This Week in The Journal

Cellular/Molecular

Mitochondrial Calcium Handling at Mossy Fiber Boutons

Doyun Lee, Kyu-Hee Lee, Won-Kyung Ho, and Suk-Ho Lee

(see pages 13603–13613)

Dentate granule cells essentially can speak with two tongues. The axonal branches of their mossy fibers innervate CA3 pyramidal cells and interneurons, each with distinct morphological and functional properties. Lee et al. stimulated mossy fibers and compared the response of putative large boutons at hilar mossy cells with smaller en passant boutons onto hilar interneurons. Following high-frequency stimulation (HFS), the intracellular residual calcium (Ca_{res}) was sustained for tens of seconds in large mossy fiber boutons (MFBs). Smaller en passant MFBs had a much quicker drop in intracellular calcium. Inhibitors of mitochondrial pumps increased Ca_{res} in large but not small MFBs, consistent with a buffering role for mitochondria in the large boutons. During HFS, calcium uptake depended on the inner mitochondrial membrane calcium uniporter, after which the mitochondrial Na+/Ca^2+ exchanger (mitoNCX) contributed to Ca_{res}. Post-tetanic potentiation at mossy cells, but not at interneurons, was affected by blocking the mitoNCX.

Development/Plasticity/Repair

β1 Integrin and Cortical Development

Richard Belvindrah, Diana Graus-Porta, Sandra Goebbels, Klaus-Armin Nave, and Ulrich Müller

(see pages 13854–13865)

In this week’s Journal, Belvindrah et al. revisited the proposed role for the extracellular matrix receptor β1 integrin in the developing cortex. The authors used a Nex-CRE mouse line in which integrin subunits could be conditionally inactivated. Itgb1-NEXKO mice lacked β1 in precursors that generated migrating neurons. Itga3-null mice were deficient in α3 subunits in all cell types and thus did not express α3β1 integrins. As expected, cortical layers were disrupted in brains of mice lacking α3β1 in glia and neurons, but selective deletion in migrating pyramidal neurons did not affect their migration and cell layer formation, ruling out a pivotal role for integrins in migration. However, the expression of β1 integrins in glia affected the morphological differentiation of glia and neurons.

Behavioral/Systems/Cognitive

Rotating and Translating in the Thalamus

Hui Meng, Paul J. May, J. David Dickman, and Dora E. Angelaki

(see pages 13590–13602)

In this week’s Journal, Meng et al. mapped the projections that confer vestibular sensation to and from the thalamus in primates. Monkeys, seated in a vestibular turntable, experienced rotation in pitch, roll, and yaw dimensions, as well as translation in the horizontal plane. The authors made extracellular recordings from >1000 thalamic neurons, ~10% of which responded to vestibular stimulation. Although some cells responded exclusively to either tilt or translation, most showed intermediate responses to both stimuli, suggesting that they received signals from the otolith and semicircular canal. The vestibular responsive neurons were more widely dispersed than expected, with no apparent functional segregation. Injection of a neuronal tracer dye revealed that these thalamic neurons received input from cerebellar nuclei. The authors concluded that several thalamic nuclei contribute to precortical vestibular processing and that their inputs come from cerebellar as well as vestibular nuclei.

Neurobiology of Disease

Cone-Specific Degeneration in Zebrafish

George Stearns, Meredelfa Evangelista, James M. Fadool, and Susan E. Brockerhoff

(see pages 13866–13874)

Dozens of mutations contribute to progressive blindness in humans, many of which result from photoreceptor degeneration. More of these identified mutations affect rods. This week, Stearns et al. identified a mutation in cone photoreceptors that caused cone degeneration as well as “bystander” death of some rods. F3 generation progeny from fish mutagenized with ethyl nitrosourea were screened initially for their optokinetic response. The screen yielded a recessive mutation that affected the cone-specific phosphodiesterase c (pde6c) gene. Four days postfertilization, cones in mutant fish larvae had already degenerated. Cone degeneration resembled the rod degeneration seen in the mouse rd1 mutation, which affects the rod-specific pde6b. Location mattered to pde6c mutant photoreceptors, particularly to doomed rods unlucky enough to reside near cones. The lucky rods, those in densely packed cone-free areas, were spared degeneration.

The image shows a dorsal view of normal (responder) fish or fish that were blind as a result of a mutation in the cone phosphodiesterase (mutant). The size of the eye in the mutant was normal, but homozygous mutants were blind by 5 d postfertilization.