

Self-organized criticality model for brain plasticity

Lucilla de Arcangelis,¹ Carla Perrone-Capano² and Hans J. Herrmann³

¹ *Department of Information Engineering and INFM-Coherentia,
Second University of Naples, 81031 Aversa (CE), Italy*

² *University of Catanzaro "Magna Graecia",
Dept. of Pharmacobiology,*

*I-88021 Roccelletta di Borgia (CZ),
and IGB "A. Buzzati Traverso", CNR,
Via P. Castellino 111, I-80131 Napoli, Italy*

³ *Institute for Computer Applications 1,
University of Stuttgart, Pfaffenwaldring 27,
D-70569 Stuttgart, Germany*

Networks of living neurons exhibit an avalanche mode of activity, experimentally found in organotypic cultures. Here we present a model for brain plasticity based on self-organized criticality. The model consists in an electrical network with threshold firing and activity-dependent synapse strengths. The analysis of the power spectra of the electrical signal reproduces very robustly the power law behaviour with the exponent 0.8, experimentally measured in medical electroencephalograms spectra. The same value of the scaling exponent is found on small-world lattices and for leaky neurons, indicating that universality holds for a wide class of brain models.

PACS numbers: 05.65.+b, 05.45.Tp, 89.75.-k, 87.19.La

Cortical networks exhibit diverse patterns of activity, including oscillations, synchrony and waves. During a neuronal process, each neuron can receive inputs by thousands of other neurons and, when it reaches a threshold, redistributes this integrated activity back to the neuronal network. Recently it has been shown that another mode of activity is neuronal avalanches, with a dynamics similar to self-organized criticality (SOC) [1, 2], observed in organotypic cultures from coronal slices of rat cortex [3] where neuronal avalanches are stable for many hours [4]. The term SOC usually refers to a mechanism of slow energy accumulation and fast energy redistribution driving the system toward a critical state, where the distribution of avalanche sizes is a power law obtained without fine tuning: no tunable parameter is present in the model. The simplicity of the mechanism at the basis of SOC has suggested that many physical and biological phenomena characterized by power laws in the size distribution, represent natural realizations of the SOC idea. For instance, SOC has been proposed to model earthquakes [5, 6], the evolution of biological systems [7], solar flare occurrence [8], fluctuations in confined plasma [9] snow avalanches [10] and rain fall [11].

Here we apply the SOC idea to model a neural network able to exhibit plasticity. Plasticity is one of the most astonishing properties of the brain, occurring mostly during development and learning [12, 13], and can be defined as the ability to modify the structural and functional properties of synapses. In the mammalian central nervous system the refinement of neuronal connections is thought to occur during "critical periods" of early postnatal life [14], when circuits are particularly susceptible to electrical activity triggered by external sensory inputs [15].

Modifications in the strength of synapses are thought to underly memory and learning. Many cellular mechanisms of synaptic plasticity have been investigated. Among them activity dependent hebbian plasticity constitutes the most fully developed and influential model of how information is stored in neural circuits [16, 17].

In order to monitor neural activities, different time series are usually analysed through power spectra and generally power-law decay is observed. This behaviour remains unexplained. Understanding its origin is not only a major theoretical challenge but also of eminent importance in many applications, in particular to give a solid basis to the interpretation of electroencephalograms (EEG) [18, 19]. A large number of time series analyses have been performed on medical data that are directly or indirectly related to brain activity. Prominent examples are EEG data which are used by neurologists to discern sleep phases, diagnose epilepsy and other seizure disorders as well as brain damage and disease [18]. An other example of a physiological function which can be monitored by time series analysis is the human gait which is controlled by the brain [20]. For all these time series the power spectrum, i.e. the square of the amplitude of the Fourier transformation double logarithmically plotted against frequency, generally features a power law at least over one or two orders of magnitude with exponents between 1 and 0.7. On top of this background power law, additional structures give information on the details of the pathology and can point to specific resonance, frequency cut-offs and other deviations. While much focus is given to these secondary structures, the basic power law remains largely unexplained.

A large variety of models for brain activity has been

proposed, based for instance on the convolution of oscillators [21] or stochastic waiting times [22]. They are essentially abstract representations on a mesoscopic scale, but none of them is based on the behaviour of a neural network itself. In order to get real insights on the relation between these time series and the microscopic, i.e. cellular, interactions inside a neural network, it is necessary to identify the essential ingredients of the brain activity responsible for characteristic scale-free behaviour observed through the power law of the spectrum, as discussed above. This insight is the basis for any further understanding of the diverse additional features that are observed and interpreted by practitioners that analyse these time series for diagnosis. Therefore the formulation of a brain model that yields the correct power spectrum is of crucial importance for any further progress in the understanding of the living brain.

