# Intrinsically Regulated Self-Organization of Topologically Ordered Neural Maps

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# Intrinsically Regulated Self-Organization of Topologically Ordered Neural Maps

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**Abstract**

Dynamic field theory models the spatio-temporal evolution of activity within the cortex and has been successfully applied in various domains. However, the development of dynamic neural fields (DNFs) is only rarely explored. This is due to the fact that DNFs are sensible to the right balance between excitation and inhibition within the fields. Small changes to this balance will result in runaway excitation or quiescence. Consequently, learning most often focuses on the synaptic weights of projections to the DNF, thereby adapting the input-driven dynamics, but leaving the self-driven dynamics unchanged. Here we present a recurrent neural network model composed of excitatory and inhibitory units which overcomes these problems. Our approach differs insofar as we do not make any assumption on the connectivity of the field. In other words, synaptic weights of both, afferent projections to the field as well as lateral connections within the field, undergo Hebbian plasticity. As a direct consequence our model has to self-regulate in order to maintain a stable operation mode even in face of these experience-driven changes. We therefore incorporate recent advances in the understanding of such homeostatic processes. Firstly, we model the activity-dependent release of the neurotrophine BDNF (brain-derived neurotrophic factor) which is thought to underlie homeostatic synaptic scaling. BDNF has opposing effects on the scaling of excitatory synapses on pyramidal neurons and interneurons, thereby mediating a dynamic adjustment in the excitatory-inhibitory balance. Secondly, we adapt the intrinsic excitability of the model units by adjusting their resting potentials. In both processes the objective function of each neuron is to achieve some target firing rate. We experimentally show how homeostasis in form of such locally operating processes contributes to the global stability of the field. Due to the self-regulatory nature of our model, the number of free parameters reduces to a minimum which eases its use for applications in various domains. It is particularly suited for modeling cortical development, since the process of learning the mapping is self-organizing, intrinsically regulated, and only depends on the statistics of the input patterns. Self-organizing maps usually develop a topologically ordered representation by making use of distance-dependent lateral connections (e.g. Mexican Hat connectivity). Since our model does not rely on such an assumption, the learned mappings do not necessarily have to be topology preserving. In order to counteract this problem we propose to incorporate an additional process which aims at the minimization of the wiring length between the model units. This process relies on a purely local objective and runs in parallel to the above mentioned self-regulation. Our experiments confirm that this additional mechanism leads to a significant decrease in topological defects and further enhances the quality of the learned mappings.

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The structure of the recurrent neural network is shown in Fig. 1. It is composed of excitatory units E and inhibitory units I, both initially being arranged on a 2-dimensional grid mimicking the neural tissue.

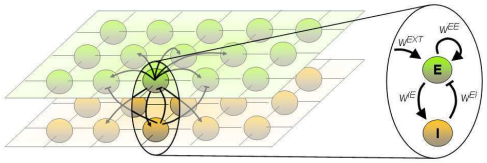


Fig. 1. The structure of the recurrent neural network.

The membrane potentials of the E- and I-cells are denoted by the variables $u$ and $v$, respectively. We use index $i$ for referring to the unit located at position $x_i$ of the cortical sheet. The spatio-temporal evolution of the activity in the field is modeled by the following differential equations:

$$\tau_E \frac{du_i}{dt} = -u_i + \sum_j g(d_{ij}) \cdot w_{ij}^{EE} \cdot f(u_j) - \sum_j w_{ij}^{EI} \cdot f(v_j) + \sum_j w_{ij}^{EXT} \cdot s_j + h_i^E \quad (1)$$

$$\tau_I \frac{dv_i}{dt} = -v_i + \sum_j g(d_{ij}) \cdot w_{ij}^{IE} \cdot f(u_j) + h^I \quad (2)$$

Here, $\tau_E$ and $\tau_I$ are time constants, $h_i^E$ and $h^I$ are the resting potentials, $s_j$ is an external input, and $w_{ij}^*$ denotes the synaptic weight of a connection from unit $j$ to unit $i$ where $* \in \{EE, EI, IE, EXT\}$ specifies the type of connection. A monotonically increasing transfer function is denoted by $f$. Furthermore, $g$ is a function which modulates synaptic efficiency depending on the distance $d_{ij}$ between the pre- and postsynaptic cells.

