Hierarchical Random Cellular Neural Networks for System-Level Brain-Like Signal Processing

Robert Kozma\textsuperscript{a}, and Marko Puljic\textsuperscript{b,a}

\textsuperscript{a}Center for Large-Scale Integrated Optimization and Networks (CLION)  
University of Memphis, Memphis, TN 38152, USA
\textsuperscript{b}Computational Sciences, Tulane University, New Orleans, LA 70118, USA

Abstract

Sensory information processing and cognition in brains are modeled using dynamic systems theory. The brain’s dynamic state is described by a trajectory evolving in a high-dimensional state space. We introduce a hierarchy of random cellular automata as the mathematical tools to describe the spatio-temporal dynamics of the cortex. The corresponding brain model is called neuropercolation which has distinct advantages compared to traditional models using differential equations, especially in describing spatio-temporal discontinuities in the form of phase transitions. Phase transitions demarcate singularities in brain operations at critical conditions, which are viewed as hallmarks of higher cognition and awareness experience. The introduced Monte-Carlo simulations obtained by parallel computing point to the importance of computer implementations using very large-scale integration (VLSI) and analog platforms.

Keywords: Neurodynamics, Perceptual Information Processing, Random Cellular Automata, Freeman K-sets, Neuropercolation, Phase Transition, Synchronization, Memristor

1. Introduction

Developments in brain monitoring techniques provide increasingly detailed insights into spatio-temporal neurodynamics and to neural correlates of large-scale cognitive processing (Leopold et al., 2003; Friston et al., 2006; Matthews et al., 2006; Freeman and Quiroga, 2012). The experimental findings can be interpreted using dynamic systems theory (Freeman, 1991, 1992).
Accordingly, the state of the brain at any time instant is described by a trajectory over the dynamic attractor landscape with a large number of valleys and hills acting as attractors and repellers. Valleys correspond to memories formed previously by the subject under reinforcement of sensory stimuli as it interacts with the environment. The brain’s basal state is a high-dimensional chaotic attractor with its dynamic trajectory wandering broadly as it explores various parts of the landscape.

Under the influence of external stimuli, cortical dynamics is destabilized and condenses intermittently to a lower-dimensional subspace. This is the act of perception, when the individual identifies the stimulus with a well-defined meaning in the context of its previous experience. The system stays briefly in the condensed, more organized state, which gives rise to a synchronized spatio-temporal amplitude modulation (AM) pattern corresponding to the stimulus in the given context. The AM pattern is meta-stable and it disintegrates as the system returns to the high-dimensional chaotic basal state. This dynamic process can be described as chaotic itinerancy through Milnor's attractor whereas the trajectory of the dynamical system intermittently visits attractor ruins as it traverses across the landscape (Tsuda, 2001; Kozma, 2003). Brain dynamics is viewed as a sequence of phase transitions with intermittent synchronization-desynchronization effects (Freeman, 2004; Kozma and Freeman, 2008; Freeman and Kozma, 2010; Freeman and Quiroga, 2012).

In this work, we utilize Freeman’s neuroscience insights manifested in his hierarchical brain model: the Freeman K-sets (Freeman, 1975). In their original form, K-sets have been described using ordinary differential equations (ODEs) with distributed parameters and with additive and multiplicative noise (Chang and Freeman, 1998a,b). This approach produced significant results, and K-sets have been applied successfully to various classification and pattern recognition problems (Freeman, 1995; Chang and Freeman, 1998c; Kozma and Freeman, 2001), the detection of chemicals (Gutierrez-Osuna and Gutierrez-Galvez, 2003), time series prediction (Beliaev and Kozma, 2007), and adaptive control (Kozma et al., 2007).

Despite their successes, ODE-based K-sets had certain shortcomings (Freeman, 1991, 1995). On the one hand, obtaining stable solutions of nonlinear ODEs and stochastic differential equations describing electrocorticogram (ECoG) activity proved to be often prohibitively difficult and time-consuming on both digital and analog platforms (Principe et al., 2001). In some applications, K-sets exhibited extreme sensitivity to model parameters due to the phenomenon called attractor crowding (Chang and Freeman, 1998c). More-
over, there are unsolved theoretical issues to bridge the vast spatial and temporal scales between microscopic properties of single neurons and macroscopic properties of vast populations of over $10^{10}$ neurons in the mammalian cortex (Freeman, 1992, 1999).

In the past decade, neuropercolation approach has proved to be an efficient tool to implement K-sets using concepts of discrete mathematics and random graph theory (Kozma, 2007). Neuropercolation is a family of probabilistic models based on the mathematical theory of probabilistic cellular automata on lattices and random graphs and it is motivated by structural and dynamical properties of neural populations. The existence of phase transitions has been proven in specific probabilistic cellular automata and percolation models (Kozma et al., 2004; Balister et al., 2006). Neuropercolation extends the concept of phase transitions to large interactive populations of nerve cells. The present study is based on applying neuropercolation to systematically implement Freeman’s principles of neurodynamics.

