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Inhibitory neurons control the consolidation of neural assemblies via adaptation to selective stimuli

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Context

Brain circuits display **modular architecture** at different scales of organization [6]. Such neural assemblies are typically associated to functional specialization, but the mechanisms leading to their emergence and consolidation still remain elusive. In particular, it has been shown that **inhibitory** plasticity has a decisive impact on the dynamics and adaptation capacity of the brain, notably in the **long-term maintenance of memory** [4].

Although many studies using **coupled oscillator** models attempt to simulate this process, few consider the case of **distinct inhibitory and excitatory units** having an **adaptation mechanism** on their synaptic weights and thus impacting the network state.

Therefore, this study [1] investigates the impact of **inhibition** on the modulation of **synchronization** and on the creation and consolidation of **modular structures** in **coupled oscillator networks** subject to **adaptation**. These results are then extended to **spiking neural networks** with STDP.

Model

Network of N θ -neurons all-to-all connected [2]

$$\frac{d\theta_i}{dt} = (1 - \cos(\theta_i)) + (1 + \cos(\theta_i)) \left[\eta_i + \frac{g}{N} \left(\sum_{j=1}^N \kappa_{ij} \sin(\theta_j - \theta_i) \right) + I_i(t) + \xi_i(t) \right]$$

With the phase $\theta_i \in [-\pi, \pi]$, the excitability parameter η_i , the global coupling g , κ_{ij} the coupling weight from pre-synaptic neuron j to post-synaptic neuron i , the external input $I_i(t)$ and the additive Gaussian noise $\xi_i(t)$.

Weights adaptation

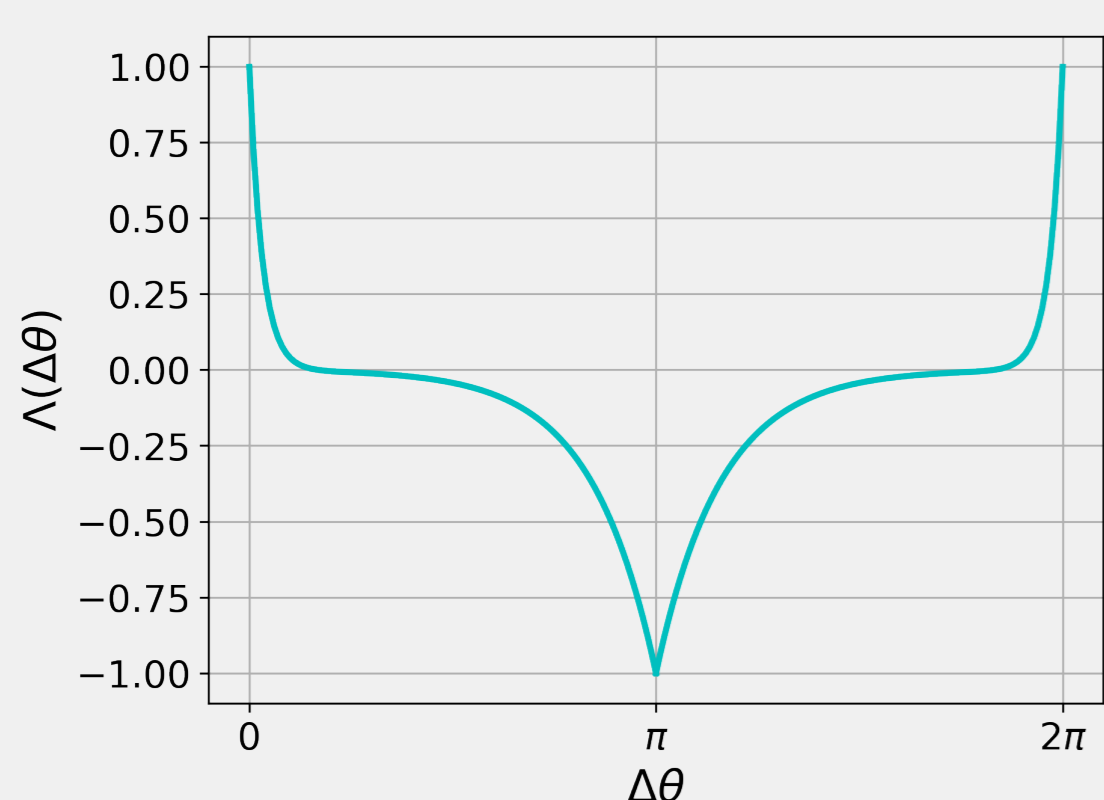
$$\frac{d\kappa_{ij}}{dt} = [\epsilon_1 + \epsilon_2 H(I_j(t))] \kappa_{ij} (1 - \kappa_{ij}) \Lambda(\Delta\theta)$$

