Synfire Chain in a Balanced Network

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Abstract

We investigate the formation of ordered spatiotemporal activations of pools of neurons in synfire chains (SFC) within a balanced network, both by simulations and by analytic tools. Using a suitable matrix of synaptic connections, we show that the results depend on the ratio between the size of an individual pool of neurons (w) and the total excitatory input of a neuron (K). In our simulations of 10,000 neurons, we obtain an asynchronous-irregular firing mode, which does not sustain a traveling pulse-packet of SFC activity. Our analysis shows that the latter may be expected to exist in larger networks in which very small w/K values can be realized.

Keywords: synfire chain, balanced network, asynchronous irregular state, common input, integrate and fire neuron.

1 Introduction

A randomly connected sparse network, where each neuron gets as much inhibition as excitation is called a “balanced network” [2]. Such a network of integrate-and-fire (IAF) neurons is known to have a regime of parameters with stable *asynchronous-irregular* (AI) activity [2]. In the AI state, population activity is *asynchronous* and the individual neuron's activity is *irregular*. When a balanced network operates in the AI regime, its neuronal firings are driven by the fluctuations of its input, rather than by its mean input. Hence, the neurons fire irregularly although they integrate a huge number of inputs. This property of balanced networks as well as their ability to respond fast to external stimuli [5] makes them an appropriate candidate for modeling a cortical neural network.

In this paper, we study the stability of the AI state of a balanced network in the presence of ordered connectivity. We show that one can depart from the random connectivity assumption and still keep the AI state stable. In particular, we are able to embed microcircuits of synfire chains (SFCs) in a balanced network without losing the AI state.

SFC [1] is a feed-forward multi-layered architecture (a *chain*), in which spiking activity can propagate in a synchronous wave of neuronal firing (a *pulse packet*). The chain is made of a sequence of *pools* of neurons. A neuron in a pool receives afferent inputs from all neurons in the previous pool, while projecting its output to all the neurons in the consecutive pool. A neuron also receives many other afferents from the network and projects many outputs to other neurons in the network. The notion of pulse packet was developed in [3], where it was found that for pool size large enough, the pulse packet converges to a synchronous firing pattern, whereas for small pool size, the pulse packet dissolves and is no longer distinguishable from the background activity.

We begin by analyzing the effect of common input on a pair of neurons in a simple neuronal model. We then use this result in our numerical study of a balanced network whose excitatory connections are superpositions of SFCs.

2 A pair of neurons in a pool

As a preparation for the understanding of pulse-packet propagation, we begin by studying the correlations of two neurons that belong to the same pool on a chain. This chain is assumed to belong to a network whose connectivity is neither entirely random nor entirely structured. In contrast to a random network, where a pair of neurons shares a small number of common inputs, our network contains many pairs of neurons that share at least a pool as their common inputs. This is due to the specific wiring of a chain, where a pair of neurons in a pool shares common input from the previous pool. Clearly, a common input to a pair of neurons induces correlation between the pair, even if the inputs are nothing but noise. We use a simple model of binary neurons to study the relation between the size of the common input and the correlation induced by the common input.

1 The simple model

We look at a pair of neurons in a pool, each having *K* excitatory synapses and *K* inhibitory synapses. *w* of their excitatory synapses receive the same input (due to the previous pool on the chain). We enumerate the synapses such that the first *w* synapses are common and the last *K*-*w* are not. We assume *w* and *K* to be large.
Let $s_i(t)$ be a stochastic point process representing the afferent input to the *i*-th synapse of a neuron. For clarity, we discard the time dependence notation of the s_i's. The s_i's are correlated binary variables with the following characteristics:

$\langle s_i \rangle = \nu$

$\mathrm{var}(s_i) = \sigma_s^2$

$\langle s_i \cdot s_j \rangle = \rho^{in}\sigma_s^2 + \nu^2$, where ρ^{in} is the correlation coefficient. ρ^{in} is 0 if *i, j* are not members of the same pool.

We assume, therefore, no correlations among inputs that are not common to both neurons. This assumption may not be valid in a full network.

2 Correlation of fields

We start by partitioning the input field of a neuron into three sub-fields: Inhibitory (I), independent-excitatory (X) and common-excitatory- (Z). We calculate the membrane potential that is the result of incoming post-synaptic potentials in each of the three sub-fields.