In the course of learning we incorporate homeostatic synaptic scaling as follows:

$$w_{ij}^{EXT}(k) = \frac{w_{ij}^{EXT}(k-1) + \alpha \cdot \Delta\widetilde{w}_{ij}^{EXT}(k)}{BDNF_i^E(k) \cdot BDNF_j^{EXT}(k)} \quad (3)$$

$$w_{ij}^{EE}(k) = \frac{w_{ij}^{EE}(k-1) + \alpha \cdot \Delta\widetilde{w}_{ij}^{EE}(k)}{BDNF_i^E(k) \cdot BDNF_j^E(k)} \quad (4)$$

$$w_{ij}^{EI}(k) = \left[w_{ij}^{EI}(k-1) + \alpha \cdot \Delta\widetilde{w}_{ij}^{EI}(k)\right] \cdot BDNF_i^E(k) \quad (5)$$

$$w_{ij}^{IE}(k) = \left[w_{ij}^{IE}(k-1) + \alpha \cdot \Delta\widetilde{w}_{ij}^{IE}(k)\right] \cdot BDNF_j^E(k) \quad (6)$$

Thereby, $\Delta\widetilde{w}_{ij}^*(k)$ denotes the weight change according to Hebbian plasticity, $\alpha$ the learning rate, and $BDNF_i^E(k)$ the level of BDNF released by the $i$-th E-cell. We model the activity-dependent release of BDNF according to (7)-(8), where $A_i(k)$, $\bar{A}_i(k)$, and $\widehat{A}$ are the instantaneous, mean, and target firing rates, respectively.

$$\bar{A}_i(k) = (1 - \frac{1}{\tau_H}) \cdot \bar{A}_i(k-1) + \frac{1}{\tau_H} \cdot A_i(k) \quad (7)$$

$$BDNF_i^E(k) = 1 + \beta_H \left( \frac{\bar{A}_i^E(k-1) - \widehat{A}}{\widehat{A}} \right) \quad (8)$$

We further adapt the resting potentials of the E-cells by which the intrinsic excitability of them becomes changed:

$$h_i^E(k) = h_i^E(k-1) + \beta_T \cdot \left( \frac{\widehat{A} - \bar{A}_i^E(k-1)}{\widehat{A}} \right) \quad (9)$$

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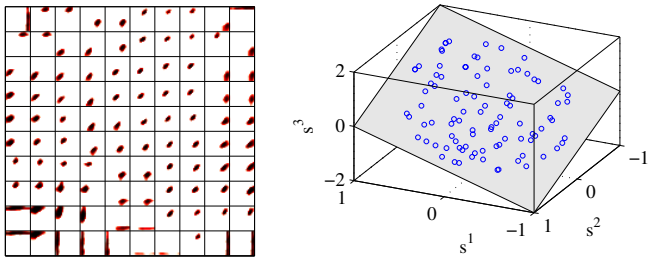



(a) (b)

Fig. 2. The developed receptive fields of all excitatory units: (a) shown in the $s^1$-$s^2$-plane; (b) the distribution of their centers in the $s^1$-$s^2$-$s^3$-space.

For the reduction of topological defects, which may occur during learning, we incorporate an additional process based on the principle of wiring length minimization (WLM). More precisely, each unit tries to dynamically adjust its position in the map such that its weighted distance to other units becomes minimized. This objective can be formalized according to (10)-(11) for E- and I-cells, respectively.

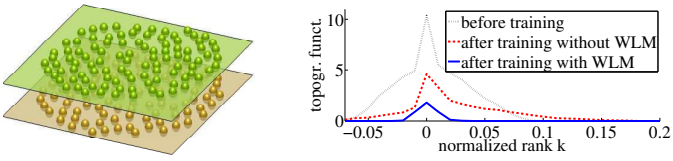
$$\sum_j w_{ij}^{EE} d_{ij}^{EE} + \sum_j w_{ji}^{EE} d_{ji}^{EE} + \sum_j w_{ji}^{IE} d_{ji}^{IE} \rightarrow \min \quad (10)$$

$$\sum_j w_{ij}^{IE} d_{ij}^{IE} + \sum_j w_{ji}^{EI} d_{ji}^{EI} \rightarrow \min \quad (11)$$

As a concrete implementation of this mechanism we suggest an iterative process in which units exert forces on each other. This includes repulsive and attractive forces (both being dependent on the distance between the units) where the attractive forces are additionally modulated by the strengths of the corresponding synapses.

In order to evaluate the model we used three continuously varying stimuli $s^1$,$s^2$,$s^3$ with $s^1, s^2 \in [-1, 1]$ and $s^3 = s^1 - s^2$. Each of them has been represented by a population code composed of 21 neuron responses, resulting in a total of 63 inputs to the network. The model should consequently map the inputs into unique representations such that the input distribution becomes adequately represented. Learning has been carried out during online operation at each timestep.

The developed receptive fields of the E-cells are shown in Fig. 2. As can be seen, the units specialized to distinct input configurations by which a proper sampling of the input space has been achieved. Fig. 3 illustrates the effect of incorporating the additional process of WLM. Firstly, it shows that WLM changes the positions of the cells within the map. Secondly, the topographic function [3] (as a measure for the degree of topological defects in the developed mapping) indicates that the incorporation of WLM significantly enhances topology preservation compared to not using WLM.




(a) (b)

Fig. 3. The final distribution of the cells on the cortical sheet (a) as well as the topographic function [3] of the developed mappings (b).

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