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# Control of network bursting discharges by local electrical stimulation in spiking neuron network

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***Abstract***. *Goal*. The paper is devoted to controlling the dynamics of spike neural networks by local periodic stimulation of various network sections. *Methods*. The simulation uses a network of synaptically connected spike neurons distributed in two-dimensional space. The dynamics of the transmembrane potential of neurons is described by the Izhikevich model, short-term synaptic plasticity is represented by the model Tsodyksa–Markram, the effects of changes in the efficiency of connections between neurons are modeled using spike-timing-dependent plasticity (STDP). *Results*. It is shown that the model reproduces the dynamics of living neural networks grown under in vitro conditions quite well. In its spontaneous dynamics, such a network exhibits a wide range of dynamic modes, including asynchronous spikes and quasi-synchronous spike bundles. It was found that due to STDP, the network can adapt to the stimulating signal, so that the network bundles become synchronized (phase-synchronized) with the stimulation signal. The analysis of the dependence of this effect on the parameters of stimulation, in particular, on the geometric dimensions of the stimulated area, as well as the connectivity of the network. *Conclusion*. With the help of local periodic stimulation of a part of the neural network,when selecting certain parameters of the stimulating signal, taking into account the characteristics of the network, externalcontrol of the dynamics of the spike neural network is possible.

***Keywords***: mathematical modeling, spiking neuron network, control, stimulation.

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# Управление сетевыми пачечными разрядами локальной стимуляцией в спайковой нейронной сети

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***Аннотация.*** *Цель.* Работа посвящена управлению динамикой спайковых нейронных сетей с помощью локальной периодической стимуляции различных участков сети. *Методы.* Для моделирования используется сеть синаптически связанных спайковых нейронов, распределенных в двумерном пространстве. Динамика трансмембранного потенциала нейронов описывается моделью Ижикевича, кратковременная синаптическая пластичность представлена моделью Цодыкса–Маркрама, эффекты изменения эффективности связей между нейронами моделируются с помощью синаптической пластичности, зависящей от времени спайков (STDP). *Результаты.* Показано, что модель достаточно хорошо воспроизводит динамику живых нейронных сетей, выращенных в условиях in vitro. В своей спонтанной динамике такая сеть демонстрирует широкий диапазон динамических режимов, включая асинхронные спайки и квазисинхронные пачки спайков. Обнаружено, что благодаря STDP, сеть может адаптироваться к стимулирующему сигналу, так что сетевые пачки становятся синхронизированными (синхронизированными по фазе) с сигналом стимуляции. Проведен анализ зависимости данного эффекта от параметров стимуляции, в частности, от геометрических размеров стимулируемой области, а также от связности сети. *Вывод.* С помощью локальной периодической стимуляции части нейронной сети при выборе определенных параметров стимулирующего сигнала с учетом характеристик сети возможно внешнее управление динамикой спайковой нейронной сети.

***Ключевые слова***: математическое моделирование, импульсная нейронная сеть, управление, стимуляция.

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## 1. Introduction

Control and synchronization of neuronal firing represent key issues of brain information processing [1–5]. Synchronization between brain networks underlies the implementation of various cognitive functions including learning and memory, motor control, decision making, navigation [6–10]. Distortions and inhibition of synchronous dynamics are also associated with neurodegenerative diseases [11–19]. In this context, the recovery of normal rhythmicity by external control represents an important practical problem in medicine.

One of the simplified neurobiological models to study neuronal network dynamics is realized by the so-called dissociated neuronal culture grown *in vitro* [20–23]. It represents a two-dimensional layer of growing neurons which send their axons randomly to establish synaptic connections. In spite of non-specific, e.g. random connectivity, such networks can exhibit quite patterned signals in the form of population network burst [24–26]. An important particular problem in these experiments is

to control the neuronal activity by local electrical stimuli imposed via local planar electrodes located on the bottom of the culture dish. Specifically, such stimuli sent in one site of the network can result in a synchronous response in the other area of the network or global synchronous activation of the whole culture. Interestingly, due to intrinsic mechanisms of synaptic plasticity, culture networks can adapt themselves and rearrange the connectivity, so that they can learn particular stimuli similar to the real brain [20, 23]. Here we address network firing control problem using a mathematical model in the form of a spiking neural network (SNN) simulating biological culture network in a dish. Our SNN is supplied with synaptic plasticity that can adaptively change network connectivity, hence firing patterns, in response to the control signal. Specifically, the external periodic signal can enslave the network synchronizing population bursts [27, 28]. We also use the STDP rule that can provide network spike trains very much like those observed experimentally [29].

