Neuronal avalanches and learning

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Abstract. Networks of living neurons represent one of the most fascinating systems of biology. If the physical and chemical mechanisms at the basis of the functioning of a single neuron are quite well understood, the collective behaviour of a system of many neurons is an extremely intriguing subject. Crucial ingredient of this complex behaviour is the plasticity property of the network, namely the capacity to adapt and evolve depending on the level of activity. This plastic ability is believed, nowadays, to be at the basis of learning and memory in real brains. Spontaneous neuronal activity has recently shown features in common to other complex systems. Experimental data have, in fact, shown that electrical information propagates in a cortex slice via an avalanche mode. These avalanches are characterized by a power law distribution for the size and duration, features found in other problems in the context of the physics of complex systems and successful models have been developed to describe their behaviour. In this contribution we discuss a statistical mechanical model for the complex activity in a neuronal network. The model implements the main physiological properties of living neurons and is able to reproduce recent experimental results. Then, we discuss the learning abilities of this neuronal network. Learning occurs via plastic adaptation of synaptic strengths by a non-uniform negative feedback mechanism. The system is able to learn all the tested rules, in particular the exclusive OR (XOR) and a random rule with three inputs. The learning dynamics exhibits universal features as function of the strength of plastic adaptation. Any rule could be learned provided that the plastic adaptation is sufficiently slow.

1. Introduction
Cells in the nervous system can be divided into two categories: neurons and a variety of supporting cells, as neuroglial cells. Neurons are specialized for electrical signaling over long distances, whereas supporting cells are not capable of electrical signaling, despite having an important role in signal transmission. The human brain is estimated to contain 100 billion neurons and several times as many supporting cells. The interconnection of nerve cells via synapses forms an intricate network, which is the foundation on which sensory processes, perception and behaviour are built. The activity of neuronal networks consists in one or more action potentials in a single neuron or an ensemble of neurons. The first case is typical of small networks, as some experimental systems in vitro, where isolated spikes can be observed. The presence of a number of action potentials in an ensemble of neurons not always is a consequence of an external stimulus. Neuronal systems exhibit an intense spontaneous activity, known since a long time, whose relation with the response to stimulation is not fully understood yet. It is however well established that spontaneous activity cannot be simply reduced to a background noise uncorrelated to the system response. Indeed, experimental results for the cat visual cortex [1] have shown that the intensity of the response to an external stimulus is roughly proportional

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to the intensity of the spontaneous activity state of the system when the stimulus is applied. The variability in the response provided to the repeated application of the same stimulus is therefore caused by the different levels of ongoing activity. A similar analysis has been performed at the intracellular level on the same system, confirming that the spatio-temporal structure of the spontaneous activity influences the response signal [2].

The typical form of spontaneous activity consists in the almost synchronous emission of action potentials in a large number of neurons, followed by periods of substantial inactivity. These high activity events, named bursts, are observed both during development and in mature systems and can last from a few to several hundreds milliseconds. In 2003 Beggs and Plenz have identified a novel form of spontaneous activity, neuronal avalanches [3, 4]. Coronal slices of rat somatosensory cortex were placed onto a 8x8 multi-electrode array (MEA) and spontaneous activity was induced by bath perfusion with the glutamate receptor agonist NMDA in combination with a dopamine receptor agonist. The intrinsic activity of the system was monitored by measuring the potential at each electrode. This local field potential (LFP) integrates the electrical activity of neurons placed in the region surrounding the electrode: negative peaks in the LFP measure the influx of positive ions and therefore the cumulative membrane potential variation of the neurons in the region. Experimental data show that before 6 days in vitro activity is mainly composed of sparse activations but during the second week simultaneous activations occur in several electrodes. The novel idea was to examine this electrophysiological signal at a finer temporal scale, which was able to evidence a complex spatio-temporal structure. Indeed, activity starting at one electrode may involve more, not necessarily neighbour, electrodes. Binning time in cells of duration \( \delta t \), allows to create a spatio-temporal grid reporting the active electrodes in each temporal cell. A neuronal avalanche is therefore defined as a sequence of successively active electrodes between two temporal bins with no activity. The total number of active electrodes, or alternatively the sum of all LFPs, is defined as the size \( s \) of an avalanche and the time interval with ongoing activity as its duration \( T \).

