Emergence of a selective synchronous response through STDP in recurrent neural networks

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Abstract

This paper presents simulation results on a balanced recurrent neural network of spiking neurons with a simple implementation of the Spike-Timing Dependent Plasticity (STDP) rule, whose potentiation and depression effects compensate. The synaptic weights and delays are randomly set and the network activity, which is a combination of an input signal and a recurrent feedback, is initially strong and irregular. Under a static stimulation, the plasticity process shapes the initial activity toward a more regular and synchronous response. The response is specific to this particular stimulus: the network has learned to select by synchrony one arbitrary stimulus from a set of random static stimuli. The encoding of multiple stimuli is then tested and we study the effect of the size on the encoding capacity.

Key words: random recurrent neural network, STDP, synchrony, perception

1. Introduction: the STDP framework

Biological evidence of long-term plasticity [2], where a synapse is potentiated when the presynaptic neuron fires shortly before the postsynaptic one and depressed in the opposite case in a nearly anti-symmetrical way has been formalized as the Spike-Timing Dependent Plasticity (STDP) rule [1, 13, 21]. This rule has inspired various and somehow contradictory studies these last years in the ANN community. An important effect of STDP is that it reduces the latency of a neuron's response to a given input [18, 8]; the behaviour of

STDP has also been studied on larger, recurrent networks, but the results are more ambiguous: while it has been shown that in some conditions it can facilitate the emergence of neuronal groups [11, 10], these results seem to depend on the precise implementation, since a slightly different rule on a different neuron model gives different results: there is no structure development, strong synapses remaining unstable [14].

It has also been shown, both in simulation [?] and in biology [5], that STDP could enhance the synchronization of a neural network; however, here again, seemingly contradictory results show that STDP can lead to desynchronization when there are non-null constant delays [?].

We present here a series of simulations where connection strength between neurons is modified by a simple implementation of STDP. We show that in our model, such a learning process allows to distinguish by synchrony an arbitrary stimulus from a set of static stimuli associated to strong and irregular self-driving activity. The section 2 presents our model (an integrate-and-fire neural network model under the influence of static stimuli). The sections 3 and 4 illustrate how a particular stimulus may trigger a specific synchronous response from a set of arbitrary stimuli. The section 5 investigates how many different stimuli can be encoded this way.

2. Model: heterogeneous weights and delays

Neuron model. The neural networks we simulate are random and recurrent, implemented with discrete leaky integrate-and-fire neurons. Consider a set of neurons labelled by indexes $i \in \{1, ..., N\}$. The neuron activity $\{S_i(s)\}_{s<t}$ is defined as a sum of Diracs corresponding to the series of spikes emitted up to the t instant (see [7]). Taking into account the absolute refractory period τ_r and the firing threshold θ, the current activity $S_i(t)$ is defined the following way:

$$S_i(t) = \begin{cases} \delta(0) \text{ if } \max_{s\in[t-\tau_r,t[}(S_i(s)) = 0 \text{ and } V_i(t) \geq \theta; \\ 0 \text{ elsewhere} \end{cases} \tag{1}$$

where $\delta(0)$ is a Dirac impulse and $V_i(t)$ is the neuron's membrane potential, defined according to the Leaky Integrate-and-Fire (LIF) differential scheme:

$$\begin{cases} V_i(t) = V_i^{in}(t) + V_i^{ext}(t) \\ \frac{dV_i^{in}}{dt} = -\frac{V_i^{in}(t)-V_0}{\tau_m} - S_i(t)(V_i^{in}(t) - V_S) + \sum_{j=1}^{N} J_{ij} S_j(t - \tau_{ij}) \end{cases} \tag{2}$$