This report starts with the description of the random cellular neural network model of cortical dynamics characterizing perception and cognitive functions. We develop a hierarchy on neuropercolation models with increasing complexity of structure and dynamics. The hierarchy includes single layer with non-zero fixed point dynamics, double layers with limit cycle oscillations, and interconnected oscillators with chaos and intermittent synchronization-desynchronization transitions. The results are used for the interpretation of electroencephalogram (EEG) and electrocorticogram (ECoG) experiments. We elaborate on computational aspects of large-scale Monte Carlo simulation and various synchronization measures derived by coarse-graining the spatial variables. We conclude with discussions on hardware implementation of the massive parallel computing for large-scale brain simulations on digital and analog platforms.

2. Hierarchical Model of Intentional Neurodynamics

2.1. Basic Principles of the Limbic System Model

Intelligent behavior is characterized by the flexible and creative pursuit of endogenously defined goals. Humans are not passive receivers of perceptual information (Freeman, 1999), they actively search for sensory input. To do so they must form hypotheses about expected future states, and express these as goals. They must formulate a plan of exploration and action, and they must inform their sensory and perceptual apparatus about the expected future
input in a process called re-afference. They must manipulate their sense organs, take information in the form of samples from all of their sensory ports, then generalize, abstract, categorize, and combine into multi-sensory percepts (Gestalts). The cyclic operation of prediction, testing by action, sensing, perceiving, and assimilation is called intentionality (Nunez and Freeman, 1999). Intentionality is endogenously rooted in the agent and cannot be implanted into it from outside by any external agency. Intentionality is manifested in and evolved through the dynamical change in the state of the agent upon its interaction with the environment (Kozma and Fukuda, 2006).

In the intentional neurodynamic model, meaningful knowledge is continuously created, processed, and dissipated in the form of sequences of oscillatory patterns of neural activity distributed across space and time (Freeman, 1999; Kozma et al., 2007; Kozma and Freeman, 2009). Cognition granulates the seemingly homogeneous temporal sequences of perceptual experiences into
meaningful and comprehensible chunks of concepts and complex behavioral schemas, which are accessed during future actions and decisions. The oscillatory spatio-temporal patterns can be viewed as intermittent representations of a generalized symbol system, with which brains compute. These dynamical symbols, however, are not rigid but flexible and they disappear soon after they emerge through spatio-temporal phase transitions.

Intentional behavior is maintained by the limbic system as part of the vertebrate paleocortex (Freeman, 1999). The major parts of the limbic system are the hippocampal formation, hypothalamus, septum, midline forebrain, the amygdala striatum, and brain stem, see Fig.1. The olfactory system is part of the limbic system, while other sensory systems in the neocortex are connected to the limbic system by sparse long intracortical projections. The convergence location and output are provided by the amygdala. In the next section, a large-scale brain model is developed, which implements major components of the limbic system as part of a cortical hierarchy.

2.2. Modeling the Limbic System by Freeman K-sets

2.2.1. Biological Motivation

We propose a hierarchical approach to spatio-temporal neurodynamics, based on Freeman K sets. Low-level K sets were introduced in the 70s, named in the honor of Aharon Kachalsky, an early pioneer of neural dynamics (Katchalsky, 1971; Freeman, 1975, 1992, 1999). K sets are multiscale models, describing increasing complexity of structure and dynamics. K sets are mesoscopic models, and represent an intermediate-level between microscopic neurons and macroscopic brain structures. K-sets are topological specifications of the hierarchy of connectivity in neuron populations.

When first introduced, K-sets have been modeled using a system of nonlinear ordinary differential equations (ODE) (Freeman, 1975, 1995). K-dynamics predict the oscillatory waveforms that are generated by cortical neural populations. K-sets describe the spatial patterns of phase and amplitude of the oscillations. They model observable fields of neural activity comprising electroencephalograms (EEG), local field potentials (LFP), and magnetoencephalograms (MEG). K sets form a hierarchy of cell assemblies with components starting from K0 to KIV (Kozma and Freeman, 2003; Freeman and Erwin, 2008).
2.2.2. K0 Sets - Zero Point Attractors

K0 sets represent non-interactive collections of neurons forming cortical microcolumns; a K0 set models a neuron population of \( \approx 10^4 \) neurons. K0 models dendritic integration in average neurons and an asymmetric sigmoid static nonlinearity for axon transmission. The K0 set is governed by a point attractor with zero output and stays at equilibrium except when perturbed. In the original K-set models, K0s are described by a state-dependent, linear 2nd order ordinary differential equation (ODE) (Freeman, 1975):

\[
(ab) \frac{d^2 P(t)}{dt^2} + (a + b) \frac{dP(t)}{dt} + P(t) = F(t). \tag{1}
\]

Here \( a \) and \( b \) are biologically determined time constants. \( P(t) \) denotes the activation of the node as function of time. \( F(t) \) includes an asymmetric sigmoid function \( Q(x) \) acting on the weighted sum of activation from neighboring nodes and any external input.