The striking result is that both size and duration have no characteristic value, i.e. their distributions exhibit a power law behaviour. The analysis at a finer temporal scale is then able to enlighten the non-synchronous character of the bursts. The exponents of these power law distributions depend on the choice for the temporal bin \( \delta t \). Indeed larger bins make active electrodes belonging to different avalanches to merge into the same larger event, leading to a smaller exponent. In order to identify the appropriate value of \( \delta t \), Beggs and Plenz verified that if this is equal to the average value of the time delay between two successive LFPs at one electrode for each culture, the exponent does not depend any longer on the specific culture. They were then able to identify the universal scaling behaviour

\[
\begin{align*}
P(s) &\propto s^{-\alpha} \quad \text{with} \quad \alpha = 1.5 \pm 0.1 \\
P(T) &\propto T^{-\beta} \quad \text{with} \quad \beta = 2.0 \pm 0.1.
\end{align*}
\]

The power law behaviour for the size distribution is followed by an exponential cutoff due to the finite size of the system, whereas for the duration distribution it extends for about one decade and the exponential cutoff sets in at about 10ms.

These results \textit{in vitro} have been confirmed by extended studies \textit{in vivo} on anaesthetized rats during development [5] and awake rhesus monkeys [6]. Synchronous activity in the bursts exhibits the same scaling behaviour found for neuronal avalanches \textit{in vitro} (Eq.1). The investigation on the spontaneous activity has been performed also for dissociated neurons from different networks as rat hippocampal neurons [7], rat embryos [8] or leech ganglia [7]. Under fixed conditions of humidity and temperature, neurons start to develop a network of synaptic connections and, after a variable period \textit{in vitro}, exhibit spontaneous electrical activity. Different behaviours are observed. Only those system exhibiting a medium level of synchronization between random spikes and synchronized bursts exhibit critical behaviour. For those cultures
the scaling behaviour is very robust and in agreement with Eq.1. In particular, the emergence of a critical state has been found to be strongly related to the ageing of the system, namely after the first few weeks in vitro, where the behaviour of the system is subcritical, some cultures may self-organize and reach the critical state as they mature [8].

2. The model
In order to formulate a new model to study neuronal activity, we have introduced [9, 10] within a SOC approach the three most important ingredients, namely threshold firing, neuron refractory period and activity-dependent synaptic plasticity. We consider a lattice of \( N \) sites on which each site represents the cell body of a neuron, each bond a synapse. Each neuron is characterized by the potential \( v_i \). Connections among neurons are established by assigning to each neuron \( i \) a random out-going connectivity degree, \( k_{\text{out},i} \). The distribution of the number of out-connections is then chosen in agreement with the experimentally measured properties of the functionality network [11] in human adults. Functional magnetic resonance imaging has indeed shown that this network has universal scale free properties, namely it exhibits a scaling behaviour \( n(k_{\text{out}}) \propto k_{\text{out}}^{-2} \), independent of the different tasks performed by the patient. We adopt this distribution for the number of pre-synaptic terminals of each neuron, over the range of possible values between 2 and 100, as in experimental data. Two neurons are then connected according to a distance dependent probability, \( p(r) \propto e^{-r/r_0} \), where \( r \) is their spatial distance [12] and \( r_0 \) a typical edge length. To each synaptic connection we then assign an initial random strength \( g_{ij} \), where \( g_{ij} \neq g_{ji} \) and an excitatory or inhibitory character, with a fraction \( p_m \) of inhibitory synapses. Whenever at time \( t \) the value of the potential at a site \( i \) is above a certain threshold \( v_i \geq v_{\text{max}} \), approximately equal to \(-55\text{mV}\) for the real brain, the neuron fires, i.e. generates an "action potential, distributing charge to its connected neighbours. The total charge released by a neuron is proportional to the number of synaptic connections, \( q_i \propto v_i k_{\text{out},i} \). Each connected neuron receives charge in proportion to the strength of the synapse \( g_{ij} \)

\[
v_j(t+1) = v_j(t) \pm \frac{q_i(t) \sum_k g_{ik}(t)}{k_{\text{in},j}}
\]

where \( k_{\text{in},j} \) is the in-degree of neuron \( j \) and the sum is extended to all out-going connections of \( i \). In Eq.2 it is assumed that the received charge is distributed over the surface of the soma of the post-synaptic neuron, proportional to the number of in-going terminals \( k_{\text{in},j} \). The plus or minus sign in Eq.2 is for excitatory or inhibitory synapses, respectively. After firing a neuron is set to a zero resting potential and in a refractory state lasting \( t_{\text{ref}} = 1 \) time step, during which it is unable to receive or transmit any charge. We wish to stress that the unit time step in Eq.2 does not correspond to a real time scale, it is simply the time unit for charge propagation from one neuron to the connected ones. In real system this time could vary and be as large as 100 ms for longer firing periods. The practical implication of the refractory period 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. The synaptic strengths have initially equal value, whereas the neuron potentials are uniformly distributed random numbers between \( v_{\text{max}} - 1 \) and \( v_{\text{max}} \). Moreover, a small random fraction (10\%) of neurons is chosen to be boundary sites, with a potential fixed to zero, playing the role of sinks for the charge. An external stimulus is imposed at a random site and, if the potential reaches the firing threshold, the neuron fires and an avalanche of firing neurons can propagate through the system.

