellular automata theory in the form of a programmable memory device [4]. In a separate development, KIII nets have been introduced which represent a prototype of dynamic memory devices based on information encoding in aperiodic (chaotic) attractors [5].

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What distinguishes brain chaos from other kinds is the filamentous texture of neural tissue called neuropil, which is unlike any other substance in the known universe [6, 7]. Neural populations stem ontogenetically in embryos from aggregates of neurons that grow axons and dendrites and form synaptic connections of steadily increasing density. At some threshold the density allows neurons to transmit more pulses than they receive, so that an aggregate undergoes a state transition from a zero point attractor to a non-zero point attractor, thereby becoming a population. Such a property has been described mathematically in random graphs, where the connectivity density is an order parameter that can instantiate state transitions [8, 9].

Information processing in the proposed chaotic dynamical memories is closely related to percolation phenomena [10, 7, 11]. For about forty years now, percolation theory has been an active area of research at the interface of probability theory, combinatorics and physics [12]. Interest in various aspects of standard percolation remains high, including estimates of critical probabilities [13, 14]. There has been much work on the family of processes know as bootstrap percolation [15 - 19]. Many percolation problems exhibit phase transitions, where for p less than some critical probability p_{crit} only finite clusters exist, and for $p > p_{crit}$ infinite clusters almost surely exist. We propose to apply the mathematical theory of percolation in lattices to analyze dynamical neural systems and their related phase transitions. First the basics of bootstrap percolations are described. This is followed by the introduction of several generalizations of standard percolation, which we call neuropercolation. Generalizations include the biologically motivated activation and deactivation functions, random transition rules, and non-local interactions. Non-local interactions mean a rewiring of the connections, which is related to small-world phenomena [20]. Related work on percolations in small-world models is described in [21, 22].

First we describe theoretical results on mean field models, and critical behavior observed in local RCAs. This is followed by the analysis of non-local models and the comparison of their behavior with Ising universality class and Toom automata [23]. Finally, we discuss the directions of combining populations of nonlinear processing elements to multi-layer architecture, in order to describe dynamic processes in cortices.

2 Model Description and Activation Density

Here we introduce the mathematical formalism of neuropercolation. A key parameter of the system is the probability p , which characterizes the stochastic nature of the transition rule. We will see that the random initialization is of secondary importance when having random transition rule. In the following considerations we describe effect related to this probability p .

Let A be a finite subset of \mathbb{Z}^d containing the origin $\mathbf{0} = (0, \dots, 0)$ and let $p: 2^A \rightarrow [0, 1]$ be a function that assigns for each subset $S \subseteq A$ a probability p_S . We define a stochastic process as a sequence of subsets $X_t \subseteq \mathbb{Z}^d$ by including each $\mathbf{x} = (\mathbf{x}_1, \dots, \mathbf{x}_d)$ in X_{t+1} independently with probability p_S where $S =$

$\Lambda \cap (X_t - \mathbf{x})$ and $X_t - \mathbf{x}$ is the translate of X_t by $-\mathbf{x}$. We start the process with X_0 given by some specified distribution. We call such a process a *random cellular automaton* on \mathbb{Z}^d . Usually we define Λ to be the closed neighborhood of \mathbf{z} , consisting of all points unit distance from \mathbf{z} together with \mathbf{z} itself. Similarly we can define random cellular automata on the d -dimensional torus $\mathbb{Z}_{n_1} \times \dots \times \mathbb{Z}_{n_d}$, or on the corresponding finite grid (with suitable boundary conditions imposed). We start by considering p of a certain special form. Assume p_S depends only on the cardinality of S and whether or not the site itself is active ($z \in S$). We write $p_r^{(0)}$ in place of p_S when $|S| = r$ and $z \notin S$ and $p_r^{(1)}$ when $|S| = r$ and $z \in S$. We shall call these models *isotropic*. Isotropic models are substantially more restrictive than the general case, but they still have complex behavior. We call the model *fully isotropic* if $p_r^{(0)} = p_r^{(1)} = p_r$ for all r . In this case, the site itself is treated on the same basis as its neighbors. By complementing the sets X_t we obtain a new model with $p_r^{(i)} = 1 - p_{|A|-i}^{(1-i)}$ (or more generally, $p'_S = 1 - p_{A \setminus S}$). We call the model *symmetric* if it is the same as its complement, so $p_r^{(1)} = 1 - p_{|A|-r}^{(0)}$ for all r (or $p_S = 1 - p_{A \setminus S}$ for non-isotropic models). For symmetric models, there is a further symmetry. If we complement every other X_t , then this is equivalent to a new symmetric model with p_S replaced by $p_S^* = 1 - p_S$. We call this new model the *dual* model of a symmetric model (p_S). Phases in the original model with density not equal to 0.5 will give oscillating phases in the new model with period two [24]. Random cellular automata generalize deterministic cellular automata such as Conway's game of life. Given the complexity of deterministic automata it should not come as a surprise that random automata display extremely complex behavior.