2.2.3. KI Sets - Non-Zero Point Attractors

KI sets are made of interacting K0 sets, either excitatory or inhibitory with positive feedback. The dynamics of KI is described as convergence to non-zero fixed point. If KI has sufficient functional connection density, then it is able to maintain a non-zero state of background activity by mutual excitation (or inhibition). KI typically operates far from thermodynamic equilibrium. Its critical contribution is the sustained level of excitatory output. Neural interaction by stable mutual excitation (or mutual inhibition) is fundamental to understanding brain dynamics.

2.2.4. KII Sets - Limit Cycle Attractors

KII sets consist of interacting excitatory and inhibitory KI sets with negative feedback. Examples include the olfactory bulb, hippocampal regions, and the prepyriform cortex. KII has four types of interactions: excitatory-excitatory, inhibitory-inhibitory, excitatory-inhibitory, and inhibitory-excitatory. KII sets are responsible for the emergence of limit cycle oscillation due to the negative feedback between the neural populations. Transitions from point attractor to limit cycle attractor can be achieved through a suitable level of feedback gain or by input stimuli. Systematic analysis has identified regions of stability and transitions between limit cycle and fixed point dynamics (Xu and Principe, 2004; Ilin and Kozma, 2006).
2.2.5. KIII Sets - Chaotic Attractors

KIII sets are made up of multiple interacting KII sets. Examples include the olfactory system and other sensory cortices, and the hippocampus with CA1, CA2, and CA3 sections. KIII sets generate broad-band, aperiodic/chaotic oscillations as background activity by combined negative and positive feedback among several KII populations with incommensurate frequencies. The increase in nonlinear feedback gain that is driven by input results in the destabilization of the background activity and leads to emergence of a spatial amplitude modulation (AM) pattern in KIII. KIII sets are responsible for the embodiment of meaning in AM patterns of neural activity shaped by synaptic interactions that have been modified through learning in KIII layers.

KIII can serve as a associative memory, encoding input data in spatio-temporal AM patterns (Chang and Freeman, 1998c; Kozma and Freeman, 2001). KIII chaotic memories have several advantages as compared to convergent recurrent networks: (1) they produce robust memories based on relatively few learning examples even in noisy environment; (2) the encoding capacity of a network with a given number of nodes is exponentially larger than their convergent counterparts; (3) they can recall the stored data very quickly, just as humans and animals can recognize a learnt pattern within a fraction of a second.

2.2.6. KIV - Itinerant Chaos for Intentionality

KIV sets made up of interacting KIII units to model intentional neurodynamics in the limbic system. KIV exhibits global phase transitions, which are the manifestations of hemisphere-wide cooperation through intermittent large-scale synchronization across the hemisphere. KIV is the domain of Gestalt formation and preafference through the convergence of external and internal sensory signals leading to intentional action (Kozma and Freeman, 2003).

In a simplified model, we have 3 KIII units corresponding to the cortical, hippocampal, and midline forebrain (interoceptor) systems Fig. 2. The olfactory system is modeled by the periglomeruli cell layer (PG), olfactory bulb (OB), anterior olfactory nucleus (AON) and the prepyriform cortex (PC). In the hippocampus we have CA1, CA2, and CA3 as KII sets. The dentate gyrus (DG) is a sheet of neurons interposed between the cortex and CA3. The midline forebrain is modeled by the interoceptory KIII and it consists of the basal ganglia (BG), hypothalamus (HT), septum, and the
Figure 2: KIV model of the limbic system; notations: PG periglomerular; OB Olfactory bulb; AON anterior olfactory nucleus; PC prepyriform cortex; DG dentate gyrus; CA1, CA2, CA3 cornu ammonis sections of the hippocampus; MF midline forebrain; BG basal ganglia; HT hypothalamus; DB diagonal band; SP septum, BS brain stem connected through the amygdala; based on (Kozma et al., 2007).
diagonal band as KII sets. The amygdala provides the convergence point of the KIII sets to achieve goal-oriented action selection for the motor system via the brain stem (BS).

ODE-based K-sets have been used successfully in various applications (Kozma et al., 2007, 2008), however, they have some shortcomings. Differential equations falter to model the broad temporal and spatial dynamic range of neurodynamics and they cannot describe adequately the transitions between levels of brain organization. Differential equations assume some degree of smoothness in the described phenomena, which renders them less suitable to the characterization of sudden transitions and singular changes in neurodynamics. Neuropercolation and random graph theory offers a new approach to address these issues.

3. Neuropercolation Model of Intentional Neurodynamics

3.1. Neuropercolation Background

Concepts of random graph theory have been used to establish rigorous models of large-scale networks (Erdős and Rényi, 1959; Bollobás, 1984; Bollobás and Riordan, 2006). To extend these studies to the domain of brain networks, these models are structured in accordance with cortical architectures. We use the hierarchy of interactive populations in networks as developed in Freeman K models (Freeman, 1975), but replace differential equations with probability distributions from the observed random networks that evolve in time. The corresponding mathematical object is called neuropercolation (Kozma, 2007) using concepts of random cellular automata. Neuropercolation theory provides a mathematical approach to describe phase transitions and critical phenomena in large-scale cortical networks. Neural percolation model is a natural mathematical domain for modeling collective properties of brain networks, especially near critical states, when the behavior of the system changes abruptly with the variation of some control parameters.

