# Neural Fields and Cortical Plasticity

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## Introduction

The aim of this work is the study of self-organization and reorganization of the brain cortex using neural fields as modeling framework. The main hypothesis of this work is that the model needs to adapt only the afferent (feed-forward) connections while its lateral connections have been considered fixed and as a way to serve competition between neurons such that after the competition stage, only a small group of neurons tunes itself toward the presented stimulus. In addition some cases of the brain cortex reorganization in the presence of cortical lesions and sensory deprivation (i.e the sensory input to a specific cortical representation is eradicated) have been taken also into consideration.

## Materials & Methods

### Neural Fields

We use a modified version of Amari's neural field equation, which was introduced firstly in [2], and is given below:

$$\tau \frac{\partial V(\mathbf{x},t)}{\partial t} = -\tau V(\mathbf{x},t) + 1 - |W_F(\mathbf{x}) - I(t)| + \int_\Omega W_L(|\mathbf{x}-\mathbf{y}|) f(V(\mathbf{y},t)) d\mathbf{y} \quad (1)$$

where, $V(\mathbf{x},t)$ is the local activity of a population of neurons, located at position $\mathbf{x}$ at time $t$, $\tau$ is the temporal decay constant of the synapse. $W_F(\mathbf{x})$ is the feed-forward weights which modulate the input, $I(\mathbf{x})$ for each position $\mathbf{x}$, $W_L(\mathbf{x})$ is the strength of connections between neurons, according to $W_L(\mathbf{x}) = W_e(\mathbf{x}) - W_i(\mathbf{x})$ where, $W_e(x)$ and $W_i(x)$ is the excitatory and inhibitory kernel, respectively,

$$W_e(x) = K_e e^{-\frac{x^2}{2\sigma_e^2}} \text{ and } W_i(x) = K_i e^{-\frac{x^2}{2\sigma_i^2}} \quad (2)$$

$f(\mathbf{x})$ is the firing rate function of a single neuron,

$$f(x) = \begin{cases} x, & \text{if } x > 0 \\ 0, & \text{if } x \leq 0 \end{cases} \quad (3)$$

We consider, also, that $\Omega$ is a compact subset of $\mathbb{R}^m, m = 1, 2$. Additionally, the field is generally considered to be homogeneous using an isotropic kernel of the form $W(|\mathbf{x}-\mathbf{y}|)$.

### Learning Rule

We use a Hebbian-like learning rule which adapts only the afferent (feed-forward) connections. The learning rule is given by the following equation:

$$\frac{\partial W_F(\mathbf{x},t)}{\partial t} = \gamma L_e(\mathbf{x},t)\,(I(\mathbf{x},t) - W_F(\mathbf{x},t)) \quad (4)$$

where $\gamma$ is the learning rate and $L_e(\mathbf{x},t)$ is the total excitation received at the point $\mathbf{x}$ which is given by the two dimensional spatial convolution between the excitatory part of the kernel function and the field activity. Therefore, we have:

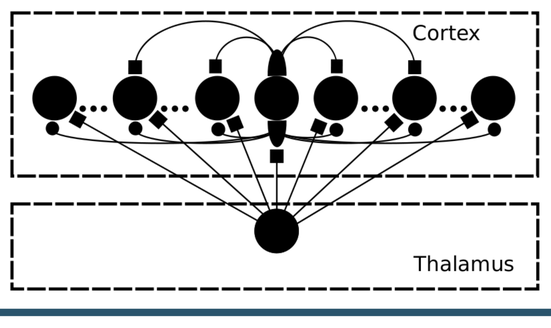
$$L_e(\mathbf{x},t) = \int_\Omega f(V(\mathbf{y},t)) W_e(|\mathbf{x}-\mathbf{y}|,t) d\mathbf{y} \quad (5)$$

