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STRUCTURAL PHASE TRANSITIONS IN NEURAL NETWORKS

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Abstract. A model is considered for a neural network that is a stochastic process on a random graph. The neurons are represented by "integrate-and-fire" processes. The structure of the graph is determined by the probabilities of the connections, and it depends on the activity in the network. The dependence between the initial level of sparseness of the connections and the dynamics of activation in the network was investigated. A balanced regime was found between activity, i.e., the level of excitation in the network, and inhibition, that allows formation of synfire chains.

1. Introduction.

1.1. **Search for synfire chains.** The idea that neuronal networks may be organized as feed-forward structures composed of multiple layers in which the activity flows from the input naturally arises from neuronal recording of precisely-timed spiking activity in brain (Abeles 1982, Abeles 1991). Beginning with the work of Abeles (1991), synfire chains have been intensively studied as a model for neural information processing in the cortex. Synfire chains are capable of generating temporally extended and precisely timed patterns of spiking activity, and thus can explain precisely timed sequential activity patterns, as demonstrated by electrophysiological recordings. Spatiotemporal activity patterns were found, for example, in monkeys (Prut et al., 1998, Ayzenshtat et al., (2010)), rats (Villa et al., 1999), and birds (Hahnloser et al., 2002; Mooney and Prather, 2005). All these studies indicate that spatiotemporal activity patterns play a role in cortical mechanisms of information processing. It has also been suggested that strictly feed-forward synfire chains may encode the syllables that make up the song of birds (Fiete et al., 2010).

It is therefore natural to use spatiotemporal patterns to encode information. Hence, synfire chains provide a possible paradigm for cognitive representations. Assuming that the basic pool-to-pool structure of synaptic connections is available, one can associate the capacity of a network to encode and process information with a number of chains effectively embedded into a network (Trengove et. al. 2012). There are common problems to all encoding paradigms (see, e.g., Rolls and Treves 2011): how to balance the number of encoded images and the ability of the network to activate a number of chains simultaneously. It seems that the use of spatiotemporal patterns or synfire chains in encoding information presents a great challenge for theory and simulations.

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Perhaps an even greater challenge is to describe a biologically plausible mechanism for growing synfire chains. Searching for the conditions for the self-sustained spontaneous activity which can lead to a formation of long synfire chains has been continuing for a couple of decades (e.g., Hertz and Prügel-Bennett 1996, Bienenstock 1995, Waddington et al. 2011, and the references therein).

To demonstrate the growth of feedforward structures usually models based on spike-timing dependent plasticity (STDP) or other plasticity mechanisms are considered. The initial assumptions are commonly accepted: a network consists of a large number (about 10,000) neurons, and each of every possible connection between the neurons is independently assigned a weight (or a probability of activation). In other words, transmission of signals is possible from each to any other of the neurons in the network.

In this context the neurons are typically modelled as integrate-and-fire devices. Then an external stimulus activates a (small) part of the network, causing a random trajectory of the propagation of the impulses in the network. The STDP can be realized by increasing the causal connections between the neurons, and on the contrary, decreasing the weights of synapses which disagree with the direction of activation. It is noted in the context of this synaptic weight modification that there is some evidence that the synaptic weights may follow a discrete distribution (Montgomery and Madison (2004)).

However, it has been observed (e.g., Iglesias and Villa (2008), see also discussion in Waddington et al. (2011)) that STDP alone does not produce the desired architecture. Therefore besides biologically justified STDP the models assume rather drastic artificial constraints. Here are a few recent examples.

Fiete et al. (2010) use an assumption that the sum of all outgoing (incoming) synaptic weights at the neuron almost does not exceed a certain limit. However, this seemingly "soft" rule transforms in the course of analysis presented by Fiete et al. (2010) into a very strong competitive constraint on synapses, resulting in a chain of single neurons. This reduces the whole complexity of the initial problem to random permutations naturally associated with ordering of the neurons into one chain. (Arguably) Fiete et al. (2010) deduce from this oversimplified model bounds on the length of the synfire chains which are claimed to be in agreement with experiments on song-birds.