Let *Z* be a random variable that represents the local field generated by the first *w* excitatory synapses: $Z = \sum_{l=1}^{w} J \cdot s_l$, where *J* is the (single) synaptic weight.

The statistics of *Z* is as follows:

$\mu_Z \equiv \langle Z \rangle = Jw\nu$

$$\left\langle Z^2 \right\rangle = J^2 \left\langle \left(\sum_{l=1}^{w} s_l \right)^2 \right\rangle = J^2 w \left\langle s^2 \right\rangle + J^2 w(w-1) \left\langle s_i \cdot s_j \right\rangle = J^2 \left[w\left(\sigma_s^2 + \nu^2\right) + w(w-1)\left(\rho^{in}\sigma_s^2 + \nu^2\right) \right]$$

Note that we assumed here that the inputs that do not come from the previous pool are uncorrelated, i.e. $< s_i s_j >= \nu^2$.

Since we assume large *w*, we can apply the central limit theorem, leading to:

$Z \sim N(\mu_Z, \sigma_Z)$, with $\mu_Z = Jw\nu$ and $\sigma_Z = J\sigma_s \sqrt{w\left(1+\rho^{in}(w-1)\right)}$.

Let X_1 and Y_1 be random variables that represent the local sub-fields of the first neuron generated by the independent *K*-*w* excitatory synapses and *K* inhibitory synapses, respectively. A derivation similar to that of sub-field Z leads to:

$X_1 \sim N(\mu_X, \sigma_X)$, with $\mu_X = J(K-w)\nu$ and $\sigma_X = J\sigma_s \sqrt{K-w}$.

$I_1 \sim N(\mu_I, \sigma_I)$, with $\mu_I = -JK\nu$ and $\sigma_I = J\sigma_s \sqrt{K}$

Let h_1 be the sum of the three uncorrelated, normally-distributed sub fields of the first neuron: $h_1 = X_1 + Z + I_1$.

The mean of *h* vanishes by construction, due to the balanced network assumption, whereas the variance is given by $\sigma_h^2 = \sigma_X^2 + \sigma_Z^2 + \sigma_I^2 = J^2\sigma_s^2\left[2K + w(w-1)\rho^{in}\right]$.

Similarly, we define $h_2 = X_2 + Z + I_2$, where X_2 and I_2 are the respective sub-fields of the second neuron.

Clearly, $\left\langle h_2 \right\rangle = \left\langle h_1 \right\rangle$ and $\left\langle h_2^2 \right\rangle = \left\langle h_1^2 \right\rangle$. The covariance of the two fields, however, depends on their common input:

$$\left\langle h_1 \cdot h_2 \right\rangle = \left\langle (X_1 + Z + I_1)(X_2 + Z + I_2) \right\rangle = \sigma_Z^2 + (\mu_X + \mu_Z + \mu_I)^2 = \sigma_Z^2$$

The correlation coefficient is:

$$(1) \qquad \rho^h \equiv \frac{\left\langle h_1 \cdot h_2 \right\rangle - \mu_h^2}{\sigma_h^2} = \frac{\sigma_Z^2}{\sigma_X^2 + \sigma_Z^2 + \sigma_I^2} = \frac{w + w(w-1)\rho^{in}}{2K + w(w-1)\rho^{in}}$$

Note that ρ^h is not a function of ν or σ_s in this model, i.e., the correlation between the fields is neither a function of the input rates, nor a function of the input variance. In Figure 1 we display the correlation of the two fields as a function of the correlation in the inputs for a series of *w*=10, 50, 500, emphasizing the fast jump in behavior from small to high correlations.

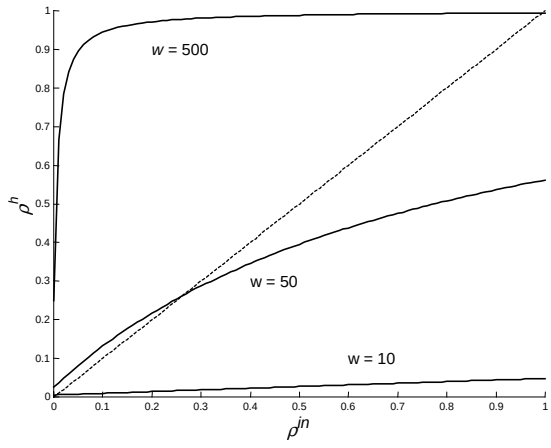

Figure 1: Correlation in the fields as a function of the correlation in the common input. *K*=1,000. All the curves are concave and intersect the diagonal (dashed line) at a single point.