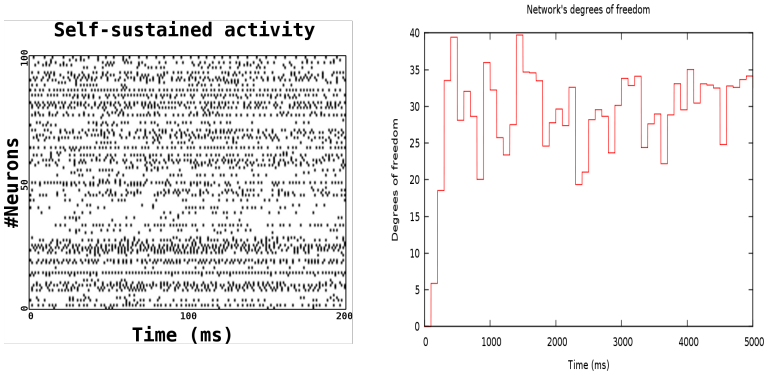
where V_i^{ext} is a superimposed potential, V_0 is the resting potential, V_S is the refractory potential, τ_{ij} and J_{ij} are respectively the transmission delay and synaptic weight from neuron j to neuron i and τ_m is the membrane time constant. We set $\tau_r = 2$ ms, $\tau_m = 10$ ms, $V_0 = 0$, $V_S = 0$ and $\theta = 1$. In the simulations, we use a simple first order integration with resolution $\delta t = 1$ ms.

Weights setting. The individual weights and delays are independent and strongly heterogeneous. Our parameters are chosen in order to allow the internal self-sustained activity to compete with the external stimulation. The expected value of the weights sum is $\mu_J = 0$ so that the excitatory influences compensate the inhibitory ones (balanced networks configuration). The weights sum standard deviation σ_J is set in the range [2-3]. This value corresponds to a strong internal influence (which is typically the case when the weights sum overtakes the threshold – here $\theta = 1$). The synaptic weights are set according to a Gaussian draw $\mathcal{N}\left(\frac{\mu_J}{N}, \frac{\sigma_J^2}{N}\right)$, while the axonal transmission delays are set according to a Poisson draw of expected value $\lambda_\tau = 10$ ms. The simulations take place on rather small neural networks composed of N neurons (we take here N in the range [100-1000]).

Stimulus presentation. The inputs sent to the network are distributed among every neuron. We define a set of P static stimuli $(\mathbf{V}^{(p)})_{p=1..P}$ which are random vectors of size N and whose values are randomly set according to a gaussian draw $\mathcal{N}(0, \sigma_V^2)$ where $\sigma_V = 1$. The stimuli are qualified as "static" i.e. when a stimulus is presented to the network, it lasts for a duration which is greater than the mixing time of the system in order to converge to a stationary response.

The external stimulus is directly sent to the potential, bypassing the membrane leak. Practically, it modifies the threshold value and thus the excitability of the neuron. It must be noticed that when $V_i^{ext} > 1$, the neuron spontaneously fires at its maximal firing rate if not inhibited by other neurons. This "potential-based" presentation garantees that a subset of neurons is active in the absence of internal feeding.

Neural networks simulations. The activity which spontaneously develops in the network depends both on the initial conditions (the initial values of the neurons potentials) and on the current stimulus. Our networks are recurrent and behave as non-linear dissipative dynamical systems. As such, they can develop a self-sustained autonomous activity. An example of such self-sustained activity is given in figure 1A.



- A - - B -

Figure 1: - A - Activity of a recurrent neural network with self-sustained activity. $\sigma_J = 3.0$; $\tau_r = 2$ ms. Raster plot with time on x-axis and neuron index on y-axis. One dot corresponds to one spike. - B - Estimated number of degrees of freedom during 5 seconds of simulation.

In the range of parameters we have chosen, the networks activity looks very irregular at first glance. First, the *asynchrony* of the activity directly results from the balance between the excitatory and inhibitory influences [3]. Second, the irregularity of the activity is a well-known feature of recurrent heterogeneous networks [6, 19].

In order to characterise this irregularity, we use an estimation of the effective number of Degrees of Freedom (#DOF) [20, 15]. A Principal Components Analysis is first applied to the data set, followed by a calculation of the entropy of the normalized principal values p_i of the transformation matrix: $S = -\Sigma_{i=1}^{N} p_i \log(p_i)$. This value is considered as an approximate log count of significant principal components weighted by their respective size, so that $\#DOF = e^S$ is an approximation of the effective number of degrees of freedom. In Figure 1B, the estimated #DOF is calculated every 100 ms during 5 s of simulation, giving values around 30 for a network of 100 neurons, showing that the individual activities are almost mutually independent.