Waddington et al. (2011) take another approach, what they call topological constraints. Namely, to achieve stability in developing a desired structure, a total suppression of spontaneous activity is imposed on a neuron as soon as it is recruited to the chain (Waddington et al. (2011)).

Furthermore, Kunkel et al. (2011) came up with an explanation of why weight-dependent STDP in a cortical-scale balanced random network architecture fails to produce the growth of synfire chain structures. They argue that an unstable fixed point in the dynamics prevents the stable propagation of structure in recurrent networks with weight-dependent STDP. Kunkel et al. (2011) use a statistical physics approach which enables them to treat biologically reasonable parameters of a network: each neuron receives a biologically realistic number of inputs (6000) and the network exhibits a biologically realistic degree of sparseness (dilution) (connection probability of 0.06).

We found another explanation for the observed instability with the help of the theory of bootstrap percolation on random graphs (Turova (2012)). Interestingly, to some extent it also relies on the fact that a dynamical system associated with

the accumulation of activity in a recurrent network of integrate-and-fire units (of a simplest version) has only unstable fixed points (in the space of reasonable parameters). As Kunkel et al. (2011) observed also, it is the fan-out tendency that underlies the instability of the fixed point in their work.

1.2. **Methods of statistical physics for finite systems.** Use of percolation models in neuronal science was predicted already by Harris (1963). However, despite a growing number of studies of related models (Kozma *et. al.* (2005), Puljic and Kozma (2005), Eckmann *et. al.* (2010)), the recent theoretical achievements in bootstrap percolation have not yet been fully incorporated into analysis by neuroscientists.

We would like to point out one trivial but an essential problem with use of random graph theory, or for that matter, any theory which describes a system *in the limit*. In our case we want to make use of the theoretical results proved when the number of model neurons goes *to infinity*. To be able to test theoretical results on *finite* systems one has to have enough data, which may be difficult in neuroscience. Here is a typical example. The results on random graphs, and in particular on bootstrap percolation on random graphs, exhibit different scalings in the limit. This means that the statements of the results are given as functions (e.g., n^α) of the number n of vertices (number of neurons in our case). Thus it is typically assumed (Kunkel et al. (2011), Rolls (2008)) that in (fully weakly interconnected) cortex each of 100000 neurons with $n = 10000$ connections onto each neuron requires on average $C = 100$ synaptic connection active to fire a neuron. Then $C = 0.01n$ as well as $C = \sqrt{n}$ if $n = 10000$. However, when $n \to \infty$ it makes a huge difference which scaling is assumed.

However, one may believe that it is only a matter of time to gain sufficient experimental evidence to test the theory, the advantage of which is a complete description of all possible scalings.

We study here a stochastic model for a network of integrate-and-fire neurons. Turova and Villa (2007) suggested a model for the evolution of random graphs for investigating networks with embedded spike timing-dependent plasticity. We found in [24] that under special choice of parameters such a model can exhibit a sustained level of activity. Here we continue study of the model from [24], and more precisely a biologically justified modification of it.

Although the model is too crude to be called a neuronal network, it captures the essential feature of propagation of impulses in a more realistic model than is considered in almost all of the work cited above. A natural question could be: how do we control the instability around a fixed point observed in all previous simulations of such networks? We use an accurate description of the stochastic process on a random graph given by the theory of bootstrap percolation (Janson *et. al.* 2012) to choose a proper balance for the inhibition in the system. Observe that the global inhibition in our model acts on each (excitatory) neuron depending only on the potential of the neuron itself, and does not use any knowledge on the topology, i.e., on the position of a neuron in a current structure. We conclude therefore that even without drastic constraints a stable feed-forward structure may grow in a self-organized regime of criticality. Properly tuned inhibition forces the system to stay in the vicinity of a fixed though unstable point, i.e., criticality.