We present a new model that introduces within a SOC approach the three most important ingredients for neuronal activity, namely threshold firing, neuron refractory period and activity-dependent synaptic plasticity. With this model we are able to monitor the time signal for electrical activity and compare the power spectra with EEG data. We consider a simple square lattice of size $L \times L$ on which each site represents the cell body of a neuron, each bond a synapse. Therefore, on each site we have a potential v_i and on each bond a conductance g_{ij} . Whenever at time t the value of the potential at a site i is above a certain threshold $v_i \geq v_{\max}$, approximately equal to $-55mV$ for the real brain, the neuron fires, i.e. generates an "action potential", distributing charges to its connected neighbours in proportion to the current flowing through each bond

$$v_j(t+1) = v_j(t) + v_i(t) \frac{i_{ij}(t)}{\sum_k i_{ik}(t)} \quad (1)$$

where $v_j(t)$ is the potential at time t of site j , nearest neighbor of site i , $i_{ij} = g_{ij}(v_i - v_j)$ and the sum is extended to all nearest neighbors k of site i that are at a potential $v_k < v_i$. The conductances are initially all set equal to unity whereas the neuron potentials are uniformly distributed random numbers between $v_{\max} - 2$ and $v_{\max} - 1$. The potential is fixed to zero at top and bottom whereas periodic boundaries are imposed in the other direction.

The external stimulus is imposed at one input site in the centre of the lattice, and the electrical activity is monitored as function of time by measuring the total current flowing in the system. The firing rate of real neurons is limited by the refractory period, i.e. the brief period after the generation of an action potential during which a second action potential is difficult or impossible to elicit. The practical implication of refractory periods is that the action potential does not propagate back toward the initiation point and therefore is not allowed to reverberate between the cell body and the synapse. In

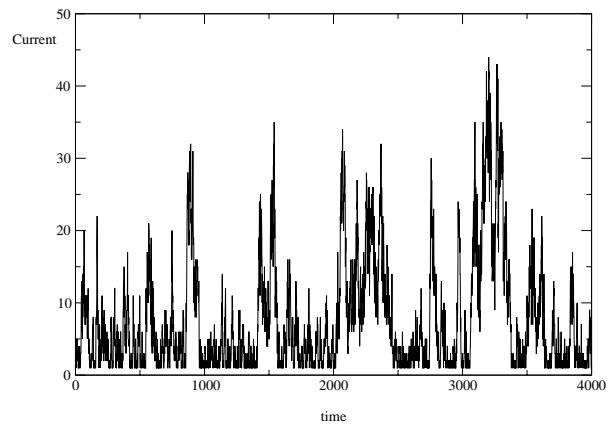


FIG. 1: Total current flowing in one lattice configuration ($L = 1000$, $\alpha = 0.03$, $\sigma_t = 0.0001$, $v_{\max} = 6$) as function of time in a sequence of several thousand stimuli.

our model, once a neuron fires, it remains quiescent for one time step and it is therefore unable to accept charge from firing neighbours. This ingredient indeed turns out to be crucial for a controlled functioning of our numerical model. In this way an avalanche of charges can propagate far from the input through the system.

As soon as a site is at or above threshold v_{\max} at a given time t , it fires according to Eq. (1), then the conductance of all the bonds that have carried a current is increased in the following way

$$g_{ij}(t+1) = g_{ij}(t) + \delta g_{ij}(t) \quad (2)$$

where $\delta g_{ij}(t) = k\alpha i_{ij}(t)$, with α being a dimensionless parameter and k a unit constant bearing the dimension of an inverse potential. After applying Eq. (2) the time variable of our simulation is increased by one unit. Eq. (2) describes the mechanism of increase of synaptic strength, tuned by the parameter α . Once an avalanche of firings comes to an end, the conductance of all the bonds with non-zero conductance is reduced by the average conductance increase per bond, $\Delta g = \sum_{i,j,t} \delta g_{ij}(t) / N_b$, where N_b is the number of bonds with non-zero conductance. The quantity Δg depends on α and on the response of the brain to a given stimulus. In this way our electrical network "memorizes" the most used paths of discharge by increasing their conductance, whereas the less used synapses atrophy. Once the conductance of a bond is below an assigned small value σ_t , we remove it, i.e. set it equal to zero, which corresponds to what is known as pruning. This remodelling of synapses mimicks the fine tuning of wiring that occurs during "critical periods" in the developing brain, when neuronal activity can modify the synaptic circuitry, once the basic patterns of brain wiring are established [12, 13]. These mechanisms simulate a hebbian form of activity dependent plasticity, where correlated presynaptic and postsynaptic activity modulate the efficiency of

