This Week in The Journal

● Cellular/Molecular

Segregation of CNS and PNS Myelin

Sarah Kucenas, Wen-Der Wang, Ela W. Knapik, and Bruce Appel

(see pages 15187–15194)

The CNS is separated from surrounding tissue by the glia limitans, a meshwork of glial processes covered in basal lamina. During development, axons of motor neurons extend through this barrier at motor exit points. The axons are myelinated by oligodendrocytes within the CNS and by Schwann cells in the periphery; in the transitional zone lies a node of Ranvier that is myelinated on one side by a Schwann cell and on the other by an oligodendrocyte. What prevents these glia from migrating through the motor exit points? Using time-lapse imaging of genetically color-coded glia, Kucenas et al. found that in mutant zebrafish lacking Schwann cells, oligodendrocytes migrated into the periphery, where they myelinated axons, suggesting interactions between oligodendrocytes and Schwann cells normally contribute to the CNS–PNS boundary. But because Schwann cells associate with motor axons before oligodendrocytes are born, a different mechanism must keep Schwann cells out of the CNS.

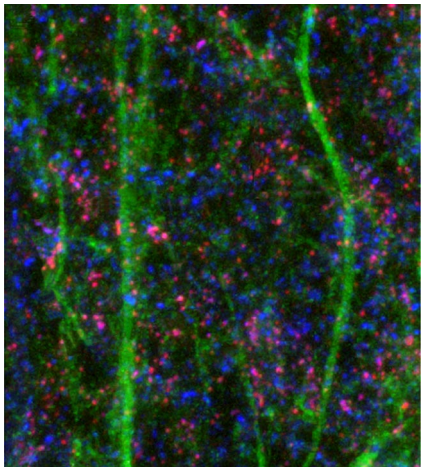
▲ Development/Plasticity/Repair

Synaptic Maturation in Adult-Born Olfactory Granule Cells

Patrizia Panzanelli, Cedric Bardy, Antoine Nissant, Marta Pallotto, Marco Sassoè-Pognetto, et al.

(see pages 15039 –15052)

Thousands of granule cells are born daily in the subventricular zone of adult mice. The cells travel along the rostral migratory stream to the olfactory bulb, where they become integrated into circuits. To study how quickly newborn neurons are integrated, Panzanelli et al. infected migrating neurons with virus encoding enhanced green fluorescent protein (eGFP) and analyzed synaptogenesis 3–7 d later. Even at 3 d, when dendrites were relatively short and unbranched, clusters of GABA receptor subunits and scaffolding proteins were present on somata and dendrites. Whole-cell recordings indicated that these cells received both GABAergic and glutamatergic input. Over the next 4 d, more migrating neurons arrived in the bulb and rapidly developed a mature morphology with highly branched, spine-covered dendrites. As soon as dendrites extended into the external plexiform layer, they made characteristic dendrodendritic synapses with tufted and mitral cells. Although the number of GABAergic synapses increased, their density remained stable, whereas the density of glutamatergic synapses increased.



Dendrites of adult-born granule cells (green) in the olfactory bulb. GABAergic synapses, marked by the GABA receptor α2 subunit (red) and the scaffolding protein gephyrin (blue) were present in newborn neurons at the earliest times examined. See the article by Panzanelli et al. for details.

■ Behavioral/Systems/Cognitive

Fear-Induced Reduction of Eating

Gorica D. Petrovich, Cali A. Ross, Pari Mody, Peter C. Holland, and Michela Gallagher

(see pages 15205–15212)

Pavlovian conditioning does not generally produce inflexible responses to conditioned stimuli (CS); the response varies with the value of the associated unconditioned stimulus (US). Associating the CS with the US is thought to require the basolateral amygdala (BLA), whereas selection of the appropriate response occurs downstream, in the central nucleus (CEA), which receives input from BLA. In addition to this serial processing, these amygdalar subregions are thought to serve some distinct functions mediated by separate projections from BLA to prefrontal cortex and direct sensory input to CEA from the thalamus. Petrovich et al. attempted to distinguish the roles of these regions in mediating the effects of conditioning on eating, because such effects might contribute to eating disorders. A tone previously paired with foot shock inhibited feeding. Although this effect appeared to be eliminated by lesion of CEA but not BLA, ANOVA found no significant effect of lesion, and the significant effect of conditioning did not depend on the type of lesion.

♦ Neurobiology of Disease

Increased Excitability of Spinal Interneurons in ALS

Mingchen Jiang, Jenna E. Schuster, Ronggen Fu, Teepu Siddique, and C. J. Heckman

(see pages 15031–15038)

The causes of motor neuron degeneration in amyotrophic lateral sclerosis (ALS) are unknown, but one possible cause is excitotoxicity. Motor neurons express calcium-permeable AMPA receptors and have poor calcium-buffering ability, making them especially vulnerable to death resulting from excess calcium influx. Furthermore, a drug that slows progression of ALS decreases excitotoxicity by interfering with glutamatergic transmission. Glutamate-induced excitotoxicity could result from intrinsic changes to motor neurons, such as changes in receptor composition, or from increases in synaptic input. To explore the latter possibility, Jiang et al. recorded nerve activity in spinal cord preparations from mice harboring a mutated form of superoxide dismutase that causes ALS. The response to short-latency input did not increase as the disease progressed, but synchronized bursting induced by blocking inhibition increased. Furthermore, spontaneous synchronous bursting appeared in the late stages of disease. The increased bursting, which was eliminated by blocking AMPA or NMDA receptors, was attributed to increased excitability of spinal interneurons.