2.1. Plastic adaptation
As soon as a site is at or above threshold \( v_{\text{max}} \) at a given time \( t \), it fires according to Eq.2, then the conductance of all the bonds, connecting to active neurons and that have carried a current,
Figure 1. One neuronal avalanche involving 15 neurons on the scale-free network with $N = 40$ neurons connected by directed bonds (direction indicated by the arrow at one edge), representing the synapses. The size of each neuron is proportional to the number of in-connections, namely the number of dendrites. The two red neurons are the two input sites, whereas the black neuron is the output. Connections involved in the avalanche propagation are shown in red, whereas inactive connections are black.

is increased in the following way

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

(3)

where $\delta g_{ij}(t) = k\alpha i_{ij}(t)$, with $\alpha$ being a dimensionless parameter and $k$ a unit constant bearing the dimension of an inverse potential. After applying Eq.3 the time variable of our simulation is increased by one unit. Eq.3 describes the mechanism of increase of synaptic strength, tuned by the parameter $\alpha$. This parameter then represents the ensemble of all possible physiological factors influencing synaptic plasticity. 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

$$\Delta g = \sum_{ij,t} \delta g_{ij}(t)/N_b$$

(4)

where $N_b$ is the number of bonds with non-zero conductance. The quantity $\Delta g$ depends on $\alpha$ 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 $\sigma_t$, we remove it, i.e. set it equal to zero, which corresponds to what is known as pruning. These mechanisms correspond to a Hebbian form of activity dependent plasticity, where the conjunction of activity at the presynaptic and postsynaptic neuron modulates the efficiency of the synapse [13]. To insure the stable functioning of neural circuits, both strengthening and weakening rule of Hebbian synapses are necessary to avoid instabilities due to positive feedback [14]. 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 [15, 16, 17]. It should be also considered that in the living brain
synapses exhibiting plasticity are not electrical but chemical. For instance, Hebbian plasticity at excitatory synapses is classically mediated by postsynaptic calcium dependent mechanisms [18]. In our approach the excitability of the postsynaptic neuron is simply modulated by the value of the electrical potential.

3. Neuronal avalanches in spontaneous activity

The plasticity procedure is applied for a series of \( N_p \) stimuli to adapt the strengths of synapses. In fact, the more the system is active strengthening the used synapses, the more the unused connections will weaken. The plastic adaptation proceeds until few bonds are pruned. The system at this stage constitutes the first approximation to a trained brain, on which measurements are performed. These consist of a new sequence of stimuli, by adding a small quantity to the voltage of a random neuron. We monitor the avalanche activity’s function of time.

We measure the size distribution of neuronal avalanches. The size is either the total number of firing neurons, or the to sum of their voltage variations. This distribution exhibits a power law behaviour, with an exponent equal to \( 1.5 \pm 0.1 \), quite stable with respect to parameters. This scaling behaviour is also robust for densities of inhibitory synapses up to 10%, whereas the scaling behaviour is lost for higher densities. Moreover, the distribution of avalanche temporal durations is also a power law with an exponent close to \(-2.0\). Both these values show an excellent agreement with experimental data. Moreover, the temporal signal for electrical activity and the power spectrum of the resulting time series have been compared with EEG data [9, 10]. The spectrum exhibits a power law behaviour, \( P(f) \sim f^{-0.8} \), with an exponent in good agreement with EEG medical data [19] and physiological signal spectra for other brain controlled activities [20]. This model therefore seems to capture many of the essential ingredients of spontaneous activity, as measured in cortical networks (Eq.1).