Neuropercolation considers populations of cortical neurons which sustain their background state by mutual excitation, and their stability is guaranteed by the neural refractory periods. Neural populations transmit and receive signals from other populations by virtue of small-world effects (Watts and Strogatz, 1998; Sporns, 2006). Large-scale, spatially coherent AM patterns emerge from and dissolve into the random background activity by spontaneous symmetry breaking (Freeman and Vitiello, 2008). The formation of a percept from sensory input is by a phase transition from a disorganized phase
to a organized phase (Freeman et al., 2012). Tools of statistical physics and finite-size scaling theory are applied to describe critical behavior and phase transitions in the neuropil.

3.2. Lattice Model of Random Cellular Automata (RCA)

Consider a graph $G(V, E)$ consisting of a set of vertices $V = \{v_i| i \in [1,\ldots,n]\}$ and directed edges $E = \{(v_i, v_j)| i, j \in [1,\ldots,n]\}$. The neighborhood of vertex $v_i$ is defined as the set of vertices $\mathcal{N}(v_i)$, from which there is an edge pointing to vertex $v_i$. A simple example over the 2-dimensional square lattice is the local neighborhood, when neighborhood $\mathcal{N}(v_i)$ contains the four direct lattice neighbors of vertex $v_i$ plus the vertex itself. We consider non-local neighborhoods as well, when some local edges are rewired by randomly selected remote edges from the lattice. Remote rewiring corresponds to long axonal connections in the neural tissue.

A binary function $s$ is defined over the vertices, i.e., any vertex $v_i$ can assume one of two possible states $s(v_i) \in \{0,1\}$. At time $t = 0$, $s(v_i)$ is randomly set to 0 or 1. For $t = 1,2,\ldots,T - 1$, an iterative update rule is applied simultaneously over all vertices. In this work we use the probabilistic version of the majority rule, i.e., a given vertex follows the majority state of its neighbors with probability $\omega$ close to 1. At the same time, this vertex may follow the state of the minority of neighbors with probability $1 - \omega$, which is close to 0. In Fig. 3, full and open circles denote inactive and active nodes, respectively; edges $(a, x), (c, x), (d, x), \text{and} (x, x)$ are excitatory, while $(b, x)$ is inhibitory. The majority of the neighbors of node $x$ have inactive (0) contribution to $x$ at time $t - 1$, so it remains inactive at time $t$ with probability $\omega$, while it may become active with probability $1 - \omega$.

3.3. Rewiring Edges in the Network

Edge rewiring is a random process in which the vertex degrees are preserved, but the locations of edges are changed from a regular lattice configuration to a partially random network. The applied random rewiring algorithm is as follows:

1. a given number of $n \leq |V|$ vertices are selected at random;
2. one incoming edge from each selected vertex are disconnected;
3. edges are reconnected at random to vertices with a missing edge.
Figure 3: Illustration of majority voting rule in 2-dimensional lattices with local neighborhood; full circles show 3 inactive nodes, open circles show 2 active nodes. Nodes $x$, $a$, $c$, and $d$ are excitatory, while node $b$ is inhibitory. In the given scenario, $s(x,t)$ is likely to remain inactive with probability $\omega$.

Figure 4: 2-dimensional torus of size $4 \times 4$ when the first row/column is connected with last row/column. Each vertex has a connection to itself. Right: connectivity after rewiring 2 edges out of the total 80 edges ($= 5 \times 4 \times 4$).

The above algorithm assures that the original vertex degrees are preserved after rewiring; see Fig. 4. Random rewiring results in reduced path lengths among vertices, as compared to completely regular, local connectivity. It will be shown that rewiring changes the nature of the dynamics from pure diffusion-like process, and the changes propagate across the lattice at increased speed.

3.4. Estimating Critical Parameters

The methodology previously developed for Ising spin glass systems (Binder, 1981) is applied here to characterize processes in RCA. The activation density at time $t$ is defined as the average activation level in the lattice $\rho_t = 1/n \sum_{i=1}^{n} s(v_i)$. If the number of active and inactive sites is equal at time $t$, $\rho_t = 0.5$. This corresponds to a basal state in magnetic materials with no magnetization. Deviations from the 0.5 level at any time gives the mag-
nitude of the magnetization as \( m(t) = |\rho_t - 0.5| \). The expected value of \( m \) is estimated for a time series of length \( T \) as follows: \( < m(t) > \approx 1/T \sum_{t=1}^{T} |\rho_t - 0.5| \). It is of interest to evaluate various statistical characteristics of the lattice dynamics, including the susceptibility \( \chi \) and the correlation length \( \xi \), see (Makowiec, 1999).

