Plasticity: An incredible brain capacity

Prof. Dr. Kelly C. Iarosz
University Center of Telêmaco Borba (UNIFATEB)
State University of Ponta Grossa (PPGFIS)

São Paulo
2024
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Brain

The central organ of the human nervous system. It controls most of the activities of the body, processing, integrating, and coordinating the information it receives from the sense organs, and making decisions as to the instructions sent to the rest of the body.
Neuroplasticity or brain plasticity is defined as the ability of the nervous system to change its activity in response to intrinsic or extrinsic stimuli by reorganizing its structure, functions, or connections. A fundamental property of neurons is their ability to modify the strength and efficacy of synaptic transmission through a diverse number of activity-dependent mechanisms, typically referred to as synaptic plasticity.
**Neuron**

Dendrites: receive or transmit information from/to other neuronal cells.

Axon: transmission of nerve impulses.

Within a nervous system, a neuron or nerve cell is an electrically excitable cell that fires electric signals called action potentials across a neural network. Neurons communicate with other cells via synapses, which are specialized connections that commonly use minute amounts of chemical neurotransmitters to pass the electric signal from the presynaptic neuron to the target cell through the synaptic gap.

https://doi.org/10.1590/S1806-111721787
Rev. Bras. Ens. Fis. 37 (2) • Jun 2015
Neuron

Cell Body - Cortex

Axon – white matter

https://anatpat.unicamp.br/bineucerebrocoronalindice.html
Neuron

Dendrites: receive or transmit information from/to other neuronal cells.

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Chemical synapses
- Neurotransmitters

Electrical synapses
- Electrical impulses and communication junctions
Time line

Plasticity

- DW. James
  - Start
  - 1890

- S. Ranón y Cajal
  - Neuronal architecture
  - Modifications according to stimuli
  - 1894

- E. Lugano
  - Mental associations occur through new communications between neurons
  - 1898
J. Konorski
Conditioned reflex occurs from changes in neuronal synapses.

1948

Pavlov Museum, Russia.
D. Hebb
Synapses used more frequently are strengthened as a result of the physiological adaptation of the neurons involved.

1949

M. Merzenich
Adults plasticity

1983
Plasticity

Brain plasticity happens all the time

Recovery from injuries and microinjuries

Acquisition of new skills

Sensory deprivation

Response to external stimuli

Hebbian Theory


https://pure.mpg.de/rest/items/item_2346268_3/component/file_2346267/content
Plasticity

Synapses

**Receptors and neurotransmitters: number modified**
- Reduction of receptors - synaptic weakening
- Increase in receptors - synaptic strengthening
- Excitatory (eSTDP) and inhibitory synapses (iSTDP)

**Structure modification**
- The same number of connections
- Rearrangement in synapses
- Synapse/dendrites sprouting
- Synapse/dendrites elimination
- Migration and neurogenesis

Long Term Potentiation (LTP)

Long Term Depression (LTD)

Synaptic rearrangements
Evolution work

Brain Dynamic Behavior
Evolution work

Brain Dynamic Behavior
Our main goal is to show that spike timing-dependent plasticity of excitatory and inhibitory synapses induces non-trivial topologies in the plastic brain.

Initial networks of neurons fully connected, evolve to a non-trivial complex network.

Consequently, this non-trivial topology alters the synchronous behavior.

We have considered: Initial network with a global coupling, with chemical synapses where the connections are unidirectional, and the local dynamics is described by the Hodgkin–Huxley model.
Model Hodgkin-Huxley

\[
\begin{align*}
C\dot{V}_i &= I_i - g_K n_i^4 (V_i - E_K) - g_Na m_i^3 h_i (V_i - E_Na) \\
&\quad - g_l (V_i - E_L) + \frac{(V_i^{\text{Exc}} - V_i)}{\alpha^{\text{Exc}}} \sum_{j=1}^{N_{\text{Exc}}} \varepsilon_{ij} S_j \\
&\quad + \frac{(V_i^{\text{Inhib}} - V_i)}{\omega^{\text{Inhib}}} \sum_{j=1}^{N_{\text{Inhib}}} \sigma_{ij} S_j + I_i, \\
\dot{n}_i &= \alpha_{n_i} (V_i) (1 - n_i) - \beta_{n_i} (V_i) n_i, \\
\dot{m}_i &= \alpha_{m_i} (V_i) (1 - m_i) - \beta_{m_i} (V_i) m_i, \\
\dot{h}_i &= \alpha_{h_i} (V_i) (1 - h_i) - \beta_{h_i} (V_i) h_i,
\end{align*}
\]
The excitatory eSTDP is given by

\[
\Delta \varepsilon_{ij} = \begin{cases} 
A_1 \exp(-\Delta t_{ij}/\tau_1), & \Delta t_{ij} \geq 0 \\
-A_2 \exp(\Delta t_{ij}/\tau_2), & \Delta t_{ij} < 0,
\end{cases}
\]

\[
\Delta t_{ij} = t_i - t_j = t_{pos} - t_{pre}.
\]

