This Week in The Journal

● Cellular/Molecular

**Mutated Glial Ca^{2+} Exchanger Underlies Seizure Phenotype**

Jan E. Melom and J. Troy Littleton

(see pages 1169 – 1178)

Seizures are thought to involve reduced activity in inhibitory networks and increased activity in recurrent excitatory networks, often stemming from abnormalities in voltage-sensitive ion channels or GABAergic signaling. Although most research on seizure initiation and propagation focuses on neurons, glia may also have a role. *Drosophila zydeco* mutants undergo seizures when exposed to environmental stressors, and Melom and Littleton have mapped the mutation to an ion exchanger that extrudes cytosolic calcium. Surprisingly, *zydeco* was not expressed in neurons, but was restricted to a glial subtype called cortex glia, which encapsulate neuronal somata. Knockdown of *zydeco* selectively in cortex glia reproduced the seizure phenotype, whereas expressing wild-type *zydeco* in these cells rescued the phenotype. The function of cortex glia is unknown, but they exhibited calcium transients that were often restricted to microdomains surrounding single neurons. Such transients were absent in *zydeco* mutants, which had constitutively high calcium concentrations. The data suggest that disrupted glial calcium regulation underlies seizure susceptibility in *zydeco* mutants.

● Development/Plasticity/Repair

**Some Low-Threshold Mechanoreceptors Are Shaped by Runx1**

Shan Lou, Bo Duan, Linh Vong, Bradford B. Lowell, and Qiufu Ma

(see pages 870 – 882)

Most small-diameter, unmyelinated fibers (c-fibers) have high mechanical thresholds characteristic of nociceptors, but low-threshold mechanosensitive c-fibers (C-LTMRs) also exist. The function of C-LTMRs is unclear: although their low threshold suggests they sense light touch, knocking out the vesicular glutamate transporter they express—VGLUT3—produces abnormal pain responses, suggesting they contribute to nociception. Lou et al. report that VGLUT3 is expressed transiently in multiple subtypes of somatosensory neurons, but persists primarily in C-LTMRs that form lanceolate endings around hairs and in a small group of c-fibers that terminate in the skin. Fibers that persistently expressed VGLUT3 also expressed the transcription factor Runx1, and knocking out Runx1 in these cells prevented formation of lanceolate endings and reduced the number of mechanosensitive neurons in dorsal root ganglion cultures. Nosicuous mechanical, thermal, and chemical stimuli evoked normal pain behaviors in Runx1-null mice, however, suggesting that a different subset of neurons was responsible for the pain phenotype found in VGLUT3-null mice.

● Systems/Circuits

**Chronic Ethanol Increases EPSC Frequency in BNST Neurons**

Yuval Silberman, Robert T. Matthews, and Danny G. Winder

(see pages 950 – 960)

Recovering drug addicts often relapse when exposed to ethanol—a treatment that produces alcohol seeking—increased spontaneous EPSC frequency in VTA-projecting BNST neurons, and this effect was blocked by CRF1 receptor antagonist.

● Behavioral/Cognitive

**Saccadic Suppression Results from Rapid Image Motion**

Michael Dorr and Peter J. Bex

(see pages 1211–1217)

Because visual acuity is much higher in the fovea than in the peripheral retina, primates continually make saccades to view points of interest with high resolution. To generate a stable representation of the world, the brain stitches together the images obtained during fixations and eliminates self-induced motion generated during saccades. Experiments using simple visual stimuli and highly controlled eye movements indicate that visual sensitivity is reduced during saccades. But whether this loss of sensitivity results from active suppression of visual input or simply from an inability to process rapidly changing visual stimuli has been debated. To address this, Dorr and Bex had people view nature videos and tested their ability to detect stimuli presented first during natural saccades, and then as the image was shifted to mimic the previous saccades. Sensitivity decreases were similar for active and passive saccades, indicating that perisaccadic sensitivity loss does not result from active suppression, but rather is a direct consequence of rapid movement of images across the retina.