The fourth order cumulants are defined as \( U(n, \omega) = \frac{<m^4>}{<m^2>^2} \), where \( n \) is the lattice size, and \( \omega \) is the parameter describing randomness. The fourth order cumulant measures the peakedness of the probability density function. Finite size scaling theory holds for a large class of lattices (Binder, 1981; Makowiec, 1999) and it tells that the fourth order cumulants are expected to intersect at a unique point which is independent of the lattice size. At this critical point, the shape of the probability distribution suddenly changes from bimodal to unimodal. This unique point is given by the critical probability \( \omega_0 \). For Ising systems, magnetization, susceptibility, and correlation length satisfy a power law scaling behavior near criticality at \( \omega \approx \omega_0 \), namely:

\[
m \sim |\omega - \omega_0|^{-\beta}, \quad \chi \sim |\omega - \omega_0|^{-\gamma}, \quad \xi \sim |\omega - \omega_0|^{-\nu}
\]  

(2)

Here \( \beta, \gamma, \) and \( \nu \) are critical exponents corresponding to magnetization, susceptibility, and correlation length, respectively. In order to test the consistency of the critical behavior, the identity relationship \( 2\beta + \gamma = 2\nu \) has been calculated, which holds for Ising systems (Binder, 1981). This identity is considered as a measure of the quality of the estimation of the critical exponents is a given system. The above statistical properties of neuropercolation processes have been evaluated based on computer simulations.

3.5. Critical Behavior in a 2D Lattice with and without Rewiring

The results summarized in this section indicate that neuropercolation in a 2-dimensional lattice (torus) exhibits critical behavior and it belongs to the weak Ising class (Kozma et al., 2005). We introduce the following general notation: \( G^{d,k}(q, \theta, \kappa) \) stands for a lattice graph in \( d \)-dimensions, containing \( k \) inhibitory populations, with \( q \) indicating the proportion of rewired connection in each layer. The case of \( q = 0 \) describes completely regular lattice connections, while \( q = 1 \) means that all connections are selected at random as in mean field models. An intermediate value of \( q \) characterizes a system with some rewiring, just as in small-world models (Watts and Strogatz, 1998).

\( \theta \) and \( \kappa \) are parameters describing the connections between excitatory and inhibitory layers and will be described in the next section. Here we use
Figure 5: Illustration of 2D activation patterns of a RCA without rewiring; the critical probability is $\omega_0 = 0.866$. Case (a) is ferromagnetic regime ($\omega > \omega_0$); (b) critical state ($\omega \approx \omega_0$) with large-scale clusters; (c) paramagnetic regime $\omega < \omega_0$.

a single 2-dimensional layer $d = 2$ without inhibition ($k = 0$), where $\theta$ and $\kappa$ play no role. In this case, for the sake of simplicity, we use a compressed graph notation $G^{2,0}(q)$ showing only parameter $q$ giving the % of rewired connections. Snapshots of activity distributions in the 2-dimensional lattice are shown in Fig. 5 for various levels of $\omega$. The emergence of large scale clusters is clearly seen at critical conditions in Fig. 5b.

Table 1: Critical Parameters of $G^{2,0}(0)$ and several other models*

<table>
<thead>
<tr>
<th>Critical Parameters</th>
<th>Neuropercolation $\beta$</th>
<th>Toom $\beta$</th>
<th>Ising $\beta$</th>
<th>$\gamma$</th>
<th>$\nu$</th>
<th>Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA</td>
<td>0.1308</td>
<td>0.12</td>
<td>0.125</td>
<td>1.8055</td>
<td>1.0429</td>
<td>0.02</td>
</tr>
<tr>
<td>CA</td>
<td>0.12</td>
<td>0.15</td>
<td>1.75</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2D</td>
<td>0.125</td>
<td>1.75</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: $G^{2,0}(0)$ is a 2D lattice graph in isolation, without rewiring

Table 1 shows critical exponents of the 2D lattice as defined in Eq.2. We consider the case of the RCA lattice graph without rewiring $G^{2,0}(0)$, Ising system in 2-D (Binder, 1981), and Toom cellular automaton (TCA) (Makowiec, 1999). The 'Error" in the last column indicates the error of the identity function $2\beta + \gamma = 2\nu$ among the critical exponents. As Table 1 shows, the identity function is satisfied with high accuracy in the studied neuropercolation RCA model. This indicates that $G^{2,0}(0)$ exhibits behavior
close to an Ising model, i.e., it belongs to the "weak Ising class" (Kozma et al., 2005).

| Critical Parameters of $G^{2,0}(q)$ with Rewiring |
|------------------|--------------------|--------------------|--------------------|--------------------|
|                  | $q = 0$            | $q = 6.25$         | $q = 12.5$         | $q = 100$          |
| Parameters       | Local              | Small World        | Small World        | Global             |
| $\beta$          | 0.1308             | 0.3071             | 0.4217             | 0.4434             |
| $\gamma$         | 1.8055             | 1.1920             | 0.9873             | 0.9371             |
| $\nu$            | 1.0429             | 0.9504             | 0.9246             | 0.9026             |
| Error            | 0.02               | 0.09               | 0.09               | 0.02               |

The critical exponents obtained for models with various degrees of small-world effects are given in Table 2; notations are same as in Table 1. In the case of small world and global systems, the critical exponents change significantly as compared to local models, but the hyperscaling relationship still approximately holds, i.e., the Error function is close to 0. This result shows that also the non-local neuropercolation systems belong to the weak Ising class (Kozma et al., 2005).