4. Learning

Next we study the learning performance of this neuronal network acting in a critical state [21]. In order to start activity we identify input neurons at which the rule is applied and the output
neuron at which the response is monitored. These nodes are randomly placed inside the network under the condition that they are not boundary sites and they are mutually separated on the network by $k_d$ nodes. $k_d$ represents the chemical distance on the network and plays the role of the number of hidden layers in a perceptron. We test the ability of the network to learn different rules: AND, OR, XOR and a random rule RAN which associates to all possible combinations of binary states at three inputs a random binary output. More precisely, the AND, OR and XOR rules are made of three input-output relations, whereas the RAN rule with three input sites implies a sequence of seven input-output relations. A single learning step requires the application of the entire sequence of states at the input neurons, monitoring the state of the output neuron. For each rule the binary value 1 is identified with the output neuron firing, namely the neuron membrane potential at a value greater or equal to $v_{\text{max}}$ at some time during the activity. Conversely, the binary state 0 at the output neuron corresponds to the physiological state of a real neuron which has been depolarized by incoming ions but fails to reach the firing threshold membrane potential during the entire avalanche propagation. Once the input sites are stimulated, their activity may bring to threshold other neurons and therefore lead to avalanches of firings. We impose no restriction on the number of firing neurons in the propagation and let the avalanche evolve to its end according to Eq.2. If at the end of the avalanche the propagation of charge did not reach the output neuron, we consider that the state of the system was unable to respond to the given stimulus, and as a consequence to learn. We therefore increase uniformly the potential of all neurons by units of a small quantity, $\beta = 0.01$, until the configuration reaches a state where the output neuron is first perturbed. We then compare the state of the output neuron with the desired output.

Plastic adaptation is applied to the system according to a non-uniform negative feedback algorithm. Namely, if the output neuron is in the correct state according to the rule, we keep the value of synaptic strengths. Conversely, if the response is wrong we modify the strengths of those synapses involved in the information propagation by $\pm \alpha / d_k$, where $d_k$ is the chemical distance of the presynaptic neuron from the output neuron. The sign of the adjustment depends on the mistake made by the system: If the output neuron fails to be in a firing state we increase the used synapses by a small additive quantity proportional to $\alpha$. Synaptic strengths are instead decreased by if the expected output 0 is not fulfilled. This adaptation rule intends to mimic the feedback to the wrong answer triggered locally at the output site, for instance by some hormones, and propagating backward towards the input sites.

![Figure 3](image_url)

**Figure 3.** Percentage of configurations learning the XOR rule as function of the number of learning steps, for $\alpha = 0.005$, $k_d = 3$, the initial minimum connectivity degree equal to 3 and different numbers of neurons $N$ (from 250 to 1000 bottom to top).
We analyse the ability of the system to learn the different rules. Fig. 2 shows the fraction of configurations learning the AND rule versus the number of learning steps for different values of the plastic adaptation strength $\alpha$. We notice that the larger the value of $\alpha$ the sooner the system starts to learn the rule, however the final percentage of learning configurations is lower. The final rate of success increases as the strength of plastic adaptation decreases. This result is due to the highly non-linear dynamics of the model, where firing activity is an all or none event controlled by the threshold. The result that all rules give a higher percentage of success for weaker plastic adaptation, is in agreement with recent experimental findings on visual perceptual learning, where better performances are measured when minimal changes in the functional network occur as a result of learning [22].

We characterize the learning ability of a system for different rules by the average learning time, i.e. the average number of times a rule must be applied to obtain the right answer, and the asymptotic percentage of learning configurations. This is determined as the percentage of learning configurations at the end of the teaching routine, namely after $10^6$ applications of the rule. The average learning time scales as $\tau \propto 1/\alpha$ for all rules and independently of parameter values. The asymptotic percentage of success increases by decreasing $\alpha$ as a very slow power law, $\propto \alpha^{-0.042 \pm 0.01}$, where the exponent is the average value over different rules. We check this scaling behaviour by appropriately rescaling the axes in Fig.2. The curves corresponding to different $\alpha$ values indeed all collapse onto a unique scaling function. Similar collapse is observed for the OR, XOR and RAN rules and for different parameters $k_d$ and $p_{in}$. The learning dynamics shows therefore universal properties, independent of the details of the system or the specific task assigned.

Finally we explicitly analyse the dependence of the learning performance and its scaling behaviour on the number of neurons. Indeed, as the system size increases the number of highly connected neurons becomes larger. A well connected system provides better performances, therefore we could expect that the size dependence reflects the same effect. In Fig.3 we show data for a set of parameters and different system sizes. The learning performance indeed improves with the system size since, for the same out degree distribution, the overall level of connectivity improves for larger systems.

5. Conclusions

Extensive simulations have been presented for a novel brain model with activity dependent plasticity, implemented on scale free lattices. The ingredients of the model are close to most functional and topological properties of real neuronal networks. The avalanche size and duration distributions show a power law behaviour with exponents $1.5 \pm 0.1$ and $2.0 \pm 0.1$, respectively, compatible the values found experimentally for neuronal avalanches. Next, we investigate the learning ability of this model. The implemented learning dynamics is a cooperative mechanism where all neurons contribute to select the right answer and negative feedback is provided in a non-uniform way. Despite the complexity of the model and the high number of degrees of freedom involved at each step of the iteration, the system can learn successfully even complex rules. The neuronal network has a “universal” learning dynamics, even complex rules can be learned provided that the plastic adaptation is sufficiently slow.

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