Control of synaptic plasticity in visual cortex

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NMDAreceptor-dependent forms of plasticity such as long-term potentiation (LTP) and long-term depression (LTD) are held to be the cellular mechanisms by which sensory experience shapes cortical circuits during development. According to a prevailing view, the NMDA receptor serves as a coincidence detector that senses the correlation between the pre- and postsynaptic activity, and translate it into an intracellular Ca signal that specifies the polarity (LTP or LTD) and magnitude of plasticity. However, a cardinal feature of cortical plasticity is that it depends not only on the patterns of activity evoked by sensory experience, but it also requires neuromodulatory inputs related to the behavioral state of the animal. Hence, we have been investigating how two major neurodulators, Acetycholine and Noradrenaline, control LTP and LTD in visual cortex. Using associative paradigms in which the timing relation between pre and post-synaptic activity is controlled, we have found that these neuromodulators gate and determine the polarity of plasticity. Activation of receptors coupled to the phospholipase C specifically promotes associative LTD; whereas activation of receptors linked to the adenylil cyclase pathway promote associative LTP. Altogether, these findings suggests that the neuromodulatory systems are important determinants of synaptic strengthening and weakening in visual cortex. Such actions of neuromodulators provide a basis for understanding more integrative functions in cortical plasticity at a cellular level.

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