3 Mean Field Models

On a finite grid or torus, we can compare random automata with the corresponding *mean field* model over \mathbb{Z}^2 . In the mean field model instead of taking $|A| - 1$ specified neighbors, we take $|A| - 1$ elements of the grid at random (with replacement). It is clear that the mean field model does not depend on the topology of the grid, and the only information of relevance in X_t is given by its cardinality $|X_t|$. We define x_t to be $|X_t|/N$ where N is the size of the finite grid or torus. Thus $x_t \in [0, 1]$ gives the density of points in X_t . Let us consider the symmetric fully isotropic model $p_0 = p_1 = p_2 = p$. It can be shown that for the mean field model, this has one fixed point at $x = 0.5$ for $p \in [\frac{7}{30}, \frac{1}{2}]$, but for $p < \frac{7}{30} = .2333$ the fixed point $x = 0.5$ is unstable and there are two other fixed points which are stable [24]. Figure 1 illustrates the average density and its variance for various p values.

Our real interest lies in random cellular automata on \mathbb{Z}^2 rather than the mean field models. So one question is how well do the mean field models approximate the corresponding random automata? Unfortunately, the answer is 'not very well in general'. Since the mean field model is equivalent to that of a fully isotropic model, the best we could hope for is that it approximates fully isotropic

models. However, even fully isotropic models can exhibit behavior that is richer than that of the mean field models. The theoretical analysis of local RCA is extremely difficult. Recently, a rigorous mathematical derivation of properties of phase transitions in RCA with very small $p \approx 0$ has been given by [25]. Phase transitions near the critical point are not tractable by rigorous mathematical tools at present. Accordingly, we rely on computer simulations with local and non-local RCAs in the following considerations.

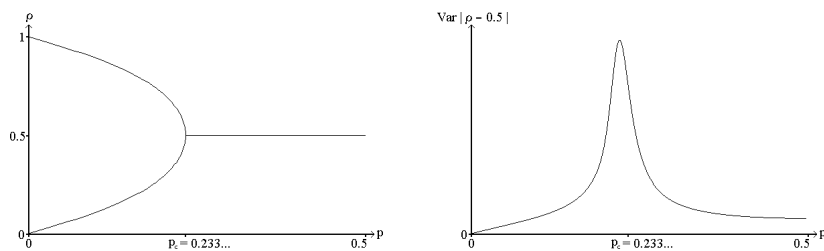


Fig. 1. Activation density ρ of the mean field with $p_c = 0.233$. The variance of $|\rho - 0.5|$ has a sharp increase as p approaches p_c .

4 Simulation of RCA on Finite Lattices

4.1 Phase Transition in Local RCA

The simulation was run on a 2-dimensional torus with noisy majority rule with $4 + 1$ local neighbors. All the sites are updated simultaneously at a certain time t . In this section we use the statistical physics methodology of finite size scaling that has been developed for Ising systems and consecutively applied to various cellular automata, including Toom automata [26, 23, 27]. Here we summarize the major results and conclusions; for details, see [28]. We define the activation density across the torus as follows:

$$x_t = \frac{X_t}{N_{total}}, \quad (1)$$

where, X_t denotes the number of active sites at time t , and N_{total} is the total number of sites in the torus. The average density over n iterations is defined as:

$$\rho = \frac{1}{n} \sum_{t=1}^n x_t. \quad (2)$$

We define the parameter of magnetization as the distance to the known fixed point $\frac{1}{2}$

$$\langle |m| \rangle = \frac{1}{n} \sum_{t=1}^n \left| x_t - \frac{1}{2} \right|. \quad (3)$$

The system exhibits three basic behaviors depending on p . For p values below the critical probability p_c , the system is of ferromagnetic type, see Fig. 2a (left panel). For p values greater than p_c , the system is like the paramagnetic state in Ising model, Fig. 2c (right panel). When p approaches the critical probability, large clusters are formed in the lattice; see Fig. 2b (center panel). In order to obtain a quantitative characterization of these observations, we have conducted a detailed analysis of critical exponents using finite size scaling theory.

