Parallel Hopfield Networks

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There is a growing body of evidence for the existence of precisely timed spikes in the brain. This idea has led to the development of a plethora of computational models exploiting precise spike timing (e.g. [1]). One such model is the Concurrent Recall Network (CRN) model of Wills [2]. In these networks, memories are represented by asynchronous firing patterns that are stored in the system by making use of variable axon delays between neurons to 'resynchronize' the otherwise asynchronous input to each neuron, a concept known as Polychronization [3]. In addition, the network employs conjunction detectors on each neuron which are simplified models of spiking dendrites [4]. These novel elements vastly improve the performance of the CRN by reducing the interference between different memories allowing multiple memories to be recalled simultaneously - hence the name Concurrent Recall Networks.

A limitation of the CRN model is that the memories are binary in that they are either activated or not. A different approach is to allow memories to be partially activated with only a subset of neurons spiking at the specified time. By creating these different sub-patterns 'within' each CRN memory, the memory capacity of the system expands rapidly.

In particular we choose to store random patterns of activity in these `sub-networks' in a prescription similar to the Hopfield model [5]. In this case, each CRN style memory acts as an independent attractor network implementing a noisy approximation to the usual Hopfield dynamics. In addition, the network retains the concurrent recall ability and is able to simultaneously enact the dynamics of multiple Hopfield networks in parallel in the same network. Hence we term these networks Parallel Hopfield Networks.

It is possible to study these networks analytically and we have obtained mean-field equations for the order parameters of the Hopfield sub-networks. Solution of these equations allows the computation of the memory capacity of each of the Hopfield sub-networks as a function of the number of stored CRN memories and the amount of background activity (caused by recall of other CRN memories or neuronal noise) in the network. Predictions from this theory are in agreement with initial simulations on networks with large numbers of neurons.

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References

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