3. Weight adaptation: emergence of synchrony

Several implementations of the STDP update mechanism have been proposed in the literature. Our implementation is all-to-all [16] and additive: the weight update depends on every previous synaptic event (the influence

of the less recent events fading with time) and doesn't take into account the current weight of the synapse. Concretely:

$$\frac{dJ_{ij}(t)}{dt} = \frac{\alpha}{N}\left[S_i(t)\varepsilon_j(t-\tau_{ij}) - \varepsilon_i(t)S_j(t-\tau_{ij})\right]$$

where the trace $\tau_m \frac{d\varepsilon_i}{dt} = -\varepsilon_i + S_i(t)$ can be considered as a short-term estimation of the firing rate of the i^{th} neuron. This update scheme divides in two terms. The first term corresponds to the synaptic potentiation: a significant weight increment takes place when a post-synaptic spike is associated with a strong pre-synaptic trace (recent pre-synaptic excitations). The second term corresponds to the synaptic depression: the weight significantly decreases when a pre-synaptic pulse is associated with a strong post-synaptic trace (i.e. after post-synaptic excitation). The result is a global facilitation of pre-post sequences of excitation and a depression of post-pre sequences of excitation. This rule is strictly antisymmetrical, i.e. the potentiation compensates the depression, so it can be considered as "balanced". From a computational point of view, the storage of a trace is not very expensive. It is moreover strictly local and as such biologically implementable.

We present in figure 2 some aspects of the activity change during the plasticity process. As we said, the initial activity is rather unstructured and irregular. The figure gives an example of a regime transition taking place during the plasticity process. This regime transition manifests in a *simplification* of the ongoing neuronal activity. The plasticity process lasts between $t = 5$ s and $t = 25$ s on a network under a static input. The value of the plasticity coefficient is $\alpha = 0.5$.

Figure 2A presents a sliding autocovariogram of the average membrane potential. The series of red stripes which appear in the autocovariogram testifies of a strong periodic shaping. This periodicity of the mean signal correponds to an increase of the synchrony of the individual neurons, with ah internal period converging to 60 ms (16 Hz). This synchrony raise is also directly visible on the activity plot. Figure 2B) shows that the estimated #DOF suddenly decreases during the plasticity process. At the end, the individual activities are correlated one to the other. Figure 2C shows the activity of every neuron between 5 s and 15 s, with a "zoom" on activity after STDP. It shows that the transition from irregular activity to synchrony isn't straightforward: there is a time interval (around $t = 10\ s$) of strong instability, where the two regimes "coexist", causing multiple transitions and a provisional burst of complexity.