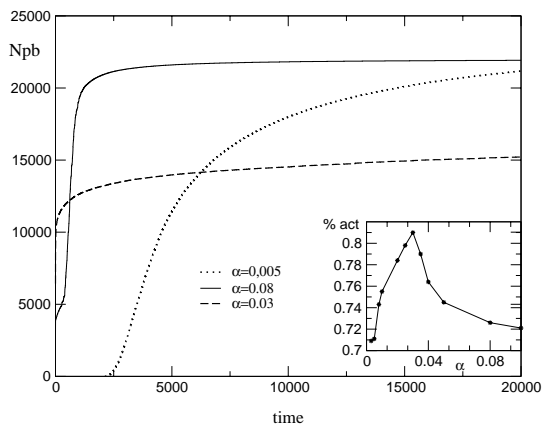


FIG. 2: The average number of pruned bonds N_{pb} as function of time in a lattice of linear size $L = 100$ for different values of α . The value of the parameters is $\sigma_t = 0.0001$ and $v_{\max} = 6$. In the inset we show the asymptotic value of the percentage of active bonds as function of α .

the synapse [16, 17]. However, differently from the well known Long Term Potentiation (LTP) and Long Term Depression (LTD) mechanisms, in our model the modulation of synaptic strength does not depend on the frequency of synapse activation [12, 23, 24]. It should be also considered that in the living brain synapses subject to plasticity are not electrical but chemical. For instance, hebbian plasticity at excitatory synapses is classically mediated by postsynaptic calcium dependent mechanisms [25]. In our approach the excitability of the post-synaptic neuron is simply modulated by the value of the electrical potential.

The external driving mechanism to the system is imposed by setting the potential of the input site to the value v_{\max} , corresponding to one stimulus. We let the discharge evolve until no further firing occurs, then we apply the next stimulus. Fig.1 shows the electrical signal as function of time: the total current flowing in the system is recorded in time during a sequence of successive avalanches. As defined above the time unit corresponds to the time necessary to propagate the signal from a neuron to next nearest neighbours. Data show that discharges of all sizes are present in the brain response, as in self-organized criticality where the avalanche size distribution scales as a power law [3].

The strength of the parameter α , controlling both the increase and decrease of synaptic strength, determines the pruning dynamics in the network. In fact, the more the system learns strengthening the used synapses, the more the unused connections will weaken. Fig. 2 shows the number of pruned bonds in the system as function of time for different values of α : For large values (e.g. $\alpha = 0.08$) the system strengthens more intensively the synapses carrying current but also very rapidly prunes the less used connections, reaching after a short transient a plateau where it prunes very few bonds. On the

contrary, for small values of α (equal to 0.005) the system takes more time to initiate the pruning process and slowly reaches a plateau. The inset of Fig.2 shows the asymptotic value of the fraction of active bonds, calculated as the total number of bonds in the unpruned network minus the asymptotic number of pruned bonds, as function of α . The number of active (non-pruned) bonds asymptotically reaches its largest value at the value $\alpha = 0.03$. This could be interpreted as an optimal value for the system with respect to plastic adaptation.

Since each avalanche may trigger the activity of a high number of neurons, large currents flow through the system, therefore after N_p stimuli the network is no longer a simple square lattice due to pruning. This complex structure constitutes the first approximation to a trained brain, on which measurements are performed. These consist of a new sequence of stimuli at the input site, by setting the voltage at threshold, during which we measure the number of firing neurons as function of time. This quantity corresponds to the total current flowing in a discharge measured by the electromagnetic signal of the EEG. In order to compare with medical data, we calculate the power spectrum of the resulting time series, i.e. the square of the amplitude of the Fourier transform as function of frequency.