Being significantly simplified relative to a real brain circuit, our SNN model still represents a very complicated dynamical systems with many independent parameters and different nonlinear functions. Obviously, the resulting dynamics will depend on these parameters and functions. To reduce the complexity of this model, we construct the network focusing on its functionality in the following way. First, we take the computationally effective Izhikevich model for a single neuron fixing its parameters so that each neuron plays in its excitable mode generating spike responding to incoming current (synaptic, noise, or externally applied). Next, the parameters of synaptic connectivity were selected so that in its autonomous dynamics the network can exhibit quasisynchronous bursting discharges. In our simulations, we fix this functionality. In turn, spontaneous bursts can be generated in a certain (natural) frequency range. Therefore, to investigate the control of the bursts by periodic stimulation pulse trains we take the frequency of the stimulation signal as one of the control parameters varying it relative to the natural frequency of burst discharges. Another intuitive parameter of the control is the location and the size of the stimulation area. Note, that both parameters can be easily tuned experimentally including real culture network experiments.

## 2. Methods

**2.1. Model.** Figure 1 illustrates the schematic representation of our model. Neurons are located in a two-dimensional plane and the control stimulation is applied locally to different sites with varying size of the affection area. Among many available spiking models, we chose the Izhikevich neuron as quite functional and computationally effective for network simulations [30]:

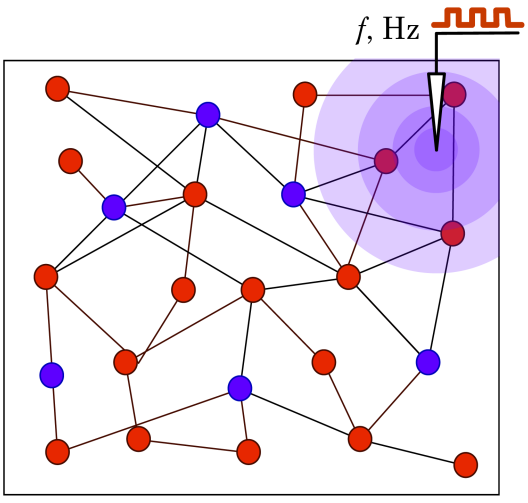


Fig. 1. Schematic representation of spiking neuron network (SNN) construction and the control strategy

$$\frac{dV}{dt} = 0.04V^2 + 5V + 140 - U + I(t), \quad (1)$$

$$\frac{dU}{dt} = a(bV - U),$$

where $V$ describes the membrane potential, $U$ is an auxiliary variable, $I$ represents the current flowing to the neuron, and $a$, $b$, $c$, $d$ are the parameters. When the potential of 30 mV is reached, the generation of a neural pulse (spike) is recorded and the values of the variables are reset:

$$\text{if } V \geqslant 30 \text{ mV}, \quad \text{then} \begin{cases} V = c \\ U = U + d. \end{cases}$$

Parameter $a$ describes the time scale of the decline of the recovery variable $U$. Lower values of the parameter lead to a slower recovery. Parameter $b$ is responsible for the sensitivity of the recovery variable $U$ to subthreshold fluctuations of the membrane potential $V$. Value $c$ is the value to which the membrane potential $V$ is reset after each spike. Parameter $d$ describes the value of post-spike resetting of the recovery variable $U$ [30].

We take the following parameter values for the Izhikevich model: $a = 0.02$, $b = 0.2$, $c = -65$, $d = 8$. They provide its excitable mode, e.g. when the stimulation is applied the neuron generates a regular pulse [30, 31].