Observe, however, that there is no requirement for inhibitory input to impose stability. Other mechanisms, as e.g., refractory period, can also control the level of

excitation. In the latter case a perturbation by brief changes of input may return the system into a steady state (consult Freeman [7]).

We show here that the architecture of syn-fire chains, or, rather "braids", can be seen in our model as a temporary (but stable for macro-times, as to be explained below) self-organized structure. We provide scaling limits (in terms of the size of the network) for the topological parameters of the established braids: the length, the width, and the in- and out- degrees.

This approach offers a broad spectra of interpretations. However, this will be narrowed, as soon as we gain enough empirical evidence to make a proper choice of scaling for a finite network as we discussed above.

1.3. **Model.** Let us consider a set of *excitatory* neurons enumerated by vertices v of $\Lambda = \{1, \dots, n\}$. Assume that with a probability $p = p(n)$ there is a directed connection between any pair of excitatory neurons (vertices in Λ). We assume that $p(n) \to 0$ as $n \to \infty$. We shall write $u \sim v$ if there is a link from u to v.

The inhibitory neurons are modelled as *one inhibitory unit* connected to each of the excitatory ones. Such modelling of inhibition is rather common (e.g., Rolls (2008)). This does not change qualitatively the behaviour of the excitatory population, but it simplifies the analysis of a network.

Assume that each neuron v, $v \in \Lambda$, has a potential $X_v(t) \geq 0$ at time $t \in \{0, 1, \dots\}$. When $X_v(t) \geq 1$ we say that the neuron v *fires* or *is active* at time t.

Let us set up the initial conditions. Fix a subset $\mathcal{A}(0) \subset \Lambda$. Let $X_v(0) = 1$ for all $v \in \mathcal{A}(0)$, and $X_v(0) = 0$, $v \notin \mathcal{A}(0)$. Then $\mathcal{A}(0) \subset \Lambda$ is the set of active neurons at time 0.

For all $t \geq 0$ given $X_v(t), \quad v \in \Lambda$, define

$$\mathcal{A}(t) = \{v : X_v(t) \geq 1,\ X_v(t-1) < 1,\ v \in \Lambda\},$$

this is the set of neurons which became active at time t. The dynamics of activation is defined recurrently as follows. Given $X_v(t), \quad v \in \Lambda$, define

$$X_v(t+1) = \frac{X_v(t)e^{-\theta} + \sum_{u \in \mathcal{A}(t): u \sim v} \omega_{uv}(t)}{1 + \omega_- \Theta\left(\sum_{s \leq t} |\mathcal{A}(s)| - a_{thr}\right)}, \quad \text{if } X_v(t) < 1, \tag{1}$$

$$X_v(t+s) = 0, \quad s = 1, \dots, R, \quad \text{if } X_v(t) \geq 1, \tag{2}$$

where

$$\Theta(x) = \begin{cases} 0, & \text{if } x < 0, \\ 1, & \text{if } x \geq 0. \end{cases}$$

Here parameter $a_{thr} > 0$ is the threshold value for the total activity in the network to trigger the inhibitory unit, while parameter $\omega_- \geq 0$ is the strength of the inhibition. Parameter $0 \leq \theta \leq \infty$ is responsible for holding once achieved value of the potential till next incoming impulse. We assume that the synaptic connections $\omega_{uv}(t)$ are subjects to the synaptic facilitation, so that

$$\omega_{uv}(t) = \omega_u(t) = \omega + \omega_a \left(\sum_{s<t} e^{-\kappa(t-s)} \mathbf{1}\{X_u(s) \geq 1\} \right), \tag{3}$$

where $\omega > 0$, and parameters $\omega_a \geq 0$ and $\kappa > 0$ regulate the strength and the duration of the facilitation, correspondingly. Form (3) is similar to that of Mongillo et. al. (2008). Notice also that assumption (2) models a refractory period, i.e., if neuron fires then at the next R time units it cannot be active.