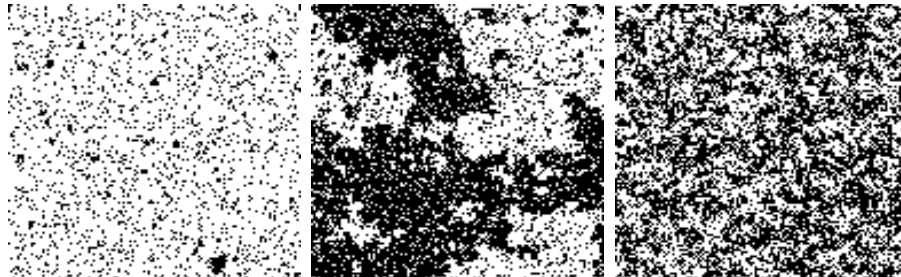


Fig. 2. Illustration of the three regimes. They correspond to the following states in Ising models: (a) $p = 0.11 < p_c$ - ferromagnetic; (b) $p = 0.13423 \approx p_c$ - critical; (c) $p = 0.20 > p_c$ - paramagnetic.

4.2 Estimation of Critical Probability and Critical Exponents

In addition to the magnetization, we introduce the susceptibility χ and the correlation length ξ . In the infinite-size limit, the following power laws are expected to hold:

$$m \sim (p - p_c)^\beta \text{ for } p \geq p_c \quad (4)$$

$$\chi \sim |p - p_c|^{-\gamma} \text{ for } p \rightarrow p_c \quad (5)$$

$$\xi \sim |p - p_c|^{-\nu} \text{ for } p \rightarrow p_c \quad (6)$$

Finite size scaling theory tells us that the fourth order cumulants of the magnetization are expected to intersect each other at the unique point which is independent of lattice size L . The intersection of the curves determines the critical probability. For example, the fourth order cumulants are defined as

$$U(L, p) = \frac{\langle m^4 \rangle}{\langle m^2 \rangle^2} \quad (7)$$

where " $\langle \rangle$ " means to take the average over all runs. We calculate $U(L, p)$ for several probabilities and for lattice sizes 45, 64, 91, 128. The critical probability estimated from their intersection points is 0.13423 ± 0.00002 . For each RCA configuration, simulations run for $10^8 \sim 10^9$ steps, until they produced a steady

estimation of the fourth order cumulants. The critical exponent, which corresponds to the correlation length ξ , is estimated according to finite size scaling theory [26]:

$$\frac{dU_L}{dp}(p_c) \propto L^{\frac{1}{\nu}} \quad (8)$$

To estimate numerically the derivatives in Eq. 8, we fit four adjacent points with cubic interpolation. This approach has shown numerically precise and stable results [27]. The graphs are depicted in Fig. 3. The exponent were estimated using $\frac{dU}{dP}|_{P_c}$, $\frac{d\log(\langle|m|\rangle)}{dP}|_{P_c}$, $\frac{d\log(\langle m^2 \rangle)}{dP}|_{P_c}$, and $\frac{d\log(\langle m^4 \rangle)}{dP}|_{P_c}$. In the calculations, the previously determined value $p_c = 0.13423$ is used. The average value over all estimations has been used to obtain the final estimation of $\nu = 1.0429$. To estimate the remaining two exponents β and γ , we again apply finite size scaling theory. The results are given in Table 1. Finally, we evaluate the consistency of

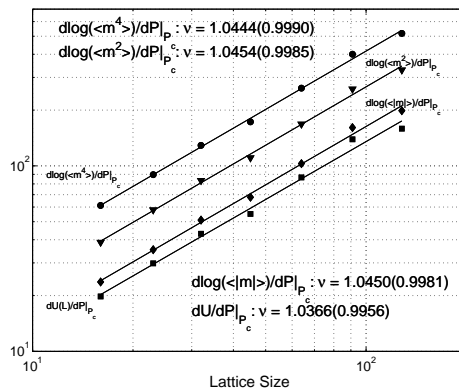


Fig. 3. The exponent of the correlation length is estimated by using finite size scaling theory. The correlation coefficient is displayed in the parenthesis followed the estimated exponent.

the estimation of the critical parameters. It is known that the identity function $2\beta + \gamma = 2\nu$ holds for Ising systems. Let us denote the difference between the left hand side and right hand side of the above equation as $I_{error} = 2\beta + \gamma - 2\nu$. Table 1 includes calculations with Toom cellular automata TCA [27] and coupled map lattices CML [29].