Control signal is taken in the following form

$$\begin{aligned} I(t) &= \sum w_{ij} g_j y_{ij}(t) + I_{ex}, \\ \frac{dx_{ij}}{dt} &= \frac{z_{ij}}{\tau_{rec}} - f_{ij} x_{ij} \delta(t - (t_{sp} + \tau_{ij})), \\ \frac{dy_{ij}}{dt} &= -\frac{y_{ij}}{\tau_I} - f_{ij} x_{ij} \delta(t - (t_{sp} + \tau_{ij})), \\ \frac{dz_{ij}}{dt} &= \frac{y_{ij}}{\tau_I} - \frac{z_{ij}}{\tau_{rec}}, \\ \frac{df_{ij}}{dt} &= -\frac{f_{ij}}{\tau_{facil}} + 0.5(1 - f_{ij}) \delta(t - (t_{sp} + \tau_{ij})). \end{aligned} \tag{2}$$

Here $I_{ex}$ represents the sum of the noise current and the current coming from the stimulating electrode, $w_{ij}$ is the weight of the synaptic connection, $g_j$ is parameter staying positive for the excitatory neurons, and negative for inhibitory ones, $y_{ij}$ is the output signal from the $j$-th neuron, calculated according to Tsodyks–Markram model [32]. Variables $x_{ij}$, $y_{ij}$, and $z_{ij}$ are parts of the synapse mediator connecting neuron $j$ to neuron $i$ in the restored, active, and inactivate states respectively, $t_{sp}$ is the time of pulse generation by the presynaptic neuron $j$, $\tau_{ij}$ is the delay determined by the time for which the signal is transmitted from neuron $j$ to neuron $i$, via the axon, and proportional to the distance between $j$-th and $i$-th neurons. $\tau_I$ the time of the mediator inactivation process, $\tau_{rec}$ is the time of mediator transition from inactivated to restored state, $\tau_{facil}$ is the time of facilitation. Variable $f$ defines the part of a mediator, which is ejected at the reception of a presynaptic pulse.

Adaptive changing of the connectivity is given by STDP spike-pair rule [33, 34] as follows:

$$\begin{aligned} \frac{ds_i}{dt} &= -\frac{s_i}{\tau_i} + \sum_{t_{sp}^i} \delta(t - t_{sp}^i), \\ \frac{ds_j}{dt} &= -\frac{s_j}{\tau_j} + \sum_{t_{sp}^j} \delta(t - (t_{sp}^j + \tau_{ij})), \\ \frac{dw_{ij}}{dt} &= -F_-(w_{ij}) s_i \delta(t - (t_{sp}^j + \tau_{ij})) + F_+(w_{ij}) s_j \delta(t - t_{sp}^i). \end{aligned} \tag{3}$$

Here $s_i$ and $s_j$ are the variables that track pulses on the post-synaptic and pre-synaptic neurons respectively, $w_{ij}$ is the strength of the connection between two neurons, where $j$ is a presynaptic neuron, $i$ is a post-synaptic neuron, $\tau_i = \tau_j = 10$ ms – reduction time of local variables, $\tau_{ij}$ describes axonal delays. $t_{sp}^j$ and $t_{sp}^i$ are the spiking times on the presynapse and postsynapse respectively. The STDP functions $F(w_{ij})$ are subject to the multiplication rule [33, 34]:

$$F_+(w_{ij}) = \lambda(1 - w_{ij}), \quad F_-(w_{ij}) = \lambda \alpha w_{ij},$$

where $\lambda = 0.01$ is the learning rate, $\alpha = 5$ is the scaling parameter that determines the ratio of the processes of decreasing and increasing the synaptic strength.

For illustration, we simulated the SNN composed of 500 neurons with 400 excitatory and 100 inhibitory ones [35]. The probability of connection between the neurons was distributed normally and made dependent on the distance between the neurons. The closer the neurons are located to each other, the more likely they are connected. In other words, the network was dominated by local connections similarly to a culture network in biological experiments. Numerical experiments were conducted for networks with different average numbers of connections per neuron: $N = 15,\ 20,\ 30,\ 80$. The results were averaged for three networks with the same topological parameters. The size of the networks corresponded to the area of $1200 \times 1200$. The pulse velocity was set to $0.05$ m/s.

In the stimulation experiments, we introduce synchronization index as the average speed of transition to synchronization mode:

$$V_{\text{synchr}}(r, f) = \frac{1}{N} \sum_j 1/t_j^{\text{synchr}}(r, f), \tag{4}$$

where $r$ is the stimulation radius, $f$ is the stimulation frequency, $t_j^{\text{synchr}}$ is the time of synchronous mode onset in the $j$-th numerical experiment, $N$ is the number of experiments for networks with the same topological parameters.

