This Week in The Journal

● Cellular/Molecular

Properties of Striatal Interneurons

Aryn H. Gittis, Alexandra B. Nelson, Myo T. Thwin, Jorge J. Palop, and Anatol C. Kreitzer

(see pages 2223–2234)

The striatum is the main input structure of the basal ganglia, and its output, carried by medium spiny neurons (MSNs), shapes motor behaviors in part by suppressing unintended behaviors. MSNs integrate inputs from cortex, thalamus, other MSNs, and local interneurons. Determining the role of interneurons in shaping MSN output has been difficult, however, because interneurons are sparse and difficult to target electrophysiologically. Gittis et al. circumvented this problem by using mice in which the two major types of striatal interneurons—persistent low-threshold spiking (PLTS) and fast spiking (FS)—were labeled with GFP. The two subpopulations differed in electrophysiological properties, expression of glutamate receptors, and connectivity. Notably, PLTS neurons rarely synapsed onto MSNs within 250 μm of their somata, whereas FS neurons frequently did. Furthermore, IPSCs evoked in MSNs by PLTS neurons were much weaker than those produced by FS neurons. These data suggest that FS neurons provide the major source of local inhibition to MSNs

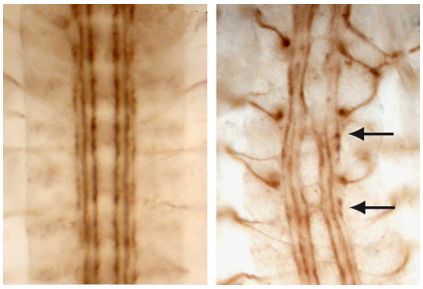
▲ Development/Plasticity/Repair

Role of Vav in Axon Guidance

Marianne Malartre, Derya Ayaz, Fatima Fernandez Amador, and Maria Dolores Martín-Bermudo

(see pages 2257–2267)

Rho GTPases link extracellular cues to changes in the cytoskeleton that underlie neurite growth and guidance. Guanine nucleotide exchange factors (GEFs) usually act upstream of Rho GTPases in this pathway, switching them from the inactive, GDP-bound state to the active, GTP-bound state. This week, Malartre et al. show that in *Drosophila*, the GEF vav is involved in axon pathfinding. In vav-null embryos, axons abnormally crossed the midline; in vav-null larvae, some photoreceptor axons failed to terminate in their target area. These results suggest vav is involved in repulsive guidance. Expression of constitutively active vav in photoreceptors also disrupted axon patterning; but this defect was reduced in flies in which the Rho GTPase Rac was mutated, suggesting that vav activates Rac. Although vav mutations were usually lethal, some larvae survived and, in these, additional axon growth defects occurred during CNS reorganization at metamorphosis. Therefore, vav functions in axon growth at all stages of development.



FasciclinII staining of axons in wild-type (left) and vav-null (right) *Drosophila* embryos. Axons abnormally crossed the midline in mutants (arrows). See the article by Malartre et al. for details.

■ Behavioral/Systems/Cognitive

Temporally Heterogeneous Activity in Olfactory Receptor Neurons

Baranidharan Raman, Joby Joseph, Jeff Tang, and Mark Stopfer

(see pages 1994–2006)

Odors are encoded in the olfactory bulb of mammals and in the antenna lobe of insects by the temporal pattern of activity in specific ensembles of neurons. These patterns were thought to arise in the central structures, which were assumed to receive temporally homogenous input from olfactory receptor neurons (ORNs). But recent studies have suggested this assumption is false. Raman et al. report that although the summed response of ORNs measured from locust antennae shows little temporal structure, the responses of individual ORNs are temporally heterogeneous, varying with odor identity, concentration, and duration. A computational model that incorporated ORN properties generated activity patterns in model antenna lobe projection neurons that closely resembled projection neuron activity *in vivo*. In contrast, temporally homogenous input did not generate realistic activity patterns in the model. Similarly, manipulating odor pulse durations to produce temporally homogenous output of ORNs *in vivo* elicited relatively simple activity patterns in the antenna lobe.

♦ Neurobiology of Disease

Effects of ATP on Bladder Afferents

Xiaowei Chen, Derek C. Molliver, and G. F. Gebhart

(see pages 2365–2372)

During most of the day, bladder muscles are relaxed and muscles of the bladder neck and urethra contract. As the bladder fills, its distension causes release of ATP, which stimulates afferent nerves. When the signal is great enough, suprapontine control centers switch urinary system control from sympathetic adrenergic pathways to parasympathetic cholinergic pathways, causing the musculature controlling outflow to relax while bladder muscles contract to effect voiding. ATP has been shown to stimulate afferent nerves via ionotropic P2X receptors. Chen et al. now show that ATP also activates metabotropic G-protein-coupled P2Y receptors and thus increases the sensitivity of dissociated sensory neurons in mice. UTP, which activates P2Y but not P2X receptors, depolarized neuronal resting membrane potential, lowered action potential threshold, and facilitated currents mediated by P2X2 and P2X3 receptors, which were present in a subset of neurons. As a result, subsequent activation of P2X receptors produced more action potentials than stimulation before P2Y activation.