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Title

Intrinsic spine dynamics are critical for recurrent network learning in models with and without autism spectrum disorder

Abstract

It is often assumed that Hebbian synaptic plasticity forms a cell assembly, a mutually interacting group of neurons that encodes memory.

However, in recurrently connected networks with pure Hebbian plasticity, cell assemblies typically diverge or fade under ongoing changes of synaptic strength. Previously assumed mechanisms that stabilize cell assemblies do not robustly reproduce the experimentally reported unimodal and long-tailed distribution of synaptic strengths.

Here, we show that augmenting Hebbian plasticity with experimentally observed intrinsic spine dynamics can stabilize cell assemblies and reproduce the distribution of synaptic strengths. Moreover, we posit that strong intrinsic spine dynamics impair learning performance. Our theory explains how excessively strong spine dynamics, experimentally observed in several animal models of autism spectrum disorder, impair learning associations in the brain.

Bio

Taro Toyoizumi is a Team Leader at RIKEN Center for Brain Science. He received his B.S. in physics from Tokyo Institute of Technology in 2001, and his M.S. and Ph.D. in computational neuroscience from the University of Tokyo in 2003 and 2006, respectively. He studied at the Center for Theoretical Neuroscience at Columbia University as a JSPS and Patterson Trust Postdoctoral Fellow. He then came to RIKEN Brain Science Institute as a Special Postdoctoral Researcher in

2010, was promoted to a Lab Head in 2011, and holds the current position since 2018. He has been studying the theory of neural plasticity by asking how neural circuits self-organize in the environment. Toyoizumi has received the International Neural Network Society, Young Investigator Award in 2008 and the Commendation for Science and Technology by the MEXT Japan, Young Scientists' Prize in 2016.