This Week in The Journal

● Cellular/Molecular

Trafficking in GABA$_A$ Receptors in C. elegans

Aaron M. Rowland, Janet E. Richmond, Jason G. Olsen, David H. Hall, and Bruce A. Bamber

(see pages 1711–1720)

As for most synaptic receptors, $GABA_A$ receptors cluster at sites of innervation by GABAergic nerve terminals. In *Caenorhabditis elegans*, postsynaptic muscles receive both acetylcholine and GABA inputs. This week, Rowland et al. controlled whether these excitatory and inhibitory motor neurons reached their targets. To do this, they manipulated expression of the netrin receptor that is necessary for motor neurons to project dorsally and innervate dorsal muscles. When GABAergic inputs were eliminated because of a lack of netrin receptors in GABAergic neurons, postsynaptic $GABA_A$ receptors became diffusely distributed in the muscle membrane. When GABAergic axons reached the muscle, clusters were normal even in the absence of acetylcholine inputs. However, when both inputs were absent, GABA receptors disappeared from the cell surface altogether and were concentrated in intracellular autophagosomes. Acetylcholine receptors were not targeted to these structures. The results suggest that presynaptic input can affect both receptor localization and stabilization in the membrane.

▲ Development/Plasticity/Repair

Reelin/Dab1 Signaling in Cortical Dendritogenesis

Eric C. Olson, Seonhee Kim, and Christopher A. Walsh

(see pages 1767–1775)

Neuronal migration and positioning in the developing mammalian cortex depends on molecules of the Reelin signaling system, including the downstream adapter protein Dab1. This week, Olson et al. suppressed expression of Dab1 in migrating cortical neurons *in vivo* using RNAi. They transfected migrating neurons with a green fluorescent protein expression vector and RNAi plasmids by *in utero* electroporation. Normally, cells migrated through the cell-dense cortical plate (CP) and entered the marginal zone (MZ), where the leading process typically became branched. At embryonic day 20 in lateral neocortex and cingulate cortex, Dab1-deficient neuronal cell bodies were displaced away from the CP/MZ border compared to controls. The leading processes and postnatal dendrite of Dab1-deficient cells failed to contact the MZ and were less branched compared to controls. Reelin/Dab1 signaling may transform the leading process into a dendrite that affects migration and cell positioning.

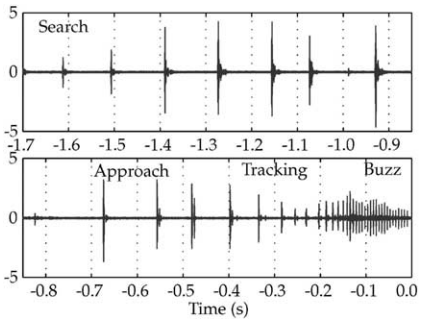
■ Behavioral/Systems/Cognitive

The Flight Plan of the Echolocating Bat

Kaushik Ghose and Cynthia F. Moss

(see pages 1704–1710)

A bat has the advantage of navigating and hunting in the dark, but the complication that echoes of the emitted ultrasonic pulses must be decoded as the animal "gazes" and grazes. This involves continuous conversion of the binaural frequency and times of the echoes into spatial location, thus directing a flight plan. Ghose and Moss examined such sensorimotor integration in the big brown bat, *Eptesicus fuscus*. Infrared cameras and microphones tracked a bat in a dark, empty room as it located and retrieved a suspended mealworm. The sonar pulses vary from 4 Hz in search mode to 150–200 Hz during prey detection. The authors describe a gain factor that links acoustic gaze (sonar direction) to locomotion (flight turn rate) that changed with behavioral state. Much like a visually guided animal, the bat can operate in scan mode without altering its movement direction, but in attack mode, the gain is high, and movement is determined by prey location.



Trains of pulses produced by a bat catching an insect in the laboratory. Capture occurred at time 0. Note the change in pattern and frequency of the sonar pulses during search, pursuit and capture. The small signals following the large amplitude initial pulses represent echoes. See Ghose and Moss for details.

♦ Neurobiology of Disease

A 5-HT$_{1A}$ Polymorphism and Transcriptional Regulation

Margaret Czesak, Sylvie Lemonde, Erica A. Peterson, Anastasia Rogaeva, and Paul R. Albert

(see pages 1864–1871)

Serotonergic neurons of the raphe nuclei express 5-HT_{1A} autoreceptors that inhibit 5-HT release. Neurons that express 5-HT_{1A} receptors are implicated in mood and emotion, and clinical depression has been linked to reduced serotonin activity. For example, serotonin-selective reuptake inhibitors may in part act by desensitizing the autoreceptors on serotonergic neurons, thereby increasing 5-HT output. The expression of these receptors may genetically predispose patients to mood disorders, based on suggestive evidence from a functional polymorphism in the 5-HT_{1A} promoter. This week, Czesak et al. examined the C(-1019)G polymorphism in the 5-HT_{1A} promoter region in a serotonergic cell line RN46A. In serotonergic neurons, the Deaf-1 transcription repressed 5-HT_{1A} expression at the C(-1019) allele, but it enhanced transcription in nonserotonergic cells lines that expressed 5-HT_{1A}. In the G(-1019) allele, both of these actions were blocked. Hes5 repressed transcription in all cell types. These cell- and allele-specific actions of Deaf-1 may contribute to regulation of neuronal 5-HT_{1A} receptors.