4. Statistical Properties of Hierarchical Neuropercolation Models

4.1. Narrow-band Oscillations in Lattices with Inhibitory Feedback

We consider graph $G^{2,1} = G^{2,0}_E \cup G^{2,0}_I$, where $G^{2,0}_E$ and $G^{2,0}_I$ are excitatory and inhibitory layers, respectively. The structure of the corresponding two-layer graph is shown in Fig. 6. Within each subgraph the edges are excitatory. In addition, edges from $G^{2,0}_E$ to $G^{2,0}_I$ are excitatory and edges from $G^{2,0}_I$ to $G^{2,0}_E$ are inhibitory. For example, graph $G^{2,1}(5, 2.5) = G^{2,0}_E(5, 2.5) \cup G^{2,0}_I(5, 2.5)$ has $q = 5\%$ of edges rewired at random in both layers and $\theta = 2.5\%$ between excitatory-inhibitory nodes.

When $\omega$ is varied while all other parameters are fixed in the presence of inhibition, two critical points have been found, $\omega_0$ and $\omega_1$ (Puljic and Kozma, 2008). The two critical points demarcate the region within which large-scale narrow-band oscillations exist. Namely, $\omega_0$ marks the transition from unimodal regime to bimodal (limit cycle) oscillations. $\omega_1$ marks the transition point from bimodal to quadro-modal distribution where large-scale synchronized oscillations diminish. For example, $G^{2,1}(5, 2.5)$ has $\omega_0 \approx 0.840$
and ω₁ ≈ 0.860, Fig. 7. G²,₁(5, 3.75) has ω₀ ≈ 0.845 and ω₁ ≈ 0.865. Note that the lattice model without inhibition gives a single critical point without a region of large-scale synchronized regime. The emergence of a region of large-scale synchrony with prominent narrow-band oscillations is crucial for building a hierarchy of cognitively-relevant brain models using interacting excitatory-inhibitory populations.

4.2. Broad-Band Oscillations in Twin Excitatory-Inhibitory Layers

We consider graph G²,₂ = G²,₁ ∪ G²,₁, see Fig. 8. Edges from G²,₀ and G²,₁ to G²,₀ and G²,₁ are inhibitory, and all other edges are excitatory. Graph G²,₂ is made of two twin oscillators, and it exhibits four possible critical points, ωᵢ, i = 0, 1, 2, 3 (Puljic and Kozma, 2010). When ω < ω₀ the vertex states are equiprobable and we observe unimodal regime. When ω₀ < ω < ω₁, the two oscillators can produce large-scale synchronization regime with narrow-band, bimodal oscillations. When ω₁ < ω < ω₂, the two connected oscillators cannot agree on a common mode, yet cannot ignore each other, so together they generate aperiodic (chaotic) background activity. When ω₂ < ω < ω₃, only one oscillator oscillates at narrow band, and together with the other oscillator it generates a complex signal with mixed narrow band and broad-band components. Finally, for ω > ω₃ none of the oscillators produce prominent oscillations and the resulting signal is a broad background noise.

Table 3 shows the evolution of the critical regions in two coupled twin oscillators as the connection density κ between the two oscillators varies. By decreasing κ, the large-scale synchronization regime narrows. This is expected, as with no connection between the oscillators (κ = 0) the two
oscillators work independently and thus cannot produce large-scale synchronized regime.

Table 3: Regions of Large-Scale Synchrony of 2 Coupled Oscillators

<table>
<thead>
<tr>
<th>$G^{2,2}$ Parameters</th>
<th>$\omega_0$</th>
<th>$\omega_1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$G^{2,2}(5, 2.5; 5, 3.75; 0.3125)$</td>
<td>0.836</td>
<td>0.840</td>
</tr>
<tr>
<td>$G^{2,2}(5, 2.5; 5, 3.75; 0.6250)$</td>
<td>0.834</td>
<td>0.842</td>
</tr>
<tr>
<td>$G^{2,2}(5, 2.5; 5, 3.75; 1.2500)$</td>
<td>0.831</td>
<td>0.845</td>
</tr>
</tbody>
</table>

*Note: $G^{2,2}(q_1, \theta_1; q_2, \theta_2; \kappa) = G^{2,1}(q_1, \theta_1) \cup G^{2,1}(q_2, \theta_2)$, where $\cup$ is realized by $\kappa$ links between the two $G^{2,1}$ units.

5. Collective Dynamics of Hierarchical Models and Their Relevance to Limbic Systems

5.1. Quantifying Synchronization in the Lattice

Intermittent large-scale synchronization is an important property of intentional brain dynamics as observed by ECoG and EEG experiments. To quantify synchronization effects, we divide the lattice graph into channels
using some granulation level. An example of such granulation is shown in Fig. 9 by dividing 64 lattice elements into 16 channels. We then compare the activation of each channel with the overall activation of the graph.