3 Fixed-point correlations

Next, we recall that our pair of neurons resides in a pool, which in turn is a part of a chain. Using equation (1), we are able to follow the evolution of correlation from the common input to the fields. If we further assume that the correlation between the output spikes, ρ^{out}, is equal to the correlation between the fields, ρ^{h}, we can calculate ρ^{out} as a function of ρ^{in}. Indeed, simulations of a pair of IAF neurons that operate in the irregular regime show that the correlation between the output spikes is almost equal to the correlation between the fields.

Hence, we equate ρ^{h} of a pair of neurons in a pool with ρ^{in} of the consecutive pool. We define $\rho^{*} \equiv \rho^{in} = \rho^{h}$, to be the fixed-point correlation, and we solve Eq. (1) for ρ^{*}. The intersections of the curves in Figure 1 with the diagonal are these fixed points. The fixed-point correlation curve is plotted in Figure 2, as a function of *w*, for several values of *K*.

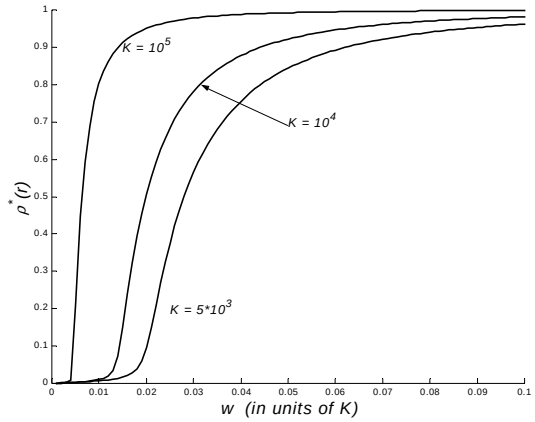

Figure 2: The fixed-point correlation as a function of *w*, for several values of *K*. Note the sharp transition between low and high fixed-point correlations.

3 The network

1 Integrate-and-fire neurons

Following Brunel [2], we use an IAF model, in which a neuron's membrane potential, $v_i(t)$, obeys the equation:

$$\tau \frac{dv_i(t)}{dt} = -v_i(t) + RI_i(t), \quad (2)$$

where $I_i(t)$ is the synaptic current arriving at the soma. Spikes are modeled by delta functions; hence, the input is written as

$$RI_i(t) = \sum_j \sum_{t_j^f} J_{ij}\delta(t - t_j^f - D), \quad (3)$$

where the first sum is over different neurons, while the second sum represents their spikes arriving at times $t = t_j^f - D$. t_j^f is the emission time of the *f*-th spike at neuron *j*, and *D* is the transmission delay. The sum is over all neurons that project their output to neuron *i*, both local and external afferents.

When $v_i(t)$ reaches the firing threshold θ, an action potential is emitted by neuron *i*, and after a refractory period τ_{rp}, during which the potential is insensitive to stimulation, the depolarization is reset to v_{reset}.
The following parameters were used in all simulations: The transmission delay D = 1.5ms, the threshold θ = 20mV, the membrane time constant τ = 10 ms, the refractory period τ_{rp} = 1ms and the resetting potential v_{reset} = 10mV.
We used the Synod simulation environment [4]. The Runge-Kutte method of order 2 was used for integration, with time steps of 0.1ms.
As this model is similar to the previous simple model, we can expect the effect of correlations in the input on the output correlations to stay the same as in the simple model. Thus, we presume the fixed-point correlations to behave as depicted by the curve in Figure 2.

2 Network architecture

We connect two neurons in the network according to the following rule:

If the connection is of one of the I->E, E->I or I->I types, then the neurons are connected with probability K/N_E. Again, we follow Brunel [2] by forcing each neuron to receive exactly *K* afferents.