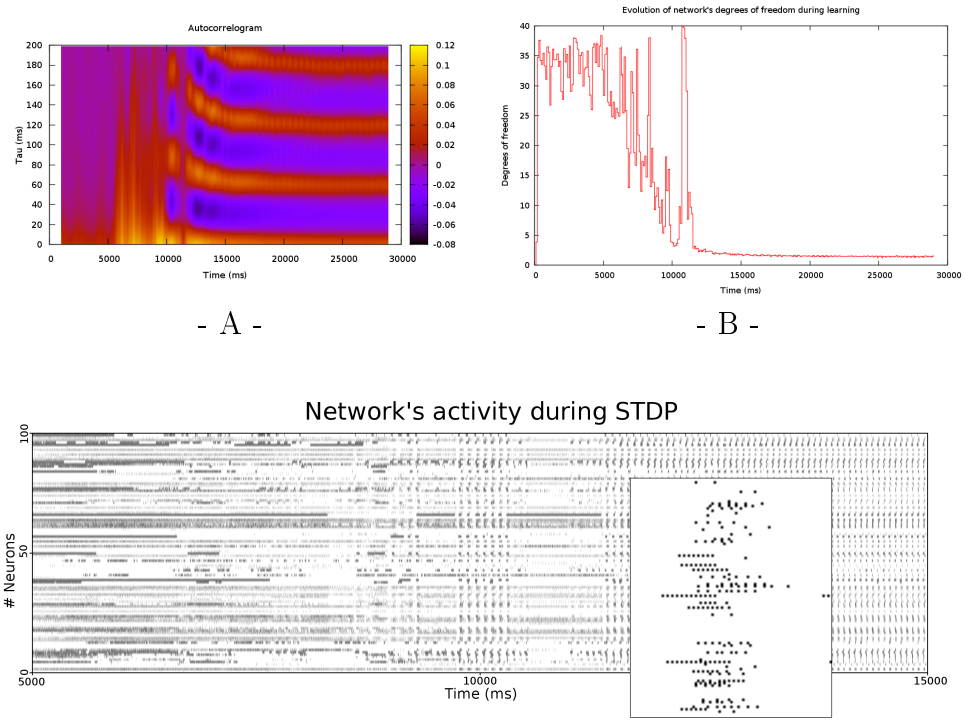


Figure 2: - **A** - Evolution of the autocovariance of the average membrane potential during the STDP process. At each time step t, the mean potential $\bar{V}(t)$ is covaried with $\bar{V}(t+\tau)$ for τ in 0..200 ms. The red zones denote a high covariance while the deep blue zones denote low covariance values. - **B** - Evolution of the #DOF measure during the STDP process - **C** - Activity of the network in the first 10s of the STDP process. The zoomed part present a detailed view of the activity after STDP on a time window of 100 ms.

The final synchronous regime is thus characterized by occcasional bursts of activity followed by silence, in a cyclic manner with a period of 60-70 ms. Under our settings, the STDP consolidates two path : an excitatory path from the externally excited neurons to a subset of internally activated neurons, and a reverse inhibitory path from the late neurons to the externally excited ones, which results in a srong inhibition of the externally excited neurons and then a progressive extinction of the network activity. The neurons with strong external excitation then progressively start to fire again and a new cycle begins.

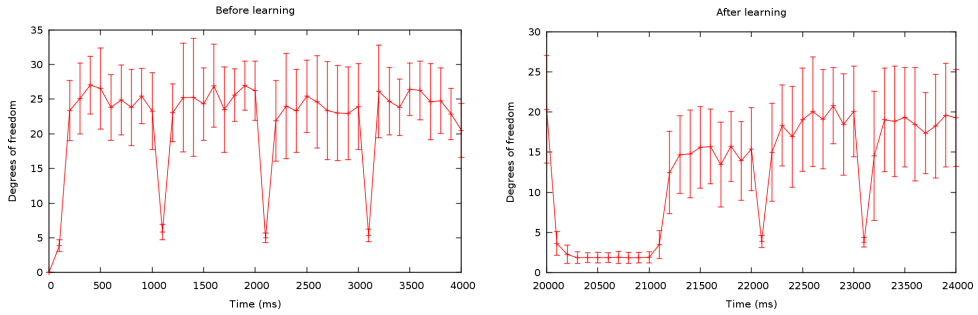


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Figure 3: Mean and variability over ten networks of the number of degrees of freedom for a similar sequential presentation of the four stimuli. - **A** - Before STDP ($0 < t < 4$ s). - **B** - After encoding the first stimulus (20 s $< t <$ 24 s).

4. Encoding and selectivity

We address here the question of encoding and input selectivity. Is the network response specific to the presented stimulus or would it respond in the same way to other stimuli? We tested the network response to 4 arbitrary stimuli before and after STDP. In order to do so, the 4 different stimuli are presented sequentially for 1 second each. STDP is applied with a strong coefficient ($\alpha = 5$) on a short period of time (1 s – between 12 s and 13 s) where only the stimulus #1 is presented. Before this session, all the stimuli trigger a response with similar complexity. After the STDP session, the pattern of activity for the first stimulus is significantly simplified, making it dissociable from the other patterns of activity. We tested the reproducibility of this experiment performing the same simulation on 10 different networks (identical parameters, but different initial random settings for the weights, delays and inputs): every network gives a similar reduction of the #DOF measure (see figure 3).