By the definition (1) conditionally on $\sum_{s\le t}|\mathcal{A}(s)|<a_{thr}$, neuron v fires for the first time at $t+1$ if $X_v(s)<1$, $s\le t$, and

$$X_v(t+1)=X_v(t)e^{-\theta}+\sum_{u\in\mathcal{A}(t):u\sim v}\omega_{uv}(t)=\sum_{s\le t}\sum_{u\in\mathcal{A}(s):u\sim v}\omega_{uv}(t)e^{-\theta(t-s)}\ge 1, \quad (4)$$

where by the definition (3)

$$\omega\le\omega_{uv}(t)\le\omega+\omega_a\sum_{s<t}e^{-\kappa(t-s)}=\omega+\omega_a\frac{1-e^{-\kappa t}}{\kappa}<\omega+\omega_a/\kappa. \quad (5)$$

Hence, as long as $\sum_{s\le t}|\mathcal{A}(s)|<a_{thr}$, we have the following bounds

$$\sum_{u\in\mathcal{A}(t):u\sim v}\omega\le\sum_{u\in\mathcal{A}(t):u\sim v}\omega_{uv}(t)\le X_v(t+1)\le\sum_{s\le t}\sum_{u\in\mathcal{A}(s):u\sim v}(\omega+\omega_a/\kappa). \quad (6)$$

We point out that in this model the memory of the transmitted impulses is stored by the synapses, as well as by the potentials $X_v(t)$, which accumulate the impulses coming at different moments of time.

Observe also that if $\theta=0$, $\omega_a=0$ and $\omega_-=0$, then system (1) describes the so-called bootstrap percolation process on a random graph $G_{n.p}$ (which is a graph on n vertices with random edges, each of which is present independently with probability p). This model was studied by Janson *et. al.* (2012) whose results we shall use here.

1.4. **Accumulation of activation level before triggering inhibition.** Consider the dynamics of activation in the network until the first time τ when

$$\sum_{s\le\tau}|\mathcal{A}(s)|\ge a_{thr}.$$

Let us first consider a simple case when both $\omega_a=0$ and $\theta=0$. This means that we do not assume a facilitation of synapses, but on the other hand we allow a potential of a neuron to accumulate impulses coming at different times. Then for all $t<\tau$ we derive from (5)

$$X_v(t+1)=\sum_{s\le t}\sum_{u\in\mathcal{A}(s):u\sim v}\omega=\omega\#\{u\sim v:u\in\cup_{s\le t}\mathcal{A}(s)\},\quad \text{if}\quad X_v(t)<1. \quad (7)$$

This is an intermediate case with respect to the bounds in (6), and hence analysis of this case should be instructive for understanding the behaviour for a more general set of parameters.

Let us consider first the set of neurons which were activated at least once. Denote

$$\mathcal{U}(t)=\cup_{s\le t}\mathcal{A}(s)$$

the set of neurons which were activated at least once up to time t, and let

$$A(t)=|\mathcal{U}(t)|.$$

As long as $A(t)<a_{thr}$ the inhibition does not play a role, and therefore the system follows the dynamics (7). In other words, a new neuron becomes active at time t if it has at least $[1/\omega]$ connections to the set $\mathcal{U}(t)$. This is a bootstrap percolation process analyzed by Janson *et. al.* (2012).

To study the dynamics of $A(t)$ let us introduce auxiliary independent identical Bernoulli random variables ξ_i, $i \geq 1$, which take value 1 with probability p. Then for all $t > 1$ we can compute according to (7)

$$\mathbf{E}\{A(t+1) \mid A(t)\} = (n - A(t))\mathbf{P}\left\{\sum_{i=1}^{A(t)} \xi_i \geq 1/\omega \mid \sum_{i=1}^{A(t-1)} \xi_i < 1\right\}. \tag{8}$$

Denote here

$$r := \left[\frac{1}{\omega}\right] + 1.$$

One can show (consult Janson *et. al.* (2012)) that as long as $A(t) \leq 3t_c$ ($t_c = o(n)$ as defined below) the dynamics of $A(t)$ follows closely

$$A(t+1) \approx S(A(t)),$$

where

$$S(t) = A(0) + n\frac{p^r}{r!}t^r. \tag{9}$$

Thus, a stable point of a system

$$t_{k+1} = S(t_k) \tag{10}$$

with initial state $t_0 = A(0)$ is critical for our original system as well.