$$\frac{d\kappa_{ij}}{dt} = \epsilon_1 \kappa_{ij} (1 - \kappa_{ij}) \Lambda(\Delta\theta)$$

$$\frac{d\kappa_{ij}}{dt} = \epsilon_1 (\kappa_{ij} + 1) (-\kappa_{ij}) \Lambda(\Delta\theta)$$

With $\epsilon_1 \ll \epsilon_2 \ll 1$ the learning rates for slow and fast adaptations and $H(x)$ the Heaviside function.

Symmetric phase difference-dependent plasticity rule [3, 5]



$$\Lambda(\Delta\theta) = \begin{cases} e^{-\frac{\Delta\theta}{0.1}} - e^{-\frac{\Delta\theta-\pi}{0.5}}, & \text{if } 0 \leq \Delta\theta < \pi \\ e^{\frac{\Delta\theta-2\pi}{0.1}} - e^{-\frac{\Delta\theta-\pi}{0.5}}, & \text{if } \pi \leq \Delta\theta < 2\pi \end{cases}$$

$$\text{with } \Delta\theta = |\theta_j - \theta_i|$$

Figure 1. Hebbian plasticity function with a larger time window for depression than for potentiation.

Discussion

θ -neurons

- The creation of **clusters** is induced by the **spatio-temporal correlations** of the stimuli applied to the network and by the resulting **adaptation** mechanism, thus maintaining a certain **synchrony** within them.
- Networks made of **purely excitatory** neurons or of neurons **not respecting Dale's principle** are **unable to maintain** the modular architecture induced by the entrained stimuli.
- By preserving a specific dynamics to each cluster, **inhibitory** neurons allow to **maintain and consolidate** the learned structure over the **long-term** \rightarrow memory consolidation.

Spiking neurons

- Feed-back and feed-forward **inhibition** favour the **formation** of the modular structures and their **spontaneous recall** during a resting state of asynchronous irregular dynamics.
- Spontaneous recall** of the learned items seems to promote their **maintenance and consolidation** over the **long-term** \rightarrow memory consolidation.

Creating and maintaining modular structures

- Learning of **2 temporally alternating stimuli** applied to separate areas of the network.
- We observe the evolution of the network at rest over the **short and long term** in networks of: (1) **excitatory and inhibitory** neurons, (2) with **only excitatory** neurons, and (3) with **unlabelled** neurons (both excitatory and inhibitory).
- Without distinct inhibitory units, the 2 clusters created **synchronize** at rest and consequently are **forgotten** in the long term.
- The presence of **inhibition** allows the **structure to consolidate**, where each inhibitory neuron associates with a cluster.

