

Spike timing-dependent plasticity and the didactic reorganization of cortical receptive fields.

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The precise nature of the synaptic plasticity that governs neuronal receptive field properties *in vivo* is not known. V1 neurons that have their receptive fields eclipsed by a circumscribed retinal lesion subsequently form ‘new’ receptive fields on intact retina outside the lesion. We studied the specific directions in which these receptive field ‘shifts’ occur, and compared them to those produced by a network model of V1. We observed that the *in vivo* receptive fields rarely shifted towards the closest available region of intact retina. This result is surprising, since it is inconsistent with the expectation that neurons respond to feedforward input loss by potentiating their strongest available intracortical connections [1, 2]. Furthermore, even when these receptive fields were separated by large distances prior to lesioning, creation of the lesion caused them to shift in a correlated, convergent manner. The V1 model represented a 2D topographic arrangement of neuron populations with narrow feedforward and broad intracortical input connections that were constrained by electrophysiological and anatomical data. These connections were modulated by both homeostatic plasticity (eg ‘synaptic scaling’ [3]) and homosynaptic plasticity (LTP/LTD). A detailed comparison of our empirical and simulation results revealed that the *in vivo* receptive field shifts we observed are consistent with synaptic modifications at V1 intracortical connections that are dependent upon the temporal order of pre- and post-synaptic spikes (spike timing-dependent plasticity) and are inconsistent with modifications that depend only on the temporal correlation between these spikes (correlation-dependent plasticity). Consistent with previous theoretical work [4], our results indicate that spike timing-dependent plasticity drives receptive field shift convergence by creating competition between neurons for the control of spike timing within the network. In our model the spatial scale of this competition is controlled by the network’s homeostatic balance of excitation and inhibition, revealing a novel means by which the theorised ‘didactic’ capacity of spike timing-dependent plasticity to transfer response properties between neurons [4] can be effectively switched on and off.

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References

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