

Neurochemical Mechanisms of Perceptual Deficits in Schizophrenic Patients – A Spiking Neural Network Approach

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Categories: Psychiatry

Keywords:

How to cite this poster

Metzner C, Demeter S J, Zurowski B (2012) Neurochemical Mechanisms of Perceptual Deficits in Schizophrenic Patients – A Spiking Neural Network Approach. Cureus 4(9): e110.

Abstract

Schizophrenia is a mental disorder which is characterized by positive symptoms (psychosis, hallucinations and paranoia) and negative symptoms (flattened affect, anhedonia) as well as cognitive and perceptual deficits [1]. Dakin et al. [2] report that schizophrenic patients are less vulnerable to 'contrast-contrast' illusions ('surround suppression', i.e. the mutual inhibition of a focal visual stimulus and its surrounding). This weaker contextual suppression can be interpreted in terms of reduced GABAergic inhibition [3]. Recently, research in schizophrenia has focused on GABAergic inhibitory neural circuits [4]. However, the exact neural basis and perceptual consequences of a compromised GABAergic system remain unclear. We modeled the effects of manipulating particular aspects of GABAergic neurotransmission (altered decay times at GABAergic synapses, reduced availability of GABA and decreased density of GABAergic interneurons [4]) on surround suppression strength. Therefore, we built a model of primary visual cortex based on anatomical and physiological data, using the neuron model from Izhikevich [5]. The model exhibits surround suppression, i.e. shows reduced activity to stimuli mimicking a high-contrast surrounding than compared to stimuli mimicking a uniform surrounding. Furthermore, increased decay times at GABAergic synapses lead to a reduction in surround suppression in our network model. This suggests one possible factor of altered perception in schizophrenia and is in agreement with previous modeling studies, e.g. in the auditory system [6].

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Published 09/19/2012

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