**2.2. SNN dynamics.** To describe network characteristics, we computed average firing frequency and population burst frequency. The average firing frequency was first calculated (in Hz) for each neuron as follows:

$$f_i = \frac{1000N}{t}, \tag{5}$$

where $N$ is the number of spikes of the $i$st neuron during time $t$ = 50 ms. Then, the average network frequency was calculated as an arithmetic mean of all neuron frequencies:

$$\overline{f} = \frac{\sum_{i=1}^{N} f_i}{N}, \tag{6}$$

The population bursts are determined by the average frequency curve peaks with a threshold value of 5 Hz. In other words, if the firing frequency peak value is greater than 5 Hz, then the corresponding spikes train is classified as a burst. Accordingly, the burst frequency is defined as the frequency of occurrence of population bursts.

**2.3. Control signal.** Stimulation of the network can trigger force-phase locking (e.g. forced synchronization) of the population bursts relative to the stimulation pulses.

The control signal is generated by a virtual stimulator affecting neurons in the area with a variable radius from $r = 20$ to 1200 μm. The stimulation signal represents a periodic sequence of short pulses with frequency varied from 1 to 27 Hz.

Accordingly, for each value of control parameters (e.g. values of the radius and the stimulation frequency) we calculated transient time $t_j^{\text{synchr}}$ to phase-locking mode. Synchronization with phase-locking occurs when the frequency of population bursts becomes equal to the frequency of applied stimuli with certain precision (we take 0.5 Hz value). In numerical experiments we set the limit observation time to 1000 s. If no locking happens during this period, then the speed is set to zero.

## 3. Results

First, let us illustrate the SNN autonomous dynamics. A typical spontaneous firing pattern is shown in Fig. 2. The upper panel shows the raster diagram of network firing where each dot corresponds to a spike. One can note the occurrence of one burst when the spike density exceeds the 5 Hz threshold value. Network frequency calculated in the lower panel shows an irregular pattern corresponding to irregular synaptic connectivity in the network. Next, we apply the control signal with a certain frequency and stimulation radius. After some transient time that varies significantly depending on the control parameters, we observe a complete force-phase locking mode illustrated in Fig. 3. Bursts become organized in a regular periodic sequence locked with the stimulation pulses (red dashed lines in Fig. 3). Note, that spiking patterns inside each burst still stay quite different between consequent bursts. This means that the system preserves a rather high degree of freedom in local functionality while staying totally enslaved at global (network) scales.

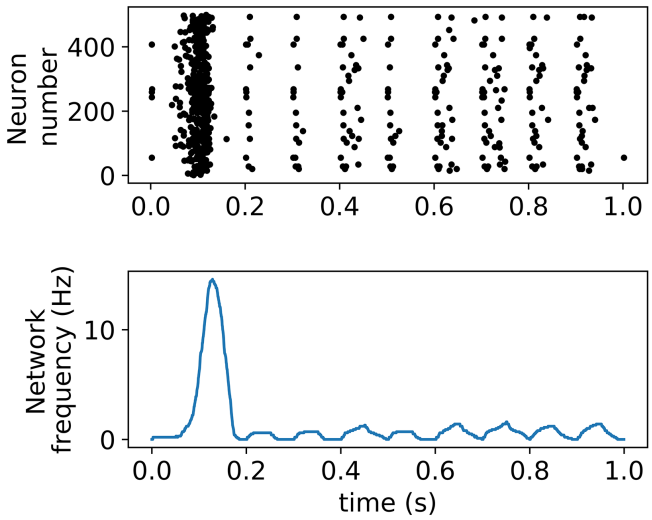


Fig. 2. Spontaneous SNN dynamics. The upper panel shows the raster plot of network firing, where each dot corresponds to a spike. The lower panel illustrates the burst frequency

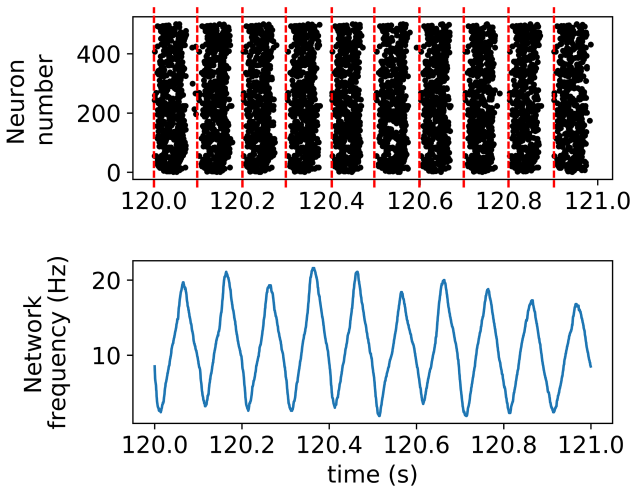


Fig. 3. Burst locking mode under control stimulation. Panels show raster plot and bursting frequency similarly to Fig. 2

