
A MODEL OF CORTICAL ASSOCIATIVE MEMORY BASED ON HEBBIAN CELL ASSEMBLIES

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ABSTRACT

A model of cortical associative memory, based on Hebb's theory of cell assemblies, has been developed and simulated. The network is comprised of realistically modelled pyramidal-type cells and inhibitory fast spiking interneurons and its connectivity is adopted from a trained recurrent artificial neural network. After-activity, pattern completion and competition between cell assemblies is readily demonstrated. If, instead of pyramidal cells, motor neurons are used as excitatory neurons in the network, spike synchronization can be observed but after-activity is hard to produce. Geometry dependent time delays below 10 ms have little effect. After-activity is facilitated by increased levels of serotonin and disrupted by low levels. Our results support the biological plausibility of Hebb's cell assembly theory.

65.1 BACKGROUND

In his classical book Hebb described a functional unit which he called a cell assembly [1]. This was a group of cells strongly connected through excitatory synapses. Such an assembly could emerge as a result of activated Hebbian synapses. Hebb proposed that the assembly so formed thereafter could serve as an internal representation of the corresponding object in the outside world. Hebb's cell assembly theory has been further elaborated [2, 3] and is still, in its general aspects, compatible with experimental findings. We also see a connection to abstract networks. A prototypical recurrent artificial neural network used as an auto-associative content-addressable memory can be regarded as a mathematical realization of Hebb's basic idea. A "memory trace" in such a network corresponds closely to a cell assembly. The dynamic recall process converging to a low energy, stable state is analogous to the triggering of activity in a cell assembly. Earlier investigators [4, 5] have found, for example, spike synchronization but no signs of after-activity of the kind hypothesized by Hebb.

However, in some of our own investigations, after-activity could indeed be produced provided that the model motor neurons were replaced by pyramidal-cell type neurons [6, 7].

65.2 CELL MODEL AND NETWORK CONNECTIVITY

A general purpose simulator, SWIM, intended for numerical simulation of networks of model neurons with a Hodgkin-Huxley type formalism, has been used [8]. In the present study, two different types of excitatory neurons were simulated, *i.e.* the “P-cell” modelled after a typical cortical pyramidal cell and the “MN-cell” with properties derived from a motor neuron. “FS-cells” modelled after cortical fast spiking cells were used as inhibitory interneurons.

The simulated network was composed of fifty pairs of one excitatory cell and one inhibitory interneuron. The interneuron receives input from excitatory cells in other pairs belonging to other assemblies and inhibits its companion excitatory cell. Excitatory cells in one and the same assembly are connected. Values for synaptic strengths were adopted from a recurrent Bayesian ANN [9] trained with 8 random patterns with 8 active units in each. The Bayesian learning rule produced excitatory synapses within the patterns and inhibitory ones between them.

65.3 ASSEMBLY OPERATION RESULTS

When stimulating all excitatory cells in an assembly comprised of P-cells as the excitatory cells, with 0.4 nA for about 40 ms, the effect was *after-activity* for about 400 ms. For an isolated cell such a stimulation resulted in two or three spikes. Now, due to the mutual synaptic excitation, the cells continue to fire. The firing frequency gradually decreases due to accumulated calcium entering through Ca²⁺- and NMDA-channels. Calcium opens Ca²⁺ dependent K⁺-channels which counteracts synaptic excitation.

Mutual excitation between neurons in the cell assembly also provides the network with a capability for *pattern completion*. With two P-cells out of eight stimulated just transient activity occurred, while with one more cell receiving stimulation activity in the entire assembly was triggered (Fig. 1a).

There is a quite potent lateral inhibition between assemblies in our network. This gives rise to *competition* between assemblies. When five cells in one assembly and three cells in another were stimulated, the first assembly prohibited the activity in the second assembly and completed its own missing cells (Fig. 1b). In another simulation, when the stimulus used activated a part of a cell assembly together with some randomly activated cells, the spurious cells were quickly silenced and the missing ones activated. This demonstrates the *noise tolerance* of the network operation.

Worth noting in this context is the rather short time required for the activation of a complete assembly. Almost without exception, even in cases of