When measuring synchrony, first we find the dominant frequency over the entire graph, or ensemble, for the duration of experiment with the discrete Fourier transform. Then, we set a time window $W$ based on the dominant frequency, and the correlations are determined according to this window between the channels and ensemble average. We correlate each channel with the ensemble for duration of $W$ at each time step. Then, the dominant frequency is found for each channel and the ensemble average and their phase shifts are compared. The phase difference at the dominant frequencies measures the phase lag between the channel and the ensemble. The vector composed of phase lags for each channel describes the synchrony of graph components as it evolves in time.

5.2. Results of Synchronization Measurements

Results for a single 2D lattice graph $G^{2,0}$ are shown in Fig. 10a. The phase lag values between channels show random distribution, i.e., the channels do not show synchronous dynamics. Fig. 10b shows the calculated power spectral densities, which exhibit a scale-free behavior over a wide frequency band of 4 Hz to 60 Hz.

Next we show results of phase synchronization studies for graph $G^{2,1}(5, 2.5)$ with an excitatory and inhibitory layer, see Fig. 11. We can distinguish several regimes. For $\omega < \omega_0$ in Fig. 11a, the channels are not synchronous and the phase values are distributed broadly. In the case of $\omega_0 < \omega < \omega_1$ in Fig.
Figure 9: Example of dividing 64 labeled vertices of $G^{2,0}$ into 16 channels. Edges are not shown.

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Figure 10: Illustration of phase synchronization results for $G^{2,0}(5)$ with 144 channels. (a) Phase lag values between channels; the channels show random phase relationships, i.e., they are never in synchrony. (b): Power spectral density function for $\omega = 0.86$. 

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Figure 11: Phase synchronization results for an oscillator graph $G^{2,1}(5, 2.5)$; (a) phase lags for $\omega < \omega_0$; the channels are not synchronous. (b) When $\omega_0 < \omega < \omega_1$, the phase lags are drastically reduced indicating significant synchrony in the graph. For high values of $\omega < \omega_0$, the channels get de-synchronized.

Figure 11b, the phase lags are drastically reduced indicating significant synchrony in the graph. For high values of $\omega < \omega_0$, the channels get de-synchronized (not shown).

We have shown previously that the behavior of coupled oscillator graphs $G^{2,2}$ is governed by four critical parameters $\omega_0 < \omega_1 < \omega_2 < \omega_3$. Of special interest is the large scale synchronization-desynchronization transition regime between $\omega_0$ and $\omega_1$. Fig. 12 illustrates synchronization effects in $G^{2,2}(5, 2.5; 5, 3.75; 1.25)$. When $\omega \approx \omega_0$, there are frequent transitions between periods of extended synchrony and brief periods of phase dispersion without synchrony, Fig. 12a. When $\omega_0 < \omega < \omega_1$, phase dispersion periods are less frequent, more crisp, and last for brief time instant only, Fig. 12b. Two connected oscillators with different frequencies generate large-scale synchronization and chaotic background activity. Figs. 12c-d show power spectral densities (PSDs) of two coupled oscillators. The PSDs exhibit scale-free behavior over a wide range of frequencies, while there is a increased PSD
amplitude related to oscillations at lower frequencies in Fig. 12d.

6. Discussions on Computational and Hardware Platforms

6.1. Computational Aspects of Monte-Carlo Simulations

Our mathematical model uses a hierarchy of 2-dimensional lattices to describe phase transitions for neuromorphic computation. There are several rigorous mathematical results in this field (Balister et al., 2006; Bollobás and Riordan, 2006; Bollobás et al., 2009), however, most of the progress results from massive computer simulations of critical behavior in lattice models. To find the critical points using Binder’s method, we need to compare kurtosis measures of at least for two graphs of different sizes. For example, to find the critical points of two coupled oscillators, we set two equivalent graphs of different order, each made of four subgraphs of size approximately 100 × 100, or even larger. Thus the number of nodes is in the order of 40,000 for each analyzed graph. The evolution of a graph is simulated for 100 million steps in parallel, with 8 threads, at 2GHz speed per thread. This calculation takes 5 days on the available hardware to achieve the required statistical accuracy for one configuration. So it takes about 8 days and 16 cores (one for each thread) to find statistics for one unique set of parameters describing a graph.

In search for critical $\omega$ values, hundreds of $\omega$ points must be evaluated for each graph. This requires over 500 days on a machine with 16 cores, having 2GHz per core. To get better results, one should approximate critical regions with at least 4 graphs, leading to doubling the time and space costs. The statistics presented in this work were collected through non-stop simulations on around 100 cores through the past couple of years at the University of Memphis High Performance Computing Cluster (HPC). Clearly, advanced hardware platforms are required to achieve the ambitious goals of brain-like signal processing using intentional dynamical models of the limbic system.

