The contribution of spontaneous activity to plasticity can explain pre-critical and critical period plasticity of ocular dominance.

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In mice, maturation of cortical inhibitory circuitry initiates the critical period (CP) for ocular dominance (OD) plasticity in response to monocular deprivation (MD). A certain threshold of inhibition is required for the initiation of the CP \cite{1}. However, the mechanism by which maturation of inhibition initiates the CP is not known. This maturation of inhibition is not simply a switch that turns on OD plasticity. An imaging study of mice revealed that the retinotopic precision of cortical receptive fields is constantly being refined by activity dependent plasticity during the pre-CP \cite{2}. MD of the stronger, contralateral eye retards the refinements of both eyes’ retinotopy without changing the relative strength of the two eyes’ responses \cite{2}. That is, before the CP, MD does not cause an OD shift, but it does affect activity-dependent refinement of retinotopy.

We study how the maturation of inhibition initiates the CP of OD plasticity, and how there can be activity-dependent plasticity without OD plasticity before the maturation of inhibition. More specifically, we study the hypothesis that the relative contribution of spontaneous activity and visually evoked activity on synaptic plasticity changes at the onset of the critical period due to maturation of inhibition and a nonlinear input-output function. Before the critical period, frequent but weak spontaneous input can drive neurons and thus modulate synapses, but maturation of cortical inhibition prevents spontaneous input from driving neurons. To investigate the effect of a reduced amount of spontaneous activity, activity dependent synaptic plasticity is modeled as a phenomenological learning rule for synaptic weights that has the Hebbian property as well as a homeostatic property. While Hebbian learning can extract a correlation structure in input, homeostatic plasticity regulates the overall activity level and stabilizes the learning.

During the pre-CP the receptive fields of neurons are retinotopically refined and strengthened through activity-dependent plasticity, partially because of weaker but frequent drive from spontaneous activity in the two eyes and partially because of stronger but occasional drive from visual activity in the eyes. The contribution of frequent spontaneous activity allows partial retinotopic refinement and strengthening under MD of the contralateral eye, but the refinement is weaker due to the abnormal visual activity of the deprived eye. The reason that MD does not change the balance between open and closed eyes is that the responses to input from each eye are roughly balanced because of the frequent spontaneous input. The maturation of inhibition at the onset of the CP reduces spontaneous activity, so that cortex becomes mainly driven by visually-evoked activity from the eyes. Application of MD will then cause an OD shift to the open eye because, under MD, there is a large imbalance in the input from two eyes. Hence, if maturation of inhibition suppresses the effect of weak spontaneous input, this can explain (1) plasticity during the pre-CP: there is activity-dependent refinement of receptive fields, and MD weakens refinement but does not cause an OD shift; and (2) the initiation of the CP – the period in which MD causes an OD shift – by the maturation of inhibition.

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References