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Modelling structural plasticity

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Introduction

Structural changes in neuronal networks occur not only during development but also in adulthood. Modern imaging techniques have shown pronounced structural plasticity in the living animal, e.g., spontaneous spine dynamics and axonal turnover. Furthermore, network rewiring is a precondition for integrating new neurons into mature neural networks, as occurs in the hippocampal dentate gyrus. In contrast to the importance of structural plasticity in biological neural networks, most models of neural networks only include synaptic plasticity. To our knowledge, only two models exist that study structural neural network formation [1,2]. However, these models lack a precise representation of individual synapses and do not allow for the modelling of neurogenesis. Here, we present a new model for activity-dependent structural plasticity that implements separate axonal and dendritic elements in order to model synaptic turnover and neurogenesis. We have applied the model to two situations. First, we used the model to explain the different response with respect to prefronto-cortical connectivity to enriched and impoverished rearing in an animal (gerbil) model of psychosis [3]. In this animal model, the PFC is disinhibited by applying methamphetamine (MA). Second, we used the model to account for the observed inverse relation between cell proliferation and synaptogenesis in the hippocampal dentate gyrus.

The model

The model consists of simple integrate-and-fire neurons, which can be either excitatory or inhibitory. When the activity of a neuron deviates from a desired value, structural changes in connectivity occur to restore the desired level (homeostatic plasticity). The activity-dependent homeostatic outgrowth rules in Van Ooyen et al. [2] were transferred to discrete excitatory and inhibitory axonal (A_i) and dendritic elements (B_i):

$$\Delta A_i := \nu \cdot \Delta s_i \cdot A_i, \quad \Delta B_i^{exc} := -\nu \cdot \Delta s_i \cdot B_i^{exc} \quad \text{and} \quad \Delta B_i^{inh} := \nu \cdot \Delta s_i \cdot B_i^{inh}$$

where ν gives the velocity of synaptic changes and Δs_i is the deviation of the neuronal average activity from a desired mean value. In addition, new cells can be added, which then integrate into the network following the above rules. Cells are deleted (apoptosis) if their average activity is very much higher or lower than the desired value [4].

Results

Prefronto-cortical connectivity

The simulation revealed that the course of structural reorganisation shaping excitatory and inhibitory connections depend on the previous network connectivity. Under enriched-rearing, early well-matured prefronto-cortical networks compensate a MA induced disinhibition by reducing excitatory contacts. Under impoverished rearing, in contrast, weakly connected networks profit from the activation (caused by disinhibition) and increase connectivity and rather compensate the disinhibition by increasing GABA inhibition. *Cell proliferation versus synaptogenesis in the hippocampus*. The inverse relation between cell proliferation (CP) and synaptogenesis occurs because high CP rates rapidly exhaust available synaptic elements, whereas moderate CP rates leave enough synaptic elements for subsequent synaptogenesis. In general, the

model suggests that activity-dependent homeostatic plasticity underlies structural changes observed in adult cortical and hippocampal networks.

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