### Experimental Setup

In all following simulations, we consider the domain $\Omega$ is discretized into $64 \times 64$ spatial elements with Dirichlet boundary conditions such that $V(\mathbf{x},t) = 0, \forall x \in \partial\Omega$. A realization of the network in 1D is illustrated in figure 1. Equation parameters are given by the table below. Input to the network is drawn from a set $S_k$ which is a set of samples generated by drawing $k$ evenly spaced values in the interval $[0,1]$. More precisely, $S_k = \{x \in \mathbb{R} | x = \frac{k}{n-1}, 1 < n \leq k \text{ and } n \in \mathbb{N}\}$. A special case of the input is the $S_\infty$, whose values are drawn uniformly from the interval $[0,1]$.

| Case | $K_e$ | $K_i$ | $\sigma_e$ | $\sigma_i$ | $\gamma$ |
|---|---|---|---|---|---|
| 2D | 1.5 | 0.75 | 0.1 | 1.0 | 0.01 |

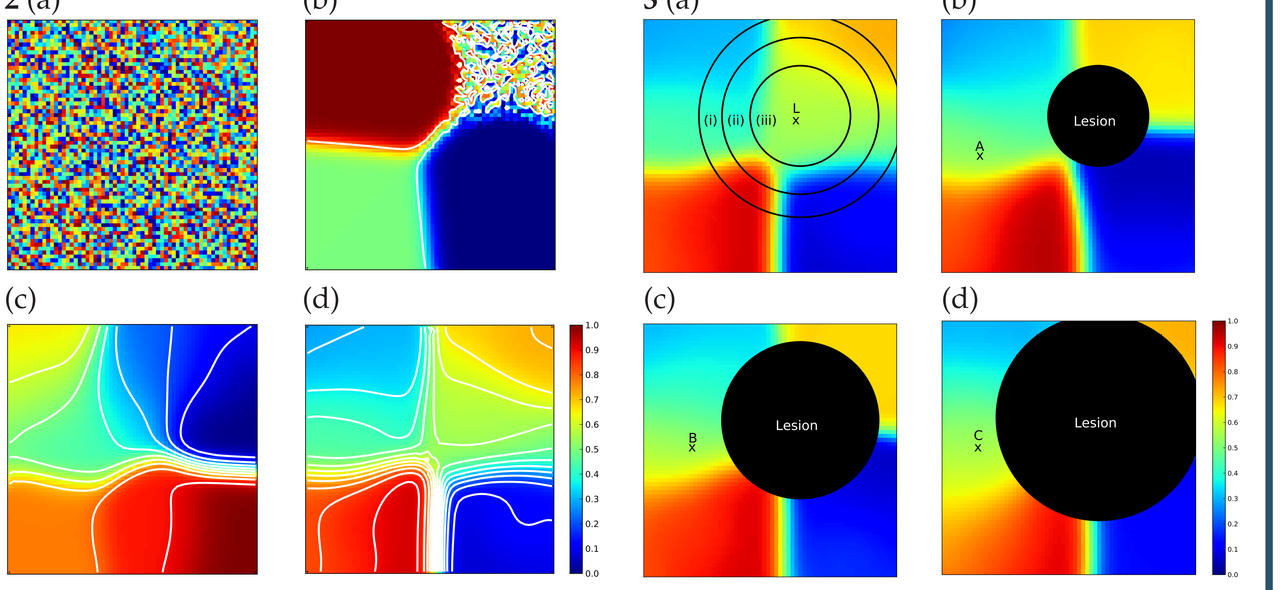
**1**




## Results

The model was trained over 5000 presented samples drawing from the input data sets $S_k$ for (2b) $k = 3$, (5a) $k = 5$, (2c) $k = 10$ and (2d) $S_\infty$. The model converged and a consistent topographic map was emerged. Hence, the model is capable of self-organization.

