Functional consequences of non-equilibrium dynamics caused by antisymmetric learning rules

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Connectivity in the brain is asymmetric, which is evident by Dale's principle of excitatory and inhibitory neurons. As a consequence, biologically realistic neuronal networks cannot be in thermodynamic equilibrium [1]. Even in a stationary state, probability fluxes perpetuate, leading to non-equilibirium steady states [2, 3]. We here investigate the computational consequences of non-equilibrium dynamics for synaptic plasticity and learning. Formulating the stochastic dynamics in sparsely connected networks of non-linear Langevin equations in terms of path integrals [4], we show that biologically plausible correlation-sensitive plasticity rules follow from first principles: maximizing a measure of irreversibility [2] by gradient descent with respect to the weights, we obtain a local learning rule sensitive to the derivative of the covariance between the pre- and postsynaptic neuron. The obtained rule can be interpreted as spike-timing-dependent plasticity (STDP) [5, 6] with a narrow asymmmetric learning window. We show that the learning rule increases synaptic weights in the direction of the (direct or indirect) causal influence between a pair of neurons. In this way indirect causal relationships are transformed into strenghtened direct connections. We derive analytical expressions that describe these effects quantitatively. The antisymmetric origin of the obtained learning rules leads to a decay of the symmetric part of the connectivity matrix. An instability of the state with vanishing weights manifests itself as spontaneous symmetry breaking and the divergence of connection weights. We show how nonlinearities in the neuronal transmission stabilize synaptic weights and how the limited dynamic range of neuronal activity mediates competition between synapses.

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