6.2. Neuromorphic Hardware Platforms

To achieve parallelism for the task ahead, we partition the set of vertices into multiple components. The most efficient way is to partition vertices into a number of subsets that is equal the number of available computer processors. Vertices and their neighborhoods are stored in shared memory, which is divided so that each piece holds one component. A piece of memory is accessed by one processor, which evolves the set of vertices. For each vertex at each time step, the vertex neighborhood is checked to determine
Figure 12: Behavior of $G^{2,2}(5, 2.5; 5, 3.75; 1.25)$: (a) phase dispersion diminishes when $\omega \approx \omega_0$; (b) phase differences are less frequent and more crisp when $\omega_0 < \omega < \omega_1$; (c) power spectral density of two coupled oscillators that are out of synchrony; (d) power spectral density of two coupled oscillators in synchrony.
the vertex’s state. When the processor finishes checking the (majority) rule of all vertices, it is paused to wait until all the processors finish their evaluation. When all the processors have finished their task on their vertices, a new cycle begins. This synchronizes the evolution of the entire graph.

Cellular automata with some level of rewiring have enormous potential for modeling complex systems. A prominent example of such architecture is the cellular neural network (CNN) (Chua and Roska, 1993). Cellular networks can be viewed as models of a spatially decentralized system made up of a number of individual processors. The direct communication between processors is limited to the interactions in the processor neighborhood. Such machines have a high degree of parallelism. Fastest implementation of graph evolution is possible with new hardware in which there would be many simpler processors and each would implement the majority rule and locally store the state of vertices. Each processor would also be directly connected or wired to its neighborhood of processors. Because of their simplicity, regularity, and the option of hard-wired neighborhoods which are not too large, cellular automata are natural candidates for VLSI implementation. In locally connected cellular automata, circuit design reduces to the design of a single simple cell, and layout is uniform; the whole mask for a large cellular-automaton array can be generated by a step-and-repeat procedure; essentially no silicon area is wasted on long interconnection lines. Our neuropercolation network models neuronal populations and is suitable for VLSI implementation; see Fig. 13.

Neuromorphic considerations indicate that a small amount of non-local connections are required for robust operation. This kind of rewiring is very
limited in brains. Most of the synapses of the cortical neurons are limited to the immediate neighborhood of the neuron. A neuron has one long axon out of several thousands of essentially local synaptic connection. So the rewiring ratio in cortical neurons is below 0.1%. This level of non-locality is considered affordable by advanced Field Programmable Gate Array (FPGA) Cellular Automaton architectures (Chua, 2010). The mix of cellular automata with small amount of rewiring gives rise to a new generation of parallel and distributed machines. An alternative approach is to pursue analog platforms to benefit from the intrinsic noise in such systems. A potential neuromorphic embodiment includes Complex Atomic Switch Arrays (CASA) (Stieg et al., 2012; Avizienis et al., 2012) in very dense nanowire networks consisting $\text{Ag}_2S$ memristive compounds. Intensive studies are underway to develop a comprehensive approach to neuromorphic hardware for neuropercolation models based on CASA platform.

7. Conclusions

In the mathematical description of complex objects such as brains, cellular automata have clear advantages compared to differential equations. From a pragmatic point of view, the use of cellular automata seems logical, as most differential equations that arise in practice cannot be solved in analytical terms, rather they are evaluated using finite difference schemes. These numerical solutions are in fact discrete algorithms operating on cellular automata. Thus, cellular automata can be viewed as approximations to differential equations. There are, however, deeper reasons requiring the reformulation of the mathematics of the brains, following the visionary insight of Von Neumann and his pioneering work on cellular automata (Neumann, 1958; Neumann and Burks, 1966).

As long as the differential equations have closed-form solutions, it is clearly justified to use them to understand the underlying phenomena. Once we give up analytical solution as a major motive for using differential equations, one starts wondering whether one should still keep them as the starting point for numerical modeling (Toffoli, 1984). If we need to discretize differential equations to evaluate their solutions, it is questionable why to make assumptions on the differentiability and smoothness of the underlying physical and biological phenomena, especially when experimental evidence points to essential discontinuities in the behavior of the system. This is exactly the case with brain dynamics, when evidence is accumulating that sudden jumps
in the form of phase transitions are key effects describing higher cognitive activity and conscious experience.

In this work we introduced a hierarchy of random cellular automata as the mathematical tool to describe the spatio-temporal dynamics of the cortex. The corresponding brain model is called neuropercolation which has distinct advantages compared to traditional models using differential equations, especially in describing spatio-temporal discontinuities in the form of phase transitions. It is shown in this work that criticality in the cortex is characterized by a critical region instead of a singular critical point, and the trajectory of the brain as a dynamical systems crosses the critical region from less organized phase to more organized phase during input induced destabilization and vice versa (Kozma et al., 2012b; Freeman et al., 2012). We provide mathematical and computational analysis and results to support system-level brain-like model in computers. With the emergence of new paradigms, such as memristors (Kozma et al., 2012a), our proposed approach can find very efficient hardware implementation platform. Research is in progress on neuropercolation implementation on massively parallel digital computers and alternatively using analog computation platforms.

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