Fig. 1(a): result obtained for \(A_1 = 1.0, A_2 = 0.5, \tau_1 = 1.8 \text{ ms}, \) and \(\tau_2 = 6.0 \text{ ms}.\)

The green dashed line: denotes the \(\Delta t_{ij}\) value at which the curves of potentiation and depression intersect.

The inset in Fig. 1(a) shows that for \(|\Delta t_{ij}| < 1.8 \text{ ms}\) the potentiation of \(\varepsilon_{ij}\) is bigger than the depression.

iSTDP (inset in Fig. 1(b)) the potentiation of \(\sigma_{ij}\) is bigger than the depression for \(|\Delta t_{ij}| > 9.8 \text{ ms}.\)

\[
\Delta \sigma_{ij} = \frac{g_0}{g_{norm}} \alpha^\delta |\Delta t_{ij}| \Delta t_{ij}^{\delta-1} \exp(-\alpha |\Delta t_{ij}|),
\]

\(g_0\) - scaling factor accounting for the amount of change in inhibitory conductance induced by the synaptic

\(g_{norm} = \beta \exp(-\beta)\) is the normalizing constant.

Fig. 1(b) exhibits the result obtained from Eq. (14)

As a consequence, \(1\sigma_{ij} > 0\) for \(1t_{ij} > 0,\) and \(1\sigma_{ij} < 0\) for \(\Delta t_{ij} < 0.\) The initial inhibitory synaptic weights \(\sigma_{ij}\) are normally distributed with mean and standard deviation equal to \(\sigma_M.\)

Then, the coupling strengths are updated according to Eq. (14), where \(\sigma_{ij} \rightarrow \sigma_{ij} + 10-31\sigma_{ij}.\) The updates for \(\varepsilon_{ij}\) and \(\sigma_{ij}\) are applied for the last postsynaptic spike.
Fig. 2(a) shows the mean order parameter (R) that is calculated for different initial conditions.

Function of the inhibitory coupling strength $\sigma_M$ for a neural network with excitatory and inhibitory synapses. Case without STDP (black circles) and STDP (red triangles).

For $\epsilon_M$ equal to 0.25 and varying $\sigma_M$, we do not observe a significant alteration of the R value without STDP - initially the network has an all-to-all topology.

Increase of $\sigma_M$ and present a large standard deviation. This standard deviation occurs due to the existence of different synchronization states.

The upper border of the inhibitory coupling $2\sigma$ and the different initial conditions are important to change the dynamics of the network with STDP and without external perturbation.

This is verified by means of the decay of the R values and the large standard deviation bar.
In Fig. 2(b) and (c): $\sigma_M = 0.675$, for different configurations of the initial networks and $\tau = 100$ ms.

The black line shows the case in which the network goes to a desynchronized state ($R \approx 0.1$), whereas the red line exhibits the case of a network that presents synchronous behavior ($R \approx 1$).

In both cases, we consider the same parameters, except the seed to generate the random distribution of the constant current density $I_j$.

Through Fig. 2(b) and (c) it is possible to verify why and when the coupling matrix suffer substantial changes. The transition occurs when the black or red curves cross the green line.

At this time, depreciation induces weak strength in the coupling matrix, and potentiation induces strong strength.
Fig. 3 exhibits the time courses of the mean excitatory (Fig. 3(a)) and inhibitory (Fig. 3(b)) coupling strengths from the multiple coexisting regimes that are shown in Fig. 2(a).

We see that for $\sigma_M = 0.25$ both $\epsilon_{ij}$ and $\sigma_{ij}$ have constant values for the time approximately greater than 700 s, and the learning produces a triangular-type connecting matrix (as shown in Fig. 4), meaning that the connections among all neurons become preferentially directed.

For $\sigma_M = 0.5$ the $\epsilon_{ij}$ values decrease to approximately 0.15, while $\sigma_{ij}$ values oscillate about 0.25, and the coupling matrix becomes partitioned, indicating the existence of larger clusters.

