This Week in The Journal

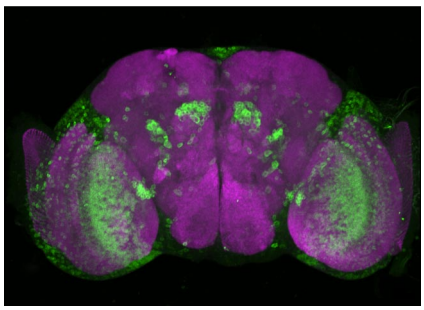
● Cellular/Molecular

Sialyltransferase Knock-out Alters Na^+ Channel Gating in Flies

Elena Repnikova, Kate Koles, Michiko Nakamura, Jared Pitts, Haiwen Li, et al.

(see pages 6466 – 6476)

Sialic acids are sugars that are added to the ends of carbohydrate structures on glycoproteins and glycolipids by sialyltransferases. They are present on all cell types, but are especially abundant in the brain, where they regulate neuronal migration, axon growth, plasticity, and gating of voltage-gated sodium channels. Investigating the role of sialic acids in vertebrates has been somewhat limited, however, by the presence of multiple, redundant sialyltransferases. *Drosophila*, in contrast, have a single sialyltransferase gene (DSiaT), allowing Repnikova et al. to study the effect of sialyltransferase knock-out. Flies lacking *DSiaT* died sooner, crawled more slowly, and were less coordinated than wild-type flies. DSiaT was expressed exclusively in specific interneurons and motor neurons in wild-type flies, and *DSiaT* knock-out reduced terminal arborization, the number of synaptic boutons, and the size of the evoked endplate potential produced by some of these neurons. The authors suggest this resulted in part from alterations in sodium channel gating.



Expression of DSiaT (green) in the fly brain. See the article by Repnikova et al. for details.

▲ Development/Plasticity/Repair

PET Detects Radiolabeled Stem Cells In Vivo

Maria Adele Rueger, Heiko Backes, Maureen Walberer, Bernd Neumaier, Roland Ullrich, et al.

(see pages 6454 – 6460)

Neural stem cells residing in the subventricular zone (SVZ) and dentate gyrus of adults can proliferate and differentiate into multiple cell types. Although the number of stem cells present in adult brains appears to be too small to produce substantial functional recovery after injury, pharmacological treatments might be able to increase the endogenous population and thereby improve outcomes. The development of such treatments would benefit from techniques to quantify the number of stem cells *in vivo*. Rueger et al. have developed such a technique using positron emission tomography (PET) and radiolabeled thymidine. When injected into rat brain, radiolabeled thymidine was taken up by proliferating stem cells, and labeling was significantly above background in SVZ and dentate gyrus. A pharmacological treatment and experimental stroke—both of which were previously shown to expand the stem cell pool—increased the area of thymidine labeling. Subsequent ex vivo staining for bromodeoxyuridine validated the technique.

■ Behavioral/Systems/Cognitive

Learning Increases Synaptic Density in Mushroom Bodies

Benoît Hourcade, Thomas S. Muenz, Jean-Christophe Sandoz, Wolfgang Rössler, and Jean-Marc Devaud

(see pages 6461 – 6465)

The mushroom bodies of insect brains, like mammalian hippocampus, are required for associative learning. Whereas much is known about the structural basis of memory formation in hippocampus, relatively little is known about structural plasticity in mushroom bodies. To address this question, Hourcade et al. conditioned honeybees to discriminate between odors and compared their brains to those of untrained bees. Olfactory input to mushroom bodies is organized into microglomeruli. The density of microglomeruli was greater in honeybees that underwent conditioning than in untrained bees. The increased density appeared to be limited to the olfactory input area of mushroom bodies: no increase occurred in visual input areas. Moreover, honeybees that were injected after the training session with transcriptional inhibitors, which block long-term memory, did not show an increase in microglomerular density. The overall volume of the olfactory area was similar in trained and control bees, suggesting that associative learning resulted in an increase in synaptic density in this structure.

♦ Neurobiology of Disease

BDNF Promotes Epileptogenesis via PLCγ1 Pathway

Xiao Ping He, Enhui Pan, Carla Sciarretta, Liliana Minichiello L, and James O. McNamara

(see pages 6188 – 6196)

Expression of brain-derived neurotrophic factor (BDNF) is elevated in people with temporal lobe epilepsy, and animal studies have shown that BDNF acting on TrkB receptors promotes epileptogenesis. TrkB initiates signaling via Shc adaptor protein and phospholipase C (PLC) γ1 pathways, and activation of each of these pathways requires phosphorylation of distinct sites in TrkB. Mutation of the site required for Shc activation was previously shown not to affect epileptogenesis. He et al. now show that mutation of the PLCγ1 activation site impairs epileptogenesis. In two mouse models of temporal lobe epilepsy—pilocarpine injection and kindling—phosphorylation of PLCγ1 increased in the mossy fiber terminal area in hippocampal CA3 after seizure induction. In knock-in mice expressing mutant *TrkB* that cannot activate PLCγ1, pilocarpine-induced increases in PLCγ1 phosphorylation were eliminated, and more stimulations were required to induce kindling seizure. In addition, long-term potentiation of mossy fiber synapses in CA3 was impaired in knock-in mice.