# This Week in The Journal

#### Cellular/Molecular

Activating EAAT4 Anion Channels
Delany Torres-Salazar and Christoph
Fahlke

(see pages 7513-7522)

The excitatory amino acid transporter 4 (EAAT4) is an unusual beast. Expressed predominantly in cerebellum Purkinje cells rather than glia, the molecule also functions as an anion-selective ion channel. The current model for transport is that a molecule of glutamate is transported independently by each subunit in the homotrimeric transporter. The crystal structure of glutamate transporters suggests that the extracellular face is a water-filled bowl with glutamate binding sites sitting at the base. This week, Torres-Salazar et al. provide data suggesting a cooperative interaction of subunits in the generation of EAAT4 anion currents. The authors expressed EAAT4 in Xenopus oocytes and heterologous cells and reported sigmoidal glutamate concentration dependence. Two mutations close to the glutamate binding site altered the dissociation constant and cooperativity of the anion current, unlike glutamate transport itself. Thus, glutamatebinding sites on different subunits must be occupied to generate the anion current, indicative of intersubunit interactions.

## ▲ Development/Plasticity/Repair

Visual Acuity and Long-Term Monocular Deprivation in Rats Karen Iny, Arnold J. Heynen, Erik Sklar, and Mark F. Bear

(see pages 7368 – 7374)

The detrimental effects of monocular deprivation during early life are among the most studied phenomenon in neuroscience. The resulting reorganization of the visual cortex leaves the deprived eve compromised once it is uncovered. However, this week Inv et al., using rats, show a bidirectional and reversible enhancement of vision in the open (nondeprived) eye. The authors measured visual acuity by training rats to use computer-generated visual cues to escape a swim box. Control rats performed at criterion (≥75% of trials correct) in response to a grating spatial frequency of 0.89 cycles per degree. After 5 months of monocular deprivation, rats suffered severe loss of acuity in the deprived eye. The threshold, measurable 3-4 weeks after uncovering, was 0.20 cycles per degree and then improved further. However, acuity in the nondeprived eye acuity was enhanced to 1.20 cycles per degree before declining gradually to normal. Opening the deprived eye led to partial reversal, indicating that this plasticity persists into adulthood.

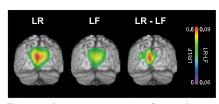
## ■ Behavioral/Systems/Cognitive

Tracking Memory Operations with MEG

Daria Osipova, Atsuko Takashima, Robert Oostenveld, Guillén Fernández, Eric Maris, and Ole Jensen

(see pages 7523–7531)

This week, Osipova et al. made use of the high temporal and spatial resolution of magnetoencephalography (MEG) to examine oscillatory activity associated with a memory task. The authors presented subjects with a series of pictures of buildings or landscapes in an encoding session. Then subjects were asked in a retrieval session whether they had previously viewed the images. During encoding, gamma (60-90 Hz) and theta (4.5–8.5 Hz) activity was stronger for items that were later remembered than for those that were later forgotten. Likewise, in the retrieval session, gamma and theta activity were stronger for recognized items than for correctly rejected



The images show source reconstruction of averaged gamma activity across subjects during the encoding session. The sources for later remembered (LR) and later forgotten (LF) conditions, and their difference (LR  $\,-$  LF), were located in Brodmann areas 18 and 19. See Osipova et al. for details.

items. Theta activity originated in parietotemporal areas, whereas gamma activity arose occipitally in Brodmann areas 18 and 19. The authors suggest that neural synchronization represented by occipital gamma activity may reflect either stronger drive to areas involved in memory or to reinforcement from those same areas.

#### ♦ Neurobiology of Disease

EGFR and Reactive Astrocytes

Bin Liu, Huiyi Chen, Terrance G. Johns, and Arthur H. Neufeld

(see pages 7532–7540)

In this week's Journal, Liu et al. provide some insight on what makes a "reactive" astrocyte reactive. The authors look at activated astrocytes using several models of optic nerve injury. The epidermal growth factor receptor (EGFR) was not expressed by quiescent astrocytes but was upregulated in rat optic nerve astrocytes after ischemia, nerve transection, and in a chronic glaucoma model. The authors used several approaches to examine EGFR signaling in astrocytes including microarray analysis and the analysis of motility of EGF-treated cultured astrocytes. Rats in a chronic glaucoma model were treated with the EGFR tyrosine kinase inhibitor AG1478 for 7 months. Untreated rats lost up to 20% of their retinal ganglion cells, whereas treated rats did not. In rats that went untreated for 3.5 months and then received AG1478, cell death was halted. The authors suggest that EGFR activation of astrocytes may play a detrimental role after injury.