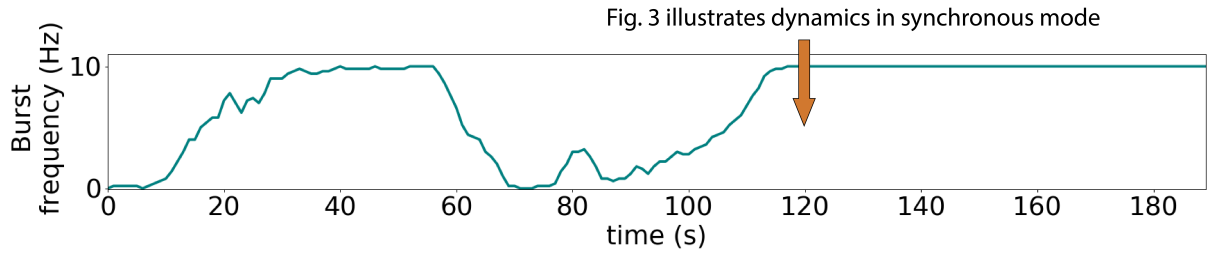


Fig. 4. Typical graph of the synchronization index. The arrow shows the moment when the bursts become locked by the control signal

In our previous studies, we tested different experimental protocols of stimulations [27, 28, 36–39]. Generally, the control was successful in certain stimulation frequency range with preferred stimulus application to be preferably in a central area. It was provided by two basic dynamical mechanisms. At first, because the SNN can generate intrinsic oscillations, e.g. spontaneous bursts, it has some "natural" frequencies defined by the time scales of local neuron dynamics (e.g. the refractory period) and by characteristic timings of synaptic plasticity. That means that the control signal should be in some range close to "natural" frequency to achieve successive locking. Next, there is the STDP mechanism that rearranges the connectivity architecture in response to the stimulation signal. This rearrangement typically reorganizes connections in some regular order providing population response to a stimulation pulse as the wave propagating from the point of stimulation. In fact, such waves indicate quasi-synchronous regularized firing of the network in response to the stimulation pulse. Consequently, it leads to successful locking. In this paper, we try to control the network by applying the stimulus in arbitrary parts of the network.

A typical scenario of burst phase locking is illustrated in Fig. 4. The SNN is stimulated by a periodic signal applied to a peripheral network area (Fig. 1) with a frequency 10 Hz. Quite a long time is required to rearrange the connectivity and to increase the burst generation frequency. However, as a result, we end up with a complete burst phase-locking mode at a control frequency 10 Hz. Different simulation trials show that there is a limited range of stimulation frequencies where network synchronization is observed. As one can expect, the larger the range, e.g. the stimulation radius $r$, the wider the frequency range for successful locking. Figure 5 illustrates the dependences of the synchronization speed on the

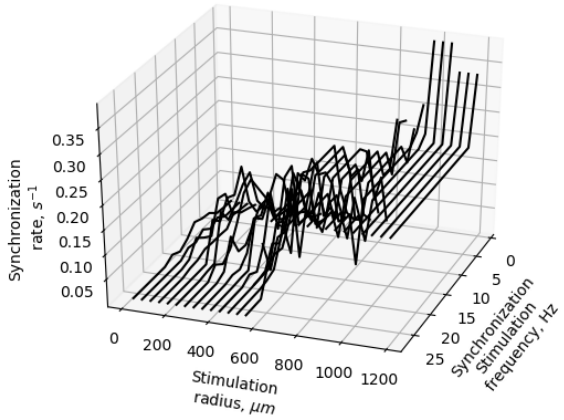


Fig. 5. Dependences of the synchronization speed on the stimulation radius for different control frequencies

