Modeling the Dynamic Interaction of Hebbian and Homeostatic Plasticity

Taro Toyoizumi

Laboratory for Neural Computation and Adaptation, RIKEN Brain Science Institute, Japan

Email of Presenting Author: taro.toyoizumi@brain.riken.jp

Hebbian and homeostatic plasticity together refine neural circuitry, but their interactions are unclear. In most existing models, each form of plasticity directly modifies synaptic strength. Equilibrium is reached when the two are inducing equal and opposite changes. We show that such models cannot reproduce ocular dominance plasticity (ODP) because negative feedback from the slow homeostatic plasticity observed in ODP cannot stabilize the positive feedback of fast Hebbian plasticity. We propose a model in which synaptic strength is the product of a synapse-specific Hebbian factor and a postsynaptic-cell-specific homeostatic factor, with each factor separately arriving at a stable inactive state. This model captures ODP dynamics and has plausible biophysical substrates. We confirm model predictions experimentally that plasticity is inactive at stable states and that synaptic strength overshoots during recovery from visual deprivation. These results highlight the importance of multiple regulatory pathways for interactions of plasticity mechanisms operating over separate timescales.