5 Critical States in Non-local Random Cellular Automata

The introduction of non-local interaction is strongly biologically motivated. The neurons have most of their connections in their direct neighborhood, but they have also axonal links with other neurons outside of their surround. Clearly,

Table 1. Comparison of RCA and several other models

	β	γ	ν	I_{error}
RCA	0.1308	1.8055	1.0429	0.02
TCA	0.12	1.59	0.85	0.13
Ising(2D)	0.125	1.75	1	-
CML	0.115	1.55	0.89	0.00

axonal conductance plays an important role in actual neuronal circuits. We approximate long-range axons by replacing several local (direct neighbor) connections with new neighbors randomly selected from the whole lattice. During this rewiring process, each site maintains the same number of connections (4). Therefore, each time a site get a non-local neighbor, we cut out (randomly) one of its local neighbors. In the extreme case, when we cut out all 4 local neighbors of every site, we get an architecture that represents a mean field model.

Table 2. Critical exponents for various connection patterns

	p_c	β	γ	ν	I_{error}
Local	0.1342	0.1308	1.8055	1.0429	0.02
25%(1)	0.1702	0.2262	1.4687	0.9760	0.03
100%(1)	0.2052	0.3943	1.0981	0.8780	0.13
100%(4)	0.2247	0.4141	0.9800	0.8759	0.06

Figure 4 shows the effect of non-local connections on the critical behavior. We observe critical p in models with non-local links. However, in accordance with the intuition, p_c becomes larger as the proportion of remote connections increases. The curves in Fig. 4 belong to models: local (circle), 25% of sites having one non-local link (dot), 100% of sites have one remote link (square), and all sites have all 4 connections remote (diamond). Table 2 includes the critical exponents obtained by the finite size scaling method for each model structure. It is interesting that the various exponents drastically deviate from the values represented by the Ising universality class. At the same time, the hyperscaling relationship remains valid to a significant degree.

6 Discussion and Conclusions

In this paper we have introduced the biologically motivated neuropercolation model. We have identified two critical parameters: the noise level, and the non-locality. Noise modeled the self-sustaining, randomized, steady state background brain activity is the source from which ordered states of macroscopic neural activity emerge, like the patterns of waves at the surfaces of deep bodies of water. Neural tissues, however, are not passive media, through which effects propagate

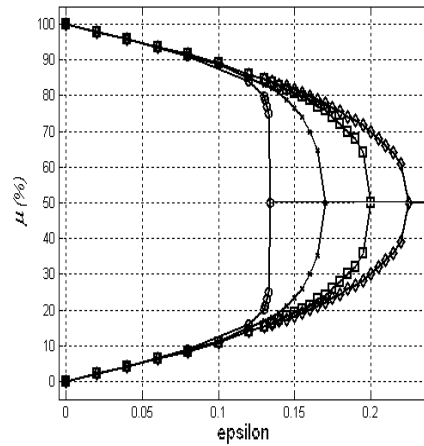


Fig. 4. Magnetization as the function of the noise level for various non-local topologies; circle - local; dot - 25% of sites have 1 nonlocal: 25%(1); square - 100%(1); diamond - 100%(4).

like waves in water. The brain medium has an intimate relationship with the dynamics through a generally weak, subthreshold interaction of neurons. The synaptic interactions of neurons provide weak constraints on the participants, and the resulting covariance appears in the form of spatiotemporal pattern. In the framework of this research we use mathematical tools of percolation theory to explore the information processing mechanisms in brains as the randomized activity of myriads of neurons that provides the basis for self-organization and higher brain functions. The present works belong to the basic building block of our neurodynamic model based on K sets [30, 31]. This is the single KI layer of homogenous elements. A higher complexity level is represented by the KII model which has interacting excitatory and inhibitory populations. KII may exhibit nontrivial limit cycle and aperiodic oscillations. The interaction of several KII sets produces the KIII system with aperiodic spatio-temporal dynamics. KIII has the potential of spontaneous phase transitions propagating in space and time. Research in this direction is in progress.

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