The average power spectrum as function of frequency is shown in a log-log-plot with the parameters $\alpha = 0.03$, $N_p = 10$, $\sigma_t = 0.0001$, $v_{\max} = 6$ and a lattice of size $L = 1000$ (Fig. 3). We see that it exhibits a power law behaviour with the exponent 0.8 ± 0.1 . This is precisely the same value for the exponent found generically on medical EEG power spectra [26, 27]. We also show in Fig. 3 the magnetoencephalography (similar to EEG) obtained from channel 17 in the left hemisphere of a male subject, as measured in ref.[27], having the exponent 0.795.

We have checked that the value of the exponent is stable against changes of the parameters α , v_{\max} , σ_t , and N_p , and it is valid also for random initial bond conductances. For $\alpha = 0$ the frequency range of validity of the power law decreases by more than an order of magnitude. We also simulate the brain dynamics on a square lattice with a small fraction of bonds, from 0 to 10%, rewired to long range connections corresponding to a small world network [28–30], which more realistically reproduces the connections in the real brain. Fig.3 shows the power spectrum for a system with 1% rewired bonds and a different set of parameters α , N_p , v_{\max} : the spectrum has some deviations from the power law at small frequencies and tends to the same universal scaling behaviour at larger frequencies over two orders of magnitude. The same behaviour is found for a larger fraction of rewired bonds.

For real systems neurons have a leakage, namely the potential decays exponentially in time with a relaxation time τ , i.e. $\frac{dv(t)}{dt} = -\gamma v(t)$, with $\gamma = 1/\tau$. Leakage has been considered in our model and the same scal-

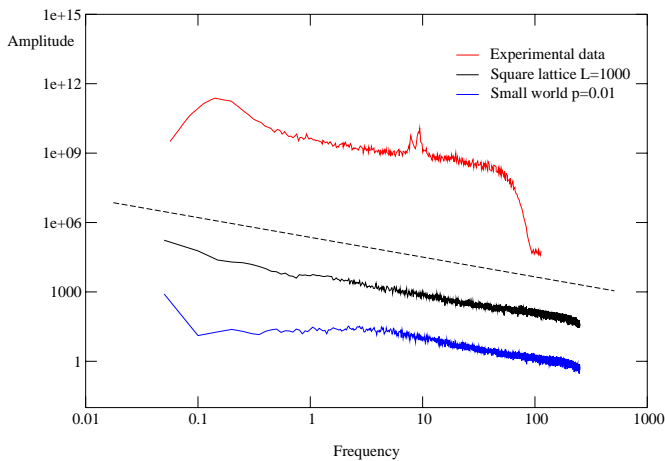


FIG. 3: Power spectra for experimental data and numerical data ($L = 1000$, $\alpha = 0.03$, $N_p = 10$, $v_{\max} = 6$) for the square lattice (middle curve) and the small world lattice (bottom curve, $L = 1000$, $\alpha = 0.05$, $N_p = 1000$, $v_{\max} = 8$) with 1% rewired bonds. The experimental data (top curve) are from ref.[19] and frequency is in Hz . The numerical data are averaged over 10000 stimuli in 10 different network configurations. The dashed line has a slope 0.8.

ing behaviour recovered (Fig.4). However for $\tau \leq 10$ (i.e. for stronger leaking), the low frequency part of the spectrum appears to be frequency independent and the scaling regime is recovered at high frequencies with an exponent in agreement with previous results.

The stability of the exponent suggests that an universal scaling characterizes a large class of brain models and physiological signal spectra for brain controlled activities. Medical studies of EEG focus on subtle details of a power spectrum (e.g. shift in peaks) to discern between various pathologies. These detailed structures however live on a background power law spectrum that shows universally an exponent of about 0.8, as measured for instance in refs. [26] and [27]. A similar exponent was also detected in the spectral analysis of the stride-to-stride fluctuations in the normal human gait which can directly be related to neurological activity [20]. Our simple model includes some fundamental features of brain activity and opens new perspectives to study pathological features of EEG spectra by including further realistic details into the neuron and synapsis behaviour.

Acknowledgements. We gratefully thank E. Novikov and collaborators for allowing us to use their experimental data. We also thank Salvatore Striano, MD, for discussions and Stefan Nielsen and Hansjörg Seybold for help. L.d.A. research is supported by EU Network MRTN-CT-2003-504712, MIUR-PRIN 2004, MIUR-FIRB 2001, CRdC-AMRA, INFM-PCI.

