Heller Lecture - Prof. Carla Shatz

June 4, 2013

On the topic of "Brain Circuit Tuning During Developmental Critical Periods"

Heller Lecture Series in Computational Neuroscience

Abstract

Connections in adult brain are highly precise, but they do not start out that way. Precision emerges during development as synaptic connections remodel in a process requiring neural activity (action potentials and synaptic transmission). Activity also regulates neuronal gene expression. In an unbiased screen, Major Histocompatibility Class I (MHC1) genes were unexpectedly discovered to be in neurons and regulated by activity and visual experience (Corriveau et al, 1998).
To assess requirements for MHCI in CNS, mutant mice lacking stable surface expression of all MHCI, or specific MHCI genes, were examined. Synapse regression in developing visual system fails, and ocular dominance (OD) plasticity in visual cortex is greater than in WT (Huh et al, 2000; Datwani et al, 2009). In a search for receptors that could interact with neuronal MHCI, PirB, an innate immune receptor, was found highly expressed in neurons throughout mouse CNS. In mutant mice lacking PirB, OD plasticity is increased (Syken et al., 2006), as is LTP in hippocampus. Thus, PirB, like MHCI, appears to act to "brake" synaptic plasticity. Moreover, the commonality of phenotypes present in these mice suggests a model (Shatz, 2009) in which PirB may bind and transduce signals from MHCI ligands in neurons. Together, results imply that this family of molecules, thought previously to function only in immunity, may also act at neuronal synapses to limit how much- or how quickly- synapse strength changes in response to new experience. These molecules may be crucial for controlling circuit excitability and stability in developing as well as adult brain. Changes in their function could contribute to developmental disorders such as Autism and Schizophrenia.

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