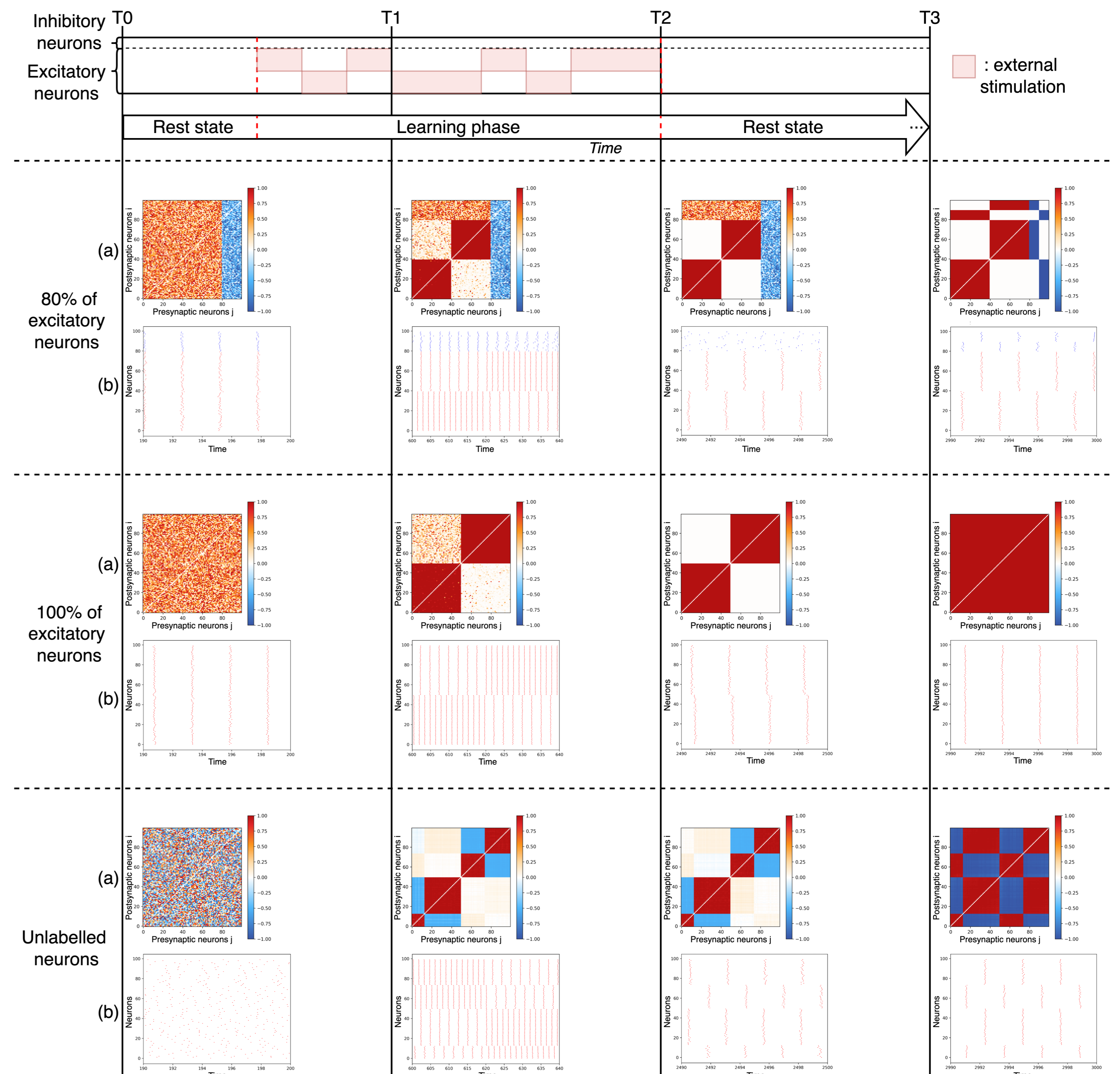


Figure 2. Experiment with 2 clusters using θ -neurons. Graphs (a) represent the weight matrices at times T0, T1, T2 and T3: the color denotes if the connection is excitatory (red) or inhibitory (blue) or absent (white). Graphs (b) are raster plots at times T0, T1, T2 and T3, displaying the firing times of excitatory (red dots) and inhibitory (blue dots) neurons. Note that the inhibitory neurons are sorted by phases at time T3 in our model to better visualize clusters, otherwise the associations with each group are random. The neurons are also sorted by phases in each cluster for the last model.

Extension to spiking neurons

- Network of **quadratic integrate and fire (QIF)** neurons [2] subject to **spike-timing-dependent plasticity**.
- Similar protocol with learning of **2 temporally alternating stimuli** applied to separate groups of excitatory and inhibitory neurons.
- At rest, we observe **asynchronous irregular dynamics**, with random epochs of **spontaneous recall** of the two learned items.

