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On the collective computational abilities of inhibitory neurons

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In many parts of the brain such as neocortex, the critical part of information processing is traditionally attributed to excitatory networks, whereas inhibitory neurons are often seen as merely having control functions. For example, theories of associative memory based on attractor networks usually assume that the memories are stored in the excitatory synaptic network, while inhibitory neurons merely provide an appropriate activation threshold or control synchronization [1,3,4].

In this work we challenge this view by analyzing the computational abilities of inhibitory neuron populations [2]. Specifically, we investigate how many sparse binary activation patterns and how much information (in bits per synapse) can be stored in a recurrent or feed-forward inhibitory network, and how the information can be retrieved in an optimal way. Our theory shows that storing a large number of activation patterns requires only a few inhibitory synapses. For realistic population sizes, these networks can already store several bits per synapse (whereas the storage capacity of the classical Willshaw and Hopfield models for infinitely large excitatory networks is limited by 0.72 and 0.14 bits per synapse, respectively).

Moreover, we argue that inhibitory networks can easily implement strategies of optimal threshold control as previously suggested for associative memory in excitatory networks [1]. These strategies significantly enhance the storage capacity by adjusting the firing threshold of a neuron based on its presynaptic input activity. However, no plausible hypothesis has been proposed how biological excitatory neurons could sense presynaptic input activity. Here we show that networks of inhibitory biological neurons have natural access to the presynaptic input. Our results indicate that inhibitory neurons with strong influence on the postsynaptic target are ideally suited for optimal threshold control, for example chandelier cells or basket cells targeting axonal initial segment and cell soma [4].

We have verified our theoretical results by additional simulation experiments of cortex-like network models involving both strongly inhibiting chandelier and basket cells, and less strong gradually inhibiting dendrite-targeting cells. Our theory closely predicts the performance of the simulations, even for realistic neuron models and small as well as large networks. Finally, we discuss possible brain structures where our theory may be of relevance, such as particular networks in the cerebral cortex, basal ganglia, and cerebellum.

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