Increasing the upper border $\sigma_M$ to 0.75 both $\epsilon_{ij}$ and $\sigma_{ij}$ tend to 0, and the coupling matrix becomes sparse.
The synaptic weights are suppressed in the desynchronized regime (Fig. 4(a)), coupling matrix presents a small number of connections. This behavior: black lines in Fig. 2(b) and (c).

In addition, the synaptic weights are potentiated (red lines in Fig. 2(b) and (c)) in the synchronized regime (Fig. 4(b)), and the coupling matrix exhibits a triangular shape.

The synchronous behavior has a dependence on the direction of synapses. When the presynaptic neurons are excitatory the synapses from the high frequency to the low frequency neurons become stronger.

Presynaptic neurons are inhibitory, the synapses from the low frequency to the high frequency neurons become stronger.

Fig. 4 shows the final topologies for two networks initially set with a global coupling topologies after being evolved by a STDP process.

We see that the STDP induces a non-trivial topology in the network resulting in networks sparsely connected, moderately connected (Fig. 4(a)), or densely connected with strong preferential attachment (Fig. 4(b)).
Considering an external perturbation ($\Gamma_i > 0$), we also study the cases without and with plasticity. In the case without STDP, we verify that the mean order-parameter has a small decay when $\sigma_M$ increases, as shown in Fig. 5(a) with black circles.

The red triangles represent the case with STDP, and unlike the case without perturbation (Fig. 2(a)), there is an abrupt transition (blue triangles), due to a first-order transition in the average order parameter.

The upper border of the inhibitory coupling is relevant to produce alteration in the dynamics, while the different initial conditions are important only at the transition.

Based on the results in the inset (Fig. 5(a)), we verify that the network in the transition can be either in one of the states: (i) high $R$ with potentiation of the average-time difference for excitatory and inhibitory connections (red lines in Fig. 5(b) and (c)), or (ii) low $R$ with excitatory average time-difference in the depression region and inhibitory in the potentiation region (black lines).
The transition from the synchronized to the desynchronized states was reported in studies on how stimulation impact on neurological disorders induced by an abnormal neuronal synchronization (Popovych & Tass, 2012; Tass & Majtanik, 2006).

A first order transition was also observed in Popovych et al. (2013) when the stimulation intensity varies in a neural network with eSTDP.

In our simulations, we observe the transition to desynchronization caused by a variation in the inhibitory coupling in neural networks with both eSTDP and iSTDP.
Fig. 6 illustrates the coupling matrix for the two states of the first-order transition.

In Fig. 6(a), we can see the coupling configuration that corresponds to high R.

The network presents high connectivity, and for this reason it is possible to observe synchronous behavior.

For the case of low R, we verify that the network has only connections from neurons belonging to the inhibitory population to any other neuron, as shown in Fig. 6(b).
Spike timing-dependent plasticity induces non-trivial topology in the brain

In our results, we have observed for some parameter conditions not only the improvement of neural spiking synchronization, but also for other parameter conditions that promote desynchronization.

The onset of synchronicity comes along side with desynchronicity in the plastic brain. This balance between different synchronous behaviors is vital to maintain a fundamental property of a brain network.

Clusters need to be sufficiently synchronous for information to be efficiently exchanged, but at the same time sufficiently desynchronous to behave independently.

Finally, we show that when there is an external perturbation, the plastic neural network has an abrupt change in behavior characterized by a first-order transition.
In conclusion, we have studied the effects of spike timing dependent plasticity on the synchronous behavior and the evolved connecting topology of neural networks constructed with Hodgkin–Huxley neurons.

Regarding the evolved topology, our main conclusion is that learning under a STDP results in evolved networks that present complex topology.

Concerning the dynamic synchronous behavior of the evolved networks, we observe that the studied networks exhibit concurrent synchronous and non-synchronous states with characteristics that depend on both the upper border of the inhibitory coupling and the initial conditions.

Specifically, we verify that the main role of the inhibitory connections is to produce a delay in the spiking time of the postsynaptic neurons.
Spike timing-dependent plasticity induces non-trivial topology in the brain

As a consequence, the increase of the inhibitory coupling strength can suppress synchronous behavior, which contributes to a decrease in the mean order parameter.

Moreover, the transition from low to a high synchronous state is smooth by alterations of the inhibitory synapses.

When a random external perturbation is introduced in the network, this transition becomes discontinuous, i.e., we observe a first-order transition.

Similarly to the non-perturbed network, we also find coexistence of synchronous and non-synchronous neurons in the perturbed networks.
ACKNOWLEDGMENTS

University Center of Telêmaco Borba (UNIFATEB)
State University of Ponta Grossa (PPGFIS)
105 Group Science
CNPq
FAPESP
CAPES

email: kiarosz@gmail.com