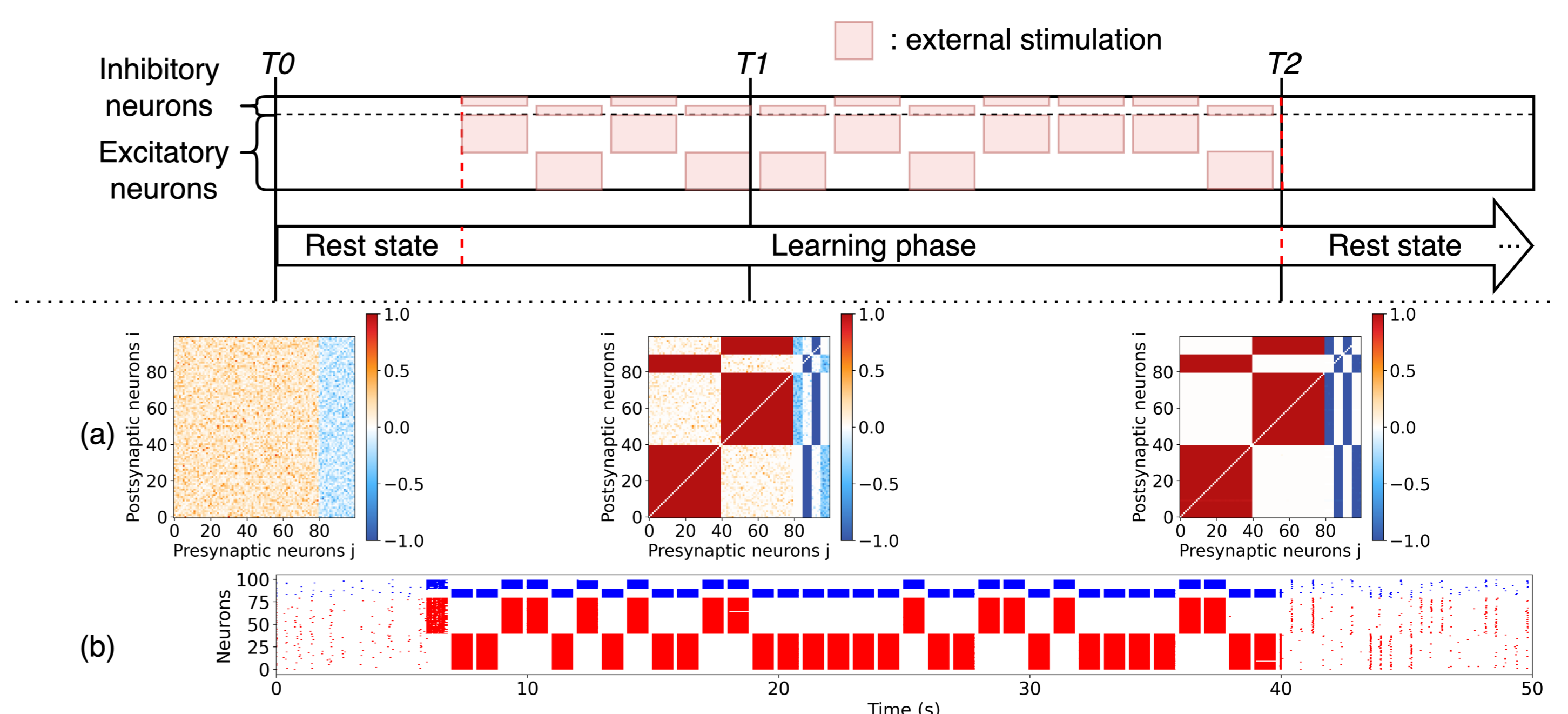


Figure 3. Experiment with 2 clusters using QIF neurons. Graphs (a) represent the weight matrices at times T0, T1 and T2: the color denotes if the connection is excitatory (red) or inhibitory (blue) or absent (white). Graph (b) is the raster plot displaying the firing times of excitatory (red dots) and inhibitory (blue dots) neurons.

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