This Week in The Journal

Cellular/Molecular

RIM3γ and RIM4γ Are Required for Dendritic Growth

Elena Alvarez-Baron, Katrin Michel, Tobias Mittelstaedt, Thoralf Opitz, Frank Schmitz, et al.

(see pages 824 – 839)

Four genes encode Rab3 interacting molecules (RIMs) in mammals. RIM1α and RIM2α are widely studied scaffolding proteins that cluster calcium channels and dock synaptic vesicles at presynaptic active zones. Considerably less is known about RIM3γ and RIM4γ, which lack most of the protein interaction domains present in α-RIMs. But Alvarez-Baron et al. report that RIM3γ and RIM4γ are expressed throughout rodent brains, with partially overlapping expression patterns. Unlike α-RIMs, RIM 3 and 4 were present not only in presynaptic terminals, but also in dendrites, including postsynaptic densities. Knockdown of RIM3γ or RIM4γ greatly reduced dendritic branching, spine density, and synapse numbers in hippocampal cultures and *in vivo*. Although γ-RIM knockdown also reduced axonal growth and branching *in vitro*, impaired glutamate release probably was not the source of dendritic effects, because these effects were not replicated by blocking AMPA receptors. γ-RIM knockdown altered the structure of the Golgi apparatus, however, indicating that impaired protein or lipid trafficking may underlie reduced axonal and dendritic growth.

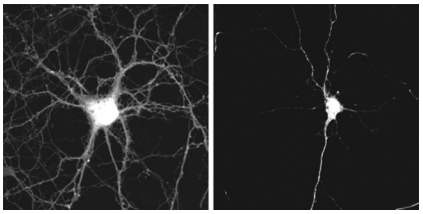
Development/Plasticity/Repair

LTP Increases Stability of Individual Spines

Travis C. Hill and Karen Zito

(see pages 678 – 686)

Dendrites continuously form new spines, which can either become stabilized or retract and disappear. Such changes are likely to be essential components of experience-dependent plasticity, circuit reorganization, and learning. Novel sensory experiences increase spine formation and stabilization, thus increasing spine density and size. Similarly, electrical and pharmacological stimuli that induce long-term potentiation (LTP) of synaptic strength promote spine growth. Using two-photon glutamate uncaging to stimulate single spines in organotypic hippocampal slice cultures from week-old rats, Hill and Zito demonstrate that stimuli that induce LTP increase the stability of individual spines without affecting the stability of unstimulated spines on the same dendrite. Increased stability was correlated with increased spine size and required activation of NMDA receptors as well as interactions between these receptors and calcium/calmodulin-dependent kinase II. Stimulation did not significantly increase stability of the largest spines, however, which were more stable than smaller spines even without exogenous stimulation.



Hippocampal neurons in which RIM3γ was knocked down beginning at 3 days *in vitro* (right) had smaller dendritic arbors 11 days later than control neurons (left). See the article by Alvarez-Baron et al. for details.

Behavioral/Cognitive

LIP Neuron Activity Is Associated with Express Saccades

Mo Chen (陈默), Yu Liu (刘昱), Linyu Wei (魏林郁), and Mingsha Zhang (张鸣沙)

(see pages 814 – 823)

Saccades are rapid eye movements that center objects of interest on the fovea. Based on their latencies, saccades can be divided into two populations, called express and regular saccades. The former are most likely to occur when a target appears in an expected location or when a delay separates the disappearance of a fixation target and the appearance of a saccade target. A prominent hypothesis suggests that express saccades are produced by a circuit that includes primary visual cortex and superior colliculus, but bypasses higher cortical areas. Contrary to this hypothesis, however, lesion and electroencephalography studies have suggested that parietal areas are important for generating express saccades. Chen et al. bolster the latter hypothesis, showing that ~34% of neurons in monkey lateral inferior parietal cortex (LIP) fired more strongly before express saccades than before regular saccades. The firing rate of these neurons was inversely correlated with the saccade reaction time. The authors propose that the increased activity in LIP represents preparation to make a saccade.

Neurobiology of Disease

Connexin30 Is Not Required for Hearing

Anne-Cécile Boulay, Francisco J. del Castillo, Fabrice Giraudet, Ghislaine Hamard, Christian Giaume, et al.

(see pages 430 – 434)

Cochlear hair cells rest on a rigid bed of cells that envelop auditory nerve terminals and provide structural support, maintain extracellular K^+ homeostasis, and probably provide nutrients to hair cells. Supporting cells are connected by gap junctions composed primarily of connexin (Cn) 26 and 30. The function of these junctions is poorly understood, but they propagate calcium waves, permit K^+ recycling, and may pass nutrients such as glucose and amino acids. Although gap junction proteins are not expressed in hair cells, they are critically important for hearing: mutations in Cn26 are the most common hereditary cause of deafness. Mutations in Cn30 have also been linked to hearing loss, and knockout of Cn30 causes deafness in mice, but because Cn26 expression is greatly reduced in these cases, the importance of Cn30 has not been established. Therefore, Boulay et al. generated a new Cn30-null mouse line in which Cx26 expression is largely maintained. Surprisingly, these mice had normal hearing, indicating that Cx30 is dispensable for this function.