In the given configuration (strong coefficient, short plasticity process), the response remains specific to the presented stimulus. We also verified that such a periodic shaping is systematically observed for other parameter configurations, even if the patterns of activity are not always synchronous. Moreover, such an encoding process relies on a fine tuning of both the coefficient value and the plasticity session duration. Some complexity decrease for the other stimuli, which is visible on the mean #DOF, may be amplified if the session lasts too long or if the STDP coefficient is too strong (saturation effect). A more systematic study of our system's loading capacity is

presented in the next section.

5. Encoding several stimuli

We now investigate whether this model can be extended to the encoding of several stimuli and what are the effects of the network size on its capacity.

Stimulus presentation. We consider several "epochs" of 10 seconds where ten stimuli are sucessively presented for a duration of 1 second, where the total simulation time is 100 seconds (10 epochs). In each epoch, except the first and last one, Spike-Timing Dependent Plasticity is applied to a different stimulus; i.e. we try to "encode" 8 stimuli out of the 10.

Ad-hoc interruption of the plasticity process. When the plasticity process is applied for a too long duration, the synchronous pattern of activity is consolidated so much (because of the periodicity) that the network's internal activity becomes more prominent than the external signal, so that the network eventually becomes oblivious to other inputs and carries on repeating the same periodical pattern. In order to limit the saturation effects and estimate to the best the capacity, we control the *duration* of the plasticity process, by stopping the synaptic plasticity when the #DOF estimation is higher than a "critical" threshold $\#DOF_c = 4$. This mechanism is not intended to be realistic, even if "some" mechanism may exist in the brain in order to regulate the plasticity and avoid, when possible, the saturation effects.

Encoding test. We test during each epoch the possibility to distinguish the encoded stimuli from the not yet encoded ones. For a given epoch k, we ignore the k^{th} stimulus, currently undergoing STDP. We calculate the mean #DOF associated with every other stimuli, and consider that the $k-1$ imprinted stimuli are distinctly encoded if and only if

$$\max(\{\#DOF(V^{(p)})\}_{p=1..k}) < \min(\{\#DOF(V^{(p)})\}_{p=k+1..P})$$

where $\#DOF(V^{(p)})$ is the mean number of degrees of freedom on the time interval where the system is fed with the stimulus $V^{(p)}$. From this statement, we estimate the capacity of the network as the greatest k for which the test is achieved.

This test is rather coarse and only addresses the distinctiveness between the encoded an non-encoded stimuli. It doesn't address the distinctiveness

among the encoded stimuli. It can however be consistently used in order to investigate the particular influence of the size effects on the network capacity.

Forgetness and Saturation. When looking precisely at the degrees of freedom of particular sets of parameters (see figure 4A), we find that increasing the size increases the ability to differentiate between the learned stimuli and the unknown ones, since the contrast between the learned stimuli #DOF and the unknown ones is stronger.

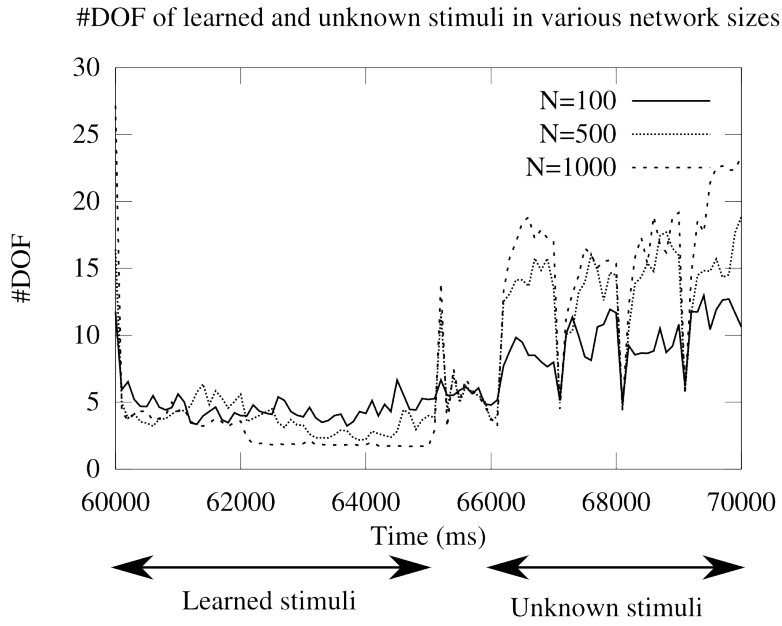
There is also a dependence on the plasticity coefficient: if it is too weak, the first stimuli having been learned are "forgotten" and carry out an irregular output with higher number of degrees of freedom (see figure4B), whereas if it is too strong the saturation takes place and even the unknow stimuli carry out low #DOF. There is thus a range of values allowing the network to produce a "correct" encoding, which depends both on the size and the initial network parameters.