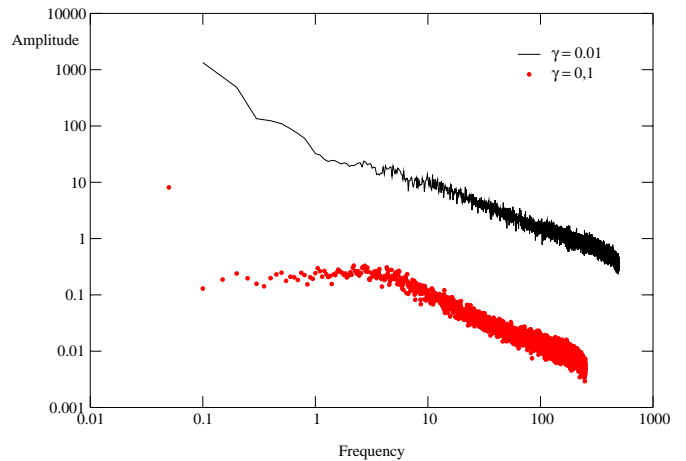


FIG. 4: Power spectra for numerical data (square lattice $L = 1000$, $\alpha = 0.03$, $N_p = 10$, $v_{\max} = 6$) for the case of leaky neurons with two different values of γ . Data are averaged over 10000 stimuli in 10 different network configurations.

-
- [1] P. Bak, *How nature works. The science of self-organized criticality* (Springer, New York, 1996).
 - [2] S. Maslov, M. Paczuski, P. Bak, *Phys. Rev. Lett.* **73**, 2162 (1994).
 - [3] J. M. Beggs, D. Plenz, *J. Neurosci.* **23**, 11167 (2003).
 - [4] J. M. Beggs, D. Plenz, *J. Neurosci.* **24**, 5216 (2004).
 - [5] P. Bak, C. Tang, *J. Geophys. Res.* **94**, 15635 (1989).
 - [6] A. Sornette, D. Sornette, *Europhys. Lett.* **9**, 197 (1989).
 - [7] P. Bak, K. Sneppen, *Phys. Rev. Lett.* **71**, 4083 (1993);
 - [8] E. T. Lu, R. J. Hamilton, *Astrophys. J.* **380**, L89 (1991)
 - [9] P. A. Politzer, *Phys. Rev. Lett.* **84**, 1192 (2000).
 - [10] J. Faillettaz, F. Louchet, J. R. Grasso, *Phys. Rev. Lett.* **93**, 208001 (2004).
 - [11] O. Peters, C. Hertlein and K. Christensen, *Phys. Rev. Lett.* **88**, 018701 (2002)
 - [12] T. D. Albraight et al, *Neuron*, Review supplement to vol. **59** (February 2000).
 - [13] L. C. Katz, C. J. Shatz, *Science* **274**, 1133 (1996).
 - [14] T. K. Hensch, *Ann. Rev. Neurosci.* **27**, 549 (2004).
 - [15] L. C. Katz, J. C. Crowley, *Nat. Rev. Neurosci.* **3**, 34 (2002).
 - [16] N. S. Desai, *J. Physiol. Paris* **97**, 391 (2003).
 - [17] S. J. Cooper, *Neurosci. Biobehav. Rev.* **28**, 851 (2005).
 - [18] A. Gevins et al, *Trends Neurosci.* **18**, 429 (1995).
 - [19] G. Buzsaki, A. Draguhn, *Science* **304**, 1926 (2004).
 - [20] J. M. Hausdorff et al., *Physica A.* **302**, 138 (2001).
 - [21] Y. Ashkenazy et al, *Physica A.* **316**, 662 (2002).
 - [22] P. Ch. Ivanov et al, *Europhys. Letters* **43**, 363 (1998).
 - [23] O. Paulsen, T. J. Sejnowski, *Curr. Opin. Neurobiol.* **10**, 172 (2000).
 - [24] K. H. Braunewell, D. Manahan-Vaughan, *Rev. Neurosci.* **12**, 121 (2001).
 - [25] G. Q. Bi, M. M. Poo, *Annu. Rev. Neurosci.* **24**, 139 (2001).
 - [26] W. J. Freeman et al, *J. Neurosci. Meth.* **95**, 111 (2000).
 - [27] E. Novikov et al, *Physical Review E.* **56**, 3 (1997).
 - [28] D. J. Watts, S. H. Strogatz, *Nature* **393**, 440 (1998).
 - [29] L. F. Lago-Fernandez et al, *Phys. Rev. Lett.* **84**, 2758 (2000).

[30] O. Shefi et al, Physical Review E. **66**, 021905 (2002).