**2** (a) (b) (c) (d)

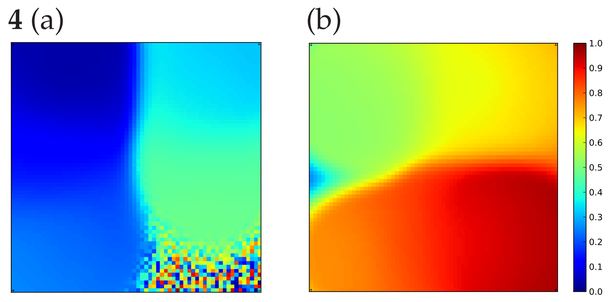


**3** (a) (b) (c) (d)



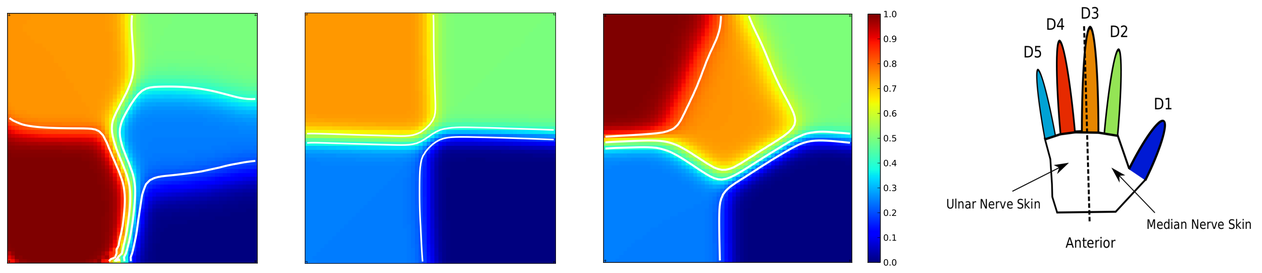

Moreover, the model is able to retain the ability of learning during its entire lifetime, which means that the model once it learns the input data is not frozen and can learn new input data. The results are depicted in figure 4, where the model was trained, initially, (a) over values drawn uniformly from the interval $[0, \frac{1}{2}]$ and after its convergence, (b) it was retrained over values from the interval $[\frac{1}{2}, 1]$.

**4** (a) (b)



In order to study lesion cases of brain cortex, we applied on the $S_\infty$-map (figure 2d) a lesion using a mask of different size, as it is depicted in figure 3a. Then we retrained the model over the same input data set. Thus, after the convergence of the model a new topographic map was revealed. This result is in accordance with the neurophysiological data, which have been shown that in the presence of a cortical lesion, neighboring areas of the lesioned area take over and the brain cortex can recover some of its previous functions (figure 3b, 3c, 3d).

**5** (a) (b) (c) (d)




Similarly, in the case of the amputation we used the $S_5$ input set in order to train the model. Once the model has converged (figure 5a) we made the assumption that the five colors indicate the representational areas of the hand (figure 5d). Then we retrained the model using the $S_5^d = S_5 \backslash \{1\}$ input set, regarding the sensory deprivation which takes place in the case of an amputation (digit D4). As one can observe, a new topographic map was emerged and the representation of the value 1 is dissapeared, figure 5b. Mamalian brain cortex has the ability to expand the intact representations to the deprived ones. Moreover, the mamalian brain cortex can also reshape the previously deprived representations if the amputated body part is reconstructed, figure 5c.

## Conclusions

The computational model of cortical plasticity enhances the role of afferent synapses from thalamus to the brain cortex onto self-organization and reorganization during the entire lifetime of the mammalian brain cortex, in accordance with neurophysiological data (lesions [5], and sensory deprivation [4], [3] has been taken into consideration). Nevertheless, a balance between excitatory and inhibitory connections is required in order to stabilize the model. In addition, the model explains the canonical connectivity pattern [1] of excitatory connections and the almost canonical connectivity of the inhibitory connections. Consequently, the model is distributed, unsupervised, plastic and robust, something which enables the model to be used as a computational framework of the brain cortex self-organization and reorganization.

## References

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