Else, for E-E connections, we consecutively choose a random pool of *w* neurons and connect them to the previous pool in an all-to-all manner. Each neuron can participate in no more than K/w pools, thus, in the process of choosing a pool, we discard any neuron that exceeds its limit of afferents, and we draw randomly another neuron. After embedding the maximum possible number of pools, which is $N_E K/w^2$, we add random excitatory connections, so that each neuron receives exactly *K* excitatory afferents.

3 Results

For the following synaptic coupling: $J_{IE} = J_{EE} = 0.1mV$, $J_{EI} = J_{II} = -0.5mV$, and external rate $\nu_{ext} = 20Hz$, we get a strong dependence of network activity on *w*, as expected from the simple model.
The population activity, i.e. the percentage of firing neurons in the excitatory population in millisecond bins, is presented in Figure 3.

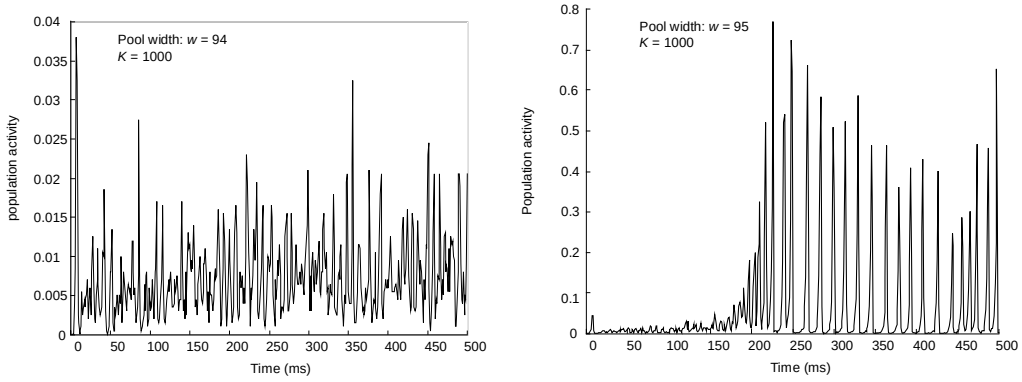

Figure 3: Population activity of 10,000 excitatory neurons in an IF network including also 2500 inhibitory neurons. Each excitatory neuron receives K=2,000 inputs (1000 from the excitatory population and 1000 reflecting external input) and 250 inhibitory inputs. Changing the size of the pool *w* from 94 To 95 switches the network from an asynchronous-irregular mode to a synchronous one, exhibiting global oscillations. Note the different scales on the two plots.

We note that global oscillations are exhibited by the population activity, as w crosses the critical value.

4 Discussion

Figure 2 demonstrates that in the limit of large K, there is a critical value of w, w_c, which separates two regimes. If $w>w_c$, the pools fire in tandem with no regards to their input, as long as their external input is larger than some threshold. With all pools activated simultaneously, the chain loses its computational meaning. If however, $w<w_c$, all neurons fire asynchronously, given an asynchronous input.
From Figure 3, we learn:

(i) It is possible to attain a stable state of AI activity in a balanced network, even if the synaptic matrix contains a superposition of chains.
(ii) If the pools' size exceeds a critical value, the asynchronous activity is replaced by global oscillations.

These observations are explained by the curve of Figure 2: The global oscillation is a result of the inability of the network to sustain a stable AI state due to excess of pair wise correlations, which in turn, are the result of $w>w_c$.

Correlation between uncoupled neurons due to common and synchronous input was also studied in [6]. Their setup is similar to our *pair of neurons in a pool*, but with conductance based IAF neurons. In accordance with our conclusions, they found that correlation of the output spikes is strongly enhances by the correlation in the common excitatory input. The level of fixed-point correlation in a chain was not studied, but their result seems to lead to the same curve depicted in Figure 2.

The $w<w_c$ regime is adequate, in principle, for transporting pulse-packets along chains. For large enough $w<w_c$, igniting the first pool, the pulse packet can propagate or dissolve, depending on the size of w, the strength and width of the pulse packet. See [3] for details. Using only $N=10^4$ neurons a traveling pulse packet cannot be obtained in our balanced network. In order to obtain large enough w, while maintaining the AI regime, we need w_c to be large; hence, also K has to be large. For our neuronal model, we estimate that for $N=10^5$ neurons with $K=10^4$, w_c is large enough for pulse-packets to propagate synchronously in an AI state of a balanced network.

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