Plasticity of rat somatosensory cortex following sensory deprivation

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Experience with sensory stimuli leads to changes of cortical circuits, a phenomenon generally referred as cortical plasticity. Understanding the mechanisms of cortical plasticity is fundamental for understanding neural basis of learning and critical for treatment and rehabilitation patients with the damaged cortex. Homeostatic plasticity is a critical component of cortical plasticity along with activity driven Hebbian plasticity. A conventional theory of homeostatic plasticity of cortical circuits states that homeostatic plasticity aims to maintain activity of cortical circuits. Here we suggest that homeostatic plasticity may not simply attempt to preserve the activity of the cortical area but to maintain a functional ability of the deprived cortical area.

The sensory cortex S1 of rodents is a powerful model system for studying neural mechanisms of cortical plasticity. Recent experiments showed that whisker trimming in young rats resulted in a reduction of recurrent excitation and inhibition in layer 2/3 of rodent S1 cortex (Shao et al., 2013). The reduction in recurrent inhibition occurred despite strengthening FS interneurons to pyramidal synapses (House et al., 2011). The weakening feedforward and reciprocal interactions between pyramidal neurons and inhibitory interneurons following sensory deprivation decreases circuit responses to a stimulus (Li et al., 2014).

One of the recent hypothesis of how sensory cortex operates is so called inhibition stabilized network (ISN), according to which strong excitation in the cortex is balanced by strong inhibition. In a mean-field model of cortical circuits, we show that the reduced neural responses in L2/3 could weaken ISN regime, but plasticity in inhibitory neurons (f-I curve change) can restore ISN regime even in the deprived sensory cortex. The model predicts that Hebbian plasticity could be responsible for weakening neural responses in L2/3 and homeostatic plasticity could responsible for changes in inhibitory neurons.