Observe that if $r > 1$ then there exists a unique critical value

$$a_c = \left(1 - \frac{1}{r}\right)\left(\frac{(r-1)!}{np^r}\right)^{1/(1-r)}, \tag{11}$$

such that if $A(0) = a_c$ there is a unique solution t_c to (9), i.e., to

$$a_c + n\frac{p^r}{r!}t^r = t,$$

and hence the dynamical system (10) has a fixed point t_c.

We shall explore the following fact proved by Janson *et. al.* (2012). If the initial state is slightly above the critical value a_c then (depending on other parameters) the original system $A(t)$ has a slow dynamics of propagation in the vicinity of the critical point t_c. Let us state this result more precisely.

Fix arbitrarily $\frac{1}{r} < \alpha < 1$, $\frac{1}{2} < \beta < 1$, and set $p = \frac{1}{n^\alpha}$. Then by (11)

$$a_c + a_c^\beta \sim n^{\gamma_0},$$

where

$$\gamma_0 := \frac{\alpha r - 1}{r - 1} < 1.$$

Assume now that

$$A(0) = a_c + a_c^\beta.$$

Fix $K \geq 2$ arbitrarily and consider the first time when the activation from the initial $A(0)$ neurons spread to $(K-1)A(0)$ other neurons; denote this time

$$\tau_1 := \min\{t : A(t) \geq KA(0)\}.$$

Janson *et. al.* (2012) proved that for any constant $K \geq 2$

$$\tau_1 \sim C(K, r)n^\gamma, \tag{12}$$

where

$$\gamma = \frac{1-\beta}{2}\,\frac{r\alpha - 1}{r - 1} = \frac{1-\beta}{2}\gamma_0 < \gamma_0 < 1.$$

(For the details we refer to Janson *et. al.* (2012).) Asymptotics (12) tells us that a system needs of order n^γ steps to increase K times the number of firing neurons. Hence, the system exhibits *slow* dynamics while passing through the "bottleneck" in the vicinity of the critical value a_c. Notice, however, that after this phase the activation (without inhibition) very rapidly propagates through the network. More precisely, within a period of time of the same order n^γ almost all, i.e., $n - o(n)$ neurons will become active. (Again, see Janson *et. al.* (2012) for the details.)

1.5. **Tuning the inhibition.** We shall argue that properly tuned inhibition leads to a *self-organized criticality*. More precisely, inhibition will keep the system for a long time in the vicinity of an unstable point of the dynamics.

Let us set the threshold for the total activation in the inhibition term to be

$$a_{thr} = KA(0)$$

with $A(0)$ defined above. Then we choose the constants $K \geq 2$ and ω_- to satisfy

$$\sum_v \mathbf{P}\left\{\frac{X_v(\tau_1) + \sum_{u\sim v: u\in\mathcal{A}(\tau_1)}\omega}{1+\omega_-} \geq 1\right\} = \frac{1}{K}\mathbf{E}\mathcal{A}(\tau_1). \tag{13}$$

Notice, that for any $K > 1$ this equation has a solution $\omega_- > 0$. Indeed, the function on the left hand side of (13) is monotone decreasing in ω_-: for $\omega_- = 0$ we have

$$\sum_v \mathbf{P}\{X_v(\tau_1) \geq 1\} = \mathbf{E}\mathcal{A}(\tau_1),$$

where the last inequality is due to the definition of τ_1, while when $\omega_- \to \infty$ the left part in (13) goes to zero.