We more generally find that the best encoding is obtained for rather high plasticity coefficients, namely $20 < \alpha < 60$, which allow for a short plasticity process duration (< 1 s).

Capacity. We apply the encoding procedure for various ranges of parameters: different network sizes and learning rates. For each parameter set, we test 10 different networks (i.e., different random seed ; different networks also receive different sets of stimuli). At each epoch, we test the ability to differentiate the k learned stimuli from the $10 - k$ non-learned ones, with k in 1..8. This ability was tested for various sizes and various plasticity coefficients.

We find that different networks (i.e. different random seed) reach their maximal performance for different plasticity coefficients. We thus calculate the optimal rate of correct encoding of each network by selecting the learning coefficient which maximizes the encoding capacity. The results are presented on figure 5. We find that for $N \geq 200$, almost every network is able encode the 8 stimuli (88% achievement for $N = 200$, 96% achievement for $N = 500$, 100% achievement for $N = 1000$ - see figure 5 -).

Classical results on the capacity of recurrent neural networks display a proportional relationship between the size N and the maximal number of stimuli that can be correctly encoded/decoded by the system [9]. As the number of encoded stimuli is limited to 8 in the simulation, the figure gives an estimation of the capacity *only* for the small networks (namely $N \leq 100$). We see that, for $10 \leq N \leq 100$, the capacity increases linearly with the



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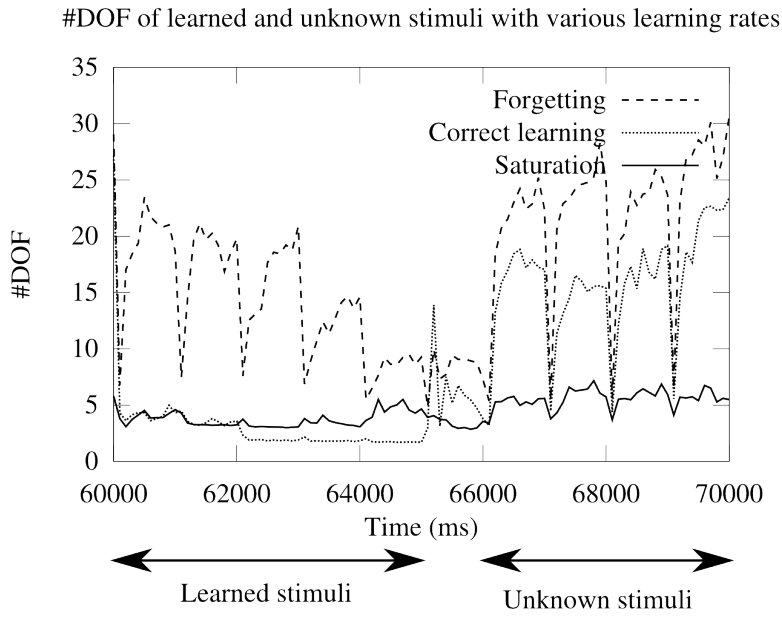

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Figure 4: Average of the number of degrees of freedom on ten networks, in the sixth epoch (the sixth stimulus is being learned). **-A-** For various network sizes: 100 neurons, 500 neurons and 1000 neurons. We take the optimal plasticity coefficient for each size, respectively 20.0, 40.0 and 60.0. **-B-** With a network of 1000 neurons, for plasticity coefficient leading to the forgetting of previously learned stimuli ($\alpha = 20.0$), correct encoding ($\alpha = 60.0$) or synaptic saturation ($\alpha = 100.0$).