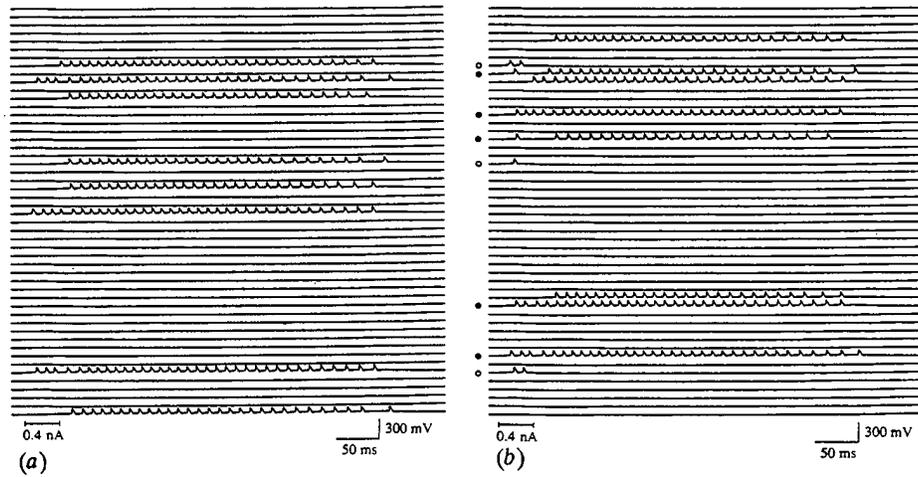


Figure 1 (a) Pattern completion and after-activity in an assembly with three cells stimulated. Each trace shows the activity of one P-cells. (b) Competition between two assemblies. The stimulated cells in the two different patterns are marked with open and closed circles.

conflicting input, a clean “interpretation” was stable 50–70 ms after stimulus onset in our simulations (see e.g. Fig. 1a, b). These *reaction times* are also short enough to support sustained processing times below the 100 ms observed in some experiments on perceptual tasks e.g. visual object identification [10].

The capability to produce after-activity of an assembly consisting of motor neurons was also investigated. After-activity was observed only in exceptional cases. One explanation seems to be that the prominent AHP of the MN-cell to a large extent masks the EPSP produced by the mutual excitatory synapses. This effect is enhanced by the high firing threshold of the MN-cells and by their tendency to *spike synchronize*. To investigate if the assembly size had been too small assemblies with 100 cells were tested. Qualitatively the same behavior was seen. At least under the conditions studied here, the P-cell assembly did not seem to give very significant spike synchronization. If continuously stimulated, an assembly consisting of MN-cells also displayed some pattern completion tendencies.

In an extended model, *time delays* have been introduced to represent separation of the cells in space. For a network with P-cells as the principal cells, an average delay of up to 10 ms was possible without any significant changes in assembly operation. With longer time delays performance began to degrade. The spike synchronization tendencies of the MN-cell network decreased already at delays of 3–4 ms. Thus, the important assembly operations do not seem very sensitive to short time delays, which means that a cell assembly may be distributed over a relatively large area and still be operating reliably.

We model the *neuromodulatory effects* of serotonin by decreasing the con-

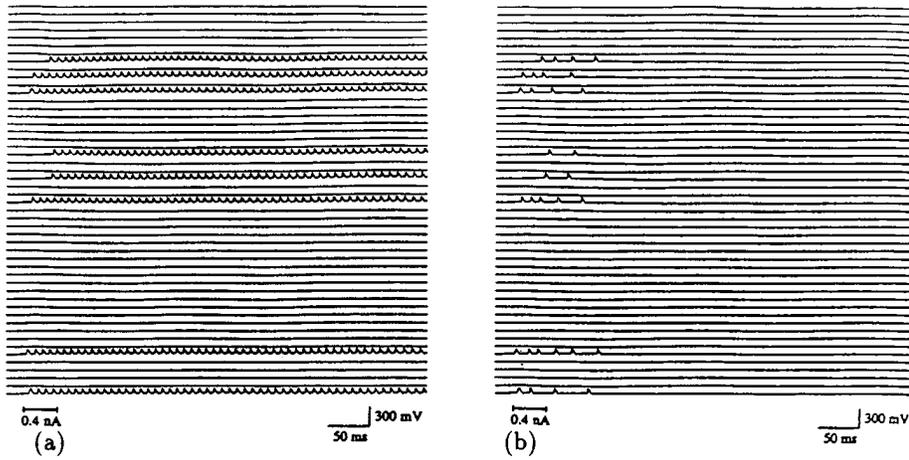


Figure 2 (a) An assembly of P-cells with reduced AHP showing enhanced assembly operation. Stimulation on 5 out of 8 cells. (b) An assembly of P-cells with increased AHP giving no after-activity.

ductance through the calcium dependent potassium channels thus decreasing the AHP amplitude [11]. This mechanism provides a means for modulating cell properties in a way that dramatically influences the assembly-related network operations [12]. When the AHP is reduced triggering is facilitated and the duration of after-activity is prolonged (Fig. 2a). When the AHP is increased these features weaken and disappear (Fig. 2b). The behavior in this case resembles more that of the motor neuron network.

65.4 CONCLUSIONS

The simulation results presented here show that Hebbian cell assembly related activity could readily be produced by a network with cortical pyramidal cells as principal excitatory cells. Such a network would also display pattern completion, noise tolerance and competitive phenomena in cases of conflicting inputs in much the same way as a recurrent ANN. One important prerequisite is that the model, of the excitatory neurons used, is provided with properties of cortical pyramidal cells rather than spinal motor neurons. When effects of serotonin were modeled the operation was strongly affected. With high levels assembly related activity was enhanced, with low levels it was suppressed. If the time delays in the network were below 10 ms, performance was little affected. Our simulation results support the biological feasibility of Hebb's cell assembly theory and could eventually provide a bridge between recurrent artificial neural network models and biological models of associative memory.

65.5 ACKNOWLEDGEMENTS

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