Then with the above choice of ω_-

$$\begin{aligned}\mathbf{E}\#\{v : X_v(\tau_1+1) \geq 1\} &= \sum_v \mathbf{P}\left\{\frac{X_v(\tau_1) + \sum_{u\sim v: u\in\mathcal{A}(\tau_1)}\omega}{1+\omega_-} \geq 1\right\}\\ =&\mathbf{E}\mathcal{A}(\tau_1) \approx A(0),\end{aligned}$$

where the approximation is valid for large n: at the first moment of exceeding threshold $KA(0)$ the value of the total excitation is approximately $KA(0)$. This means that from the moment $\tau_1 + 1$ we restart a process of accumulation excitation until next moment

$$\tau_1 + \min\{t : |\mathcal{A}(\tau_1 + t)| \geq a_{thr}\}$$

of triggering inhibition. Since we restart again with about $A(0)$ active excitatory neurons, we have on the average a *cycle* of activity.

Observe, however, that the process restarted at time $\tau_1 + 1$ will differ from the initial process due to the fact that we used already $KA(0)$ neurons. Hence, the cycles will exhibit on the average similar behaviour only as long as the total number of neurons ever activated remains to be $o(n)$. This gives us an upper bound for the number of similar cycles:

$$N_c = o(n)/A(0) = o(n^{1-\gamma_0}).$$

1.6. **STDP implementation for molding the braid structure.** Consider the sets of neurons which start to fire at the same moment of time, these are $\mathcal{A}(t)$, $t \geq 1$. They naturally form a wave of firings passing through a network.

As we discussed above, the number of consecutive firings in a cycle is of order τ_1, while the number of cycles is about $N_c = o(n^{1-\gamma_0})$. This together with (12) gives us an approximate total number of the layers of firing neurons:

$$N_l \approx N_c \tau_1 = o(n^{1-\gamma_0+\gamma}) = o(n^{1-\gamma_0 \frac{1+\beta}{2}}).$$

Recall that we can choose any $1/2 < \beta < 1$ and $0 < \gamma_0 < 1$. Therefore we can have any polynomial $N_l = n^q$ with $0 < q < 1$.

Hence, by the construction we have

$$|\mathcal{A}(t)| < KA(0) = O(n^{\gamma_0}), \quad \text{for all } t \leq N_l.$$

Since $\gamma_0 < 1$ number N_l still can grow with n. Hence, we may assume that the process runs for a long enough time to evoke the STDP. After

$$T := n^{1-\gamma_0 \frac{1+\beta}{3}}$$

steps, which is $o(n^{1-\gamma_0+\gamma})$, we increase all the excitatory connections from the neurons which were firing at least once up to time T to the neurons which were firing at least once up to time T, and weaken all the other connections from the same neurons. Some evidence that the synaptic weights may follow a discrete distribution was reported by Montgomery and Madison (2004). One can also argue that even pruning of weak connections may take place (Iglesias and Villa (2007)).

As a result we will get the following braid structure of strong synapses (embedded into the pool of other not yet modified synapses):

i) to every neuron in $\mathcal{A}(t), t < T$, there is a connection from at least one of neurons in $\mathcal{A}(t-1)$,

ii) to every neuron in $\mathcal{A}(t), t < T$, there are at most $r-1$ connections from the sets $\mathcal{A}(s), s \leq t-2$,

iii) to every neuron in $\mathcal{A}(t), t < T$, there are at least r total connections from all the preceding sets $\mathcal{A}(s), s \leq t-1$.

We call this structure "braids" rather than chains simply due to the feature that strong connections may exist not only between the consecutive layers of neurons.

The parameters of this structure are the following: the width is bounded by n^{γ_0} and the length of the chain (or braid) is about $n^{1-\gamma_0 \frac{1+\beta}{3}}$.

1.7. **Conclusions.** We describe a process of establishing feed-forward braid structures (syn-fire chains) in a network of integrate-and-fire neurons. It has been repeatedly reported that such systems usually fail to produce stable long syn-fire chains; instead either a convergent or a divergent regime is most probable. We show that properly tuned inhibition may help to keep a system in the near critical regime when the system has an almost sustained activity level. We provide quantitative bounds for the parameters of the established braids.

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