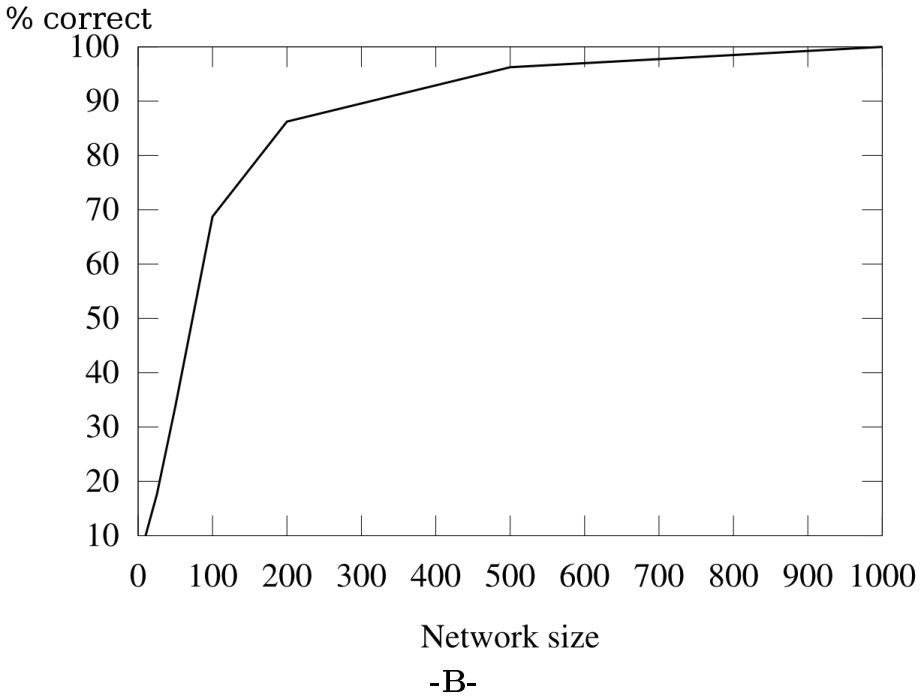

Figure 5: % of correct encoding according to size (see text), averaged on 10 networks, for $N = 10$, $N = 20$, $N = 25$, $N = 50$, $N = 75$, $N = 100$, $N = 200$, $N = 500$ and $N = 1000$.

size, with a mean capacity of 5.5 for $N = 100$ for instance (giving a loading capacity $\alpha_c \simeq 0.05$).

6. Discussion: which input selection in biological networks?

We have presented a neuronal model of perception which encompasses the effects of recurrent self-feeding activity and a simple, biologically-implementable, STDP mechanism which allows to learn to differentiate by phase transition one stimulus out of a set of several statistically equivalent stimuli. The learned stimulus is found to drive a regular and synchronous pattern of activity while the response remains unstructured for the other stimuli. While the synchronisation effect of STDP appears in other works [10, 14], albeit in different circumstances, its dependence to a particular stimulus is to our knowledge unheard of.

This study emphasises the role of qualitative transitions rather than mean activity changes, and may be considered as complementary to the classical

feed-forward input selectivity mechanisms. It is highly probable that several extraction mechanisms combinate in the real brain to produce appropriate responses. For instance, the presence of both synchronous activity and STDP may help to propagate the sensory signal to deeper layers by a synchrony consolidation effect, as proposed for instance for the olfactory bulb by [12, 4].

Some questions remain at the present stage, such as the possibility to have a biologically plausible regulation mechanism for STDP in order to neither "forget" the first stimuli encoded nor saturate the network. This study only presents the first stages of a neural encoding mechanism that may take place in perception processes, and suggests to study wether a single plasticity mechanism may allow not only to facilitate the encoding, but also the transmission and the decoding of neural activities. This study would also benefit a comparison with real data, in particular in the olfactory [12] or motor [17] systems.

Acknowledgement

This work is supported by the ANR "MAPS" (Mappings, Adaptation, Plasticity ans Spatial Computation) of the French Ministry of Research.

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