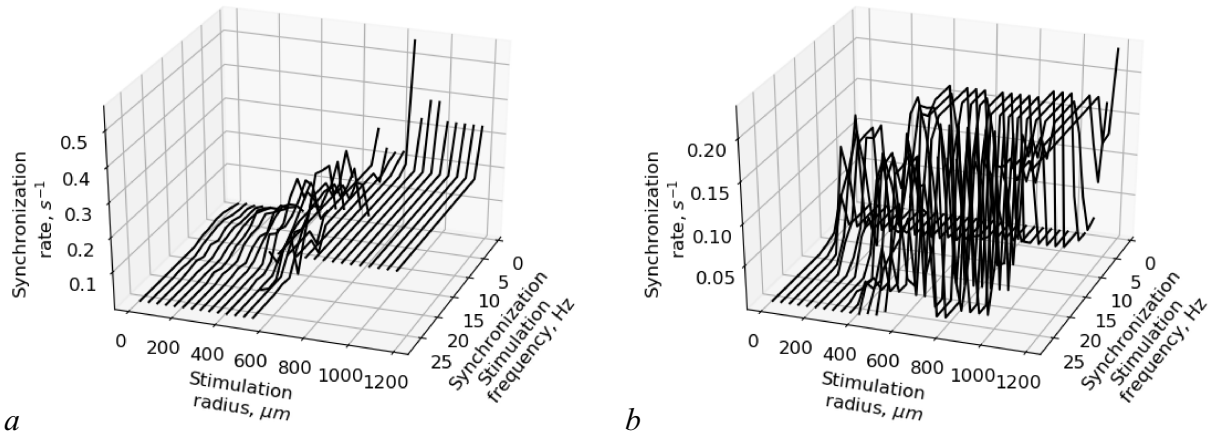


Fig. 6. Synchronization rate of population bursts in networks with different connectivity. *a* – $N = 15$ connections per neuron, *b* – $N = 80$ connections per neuron

stimulation radius for different control frequencies. Note, that number of network connections per neuron is also quite significant for the synchronization. Regarding the growing culture networks, this parameter indicates the culture age or the degree of development of the culture network. Matured cultures will have more connections and, consequently, more intensive firing patterns [25]. In accordance with these experimental facts, as the number of connections in our SNN increases, the synchronization area tends to shift to higher frequencies (Fig. 6). Note, however, that the SNN synchronization speed with high connectivity (Fig. 6, *b*) can have clusters of slow synchronization or failure in the area of sufficiently high frequencies 15–24 Hz. The frequency at which the population bursts are generated is higher than the frequency of stimulation in all cases when the synchronization failure occurs. This effect is similar to the previously described phenomena of alternating (intermittent) synchronous and turbulent activity modes or complete inability to synchronize at high neural noise values [28]. In general, it can be explained by multiple excitation loops, during which each neuron of the network is excited many times during one population burst. This creates a conflict of excitation routes without stable amplification of any of them to provide a planar response wave propagation. In experiments, more mature culture networks are characterized by increased population activity in the form of "superbursts" [25]), which may eventually imply a weakening of the ability of neural networks to be controlled externally.

## 4. Discussion

We have demonstrated how a complex neuronal network system can be controlled by a local periodic signal. This control becomes possible due to synaptic plasticity (STDP) that can rearrange network connectivity to adapt the network to the stimulation signal. An important fact here is that the application of the control can be local and is not limited to a particular place of the application. In accordance with classical synchronization theory, an oscillatory system can be phase-locked to the stimulus when the stimulation frequency is close to the natural one [40]. Besides, here the SNN has the intrinsic ability to change itself under the stimulation. Similar results have been demonstrated in recent works [41, 42]. During long-lasting (chronic) application of the stimulation, the STDP provides the adaptive rearrangement of the synaptic connections. This adaptation generally means that the initially irregular connections become ordered to be outward relative to the point of stimulus application. In terms of the SNN dynamics, it provides ordering the activity propagation from the point of control. In fact, this propagation means successive response burst generation.

We believe that possibility of STDP mediated control of complex spiking networks can have further practical applications. As we mentioned earlier, our SNN represents a quite adequate mathematical model of living culture networks *in vitro*. These networks can be used in advanced biotechnological applications of "brain-on-chip" technology design. In particular, the effect of drugs and the influence of different physical factors on the brain dynamics can be investigated using this technology. Control of the "brain-on-chip" networks by local electrical stimulation can be considered as a way to recover normal brain signal propagation and rhythmicity.

In medicine, local electrical stimulation is often applied to prevent the development of epileptic discharges and recover normal rhythmicity. We believe that our results and mechanisms of adaptive connectivity rearrangement in response to specific chronic stimulation can be useful in the design of novel robotic technologies of brain recovery based on the activation and control of network synaptic plasticity.

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