# *This Week in The Journal*

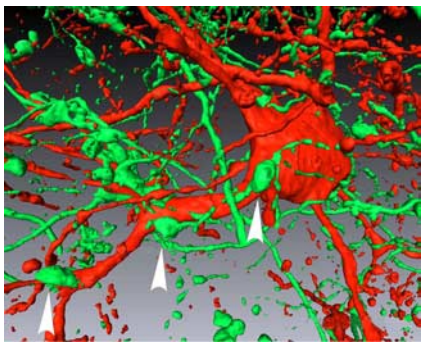
## ● Cellular/Molecular

### *Synaptic Properties of Corticothalamic Synapses*

Alexander Groh, Christiaan P. J. de Kock, Verena C. Wimmer, Bert Sakmann, and Thomas Kuner

(see pages 9652–9663)

In rodents, neurons in layer 5 of somatosensory (barrel) cortex project to, and form giant terminals on, neurons in the posterior medial thalamus. With such long-range connections, identifying synaptically coupled neurons in which to study synaptic properties is difficult. Groh et al. cleverly overcame this problem by virally expressing a fluorescent protein in rat cortical neurons, which enabled visualization of giant terminals in corticothalamic slice cultures. Whole-cell patches were used both to fill postsynaptic neurons with dye and to record responses to extracellular stimulation of presynaptic terminals. Single presynaptic spikes produced large EPSCs and triggered multiple action potentials in postsynaptic cells. But the spikes also produced rapid short-term depression, so subsequent stimuli did not generate postsynaptic action potentials unless the stimulus frequency was $<2$ Hz. Therefore, stimuli that mimicked spontaneous activity recorded *in vivo* (typically 4–6 Hz) elicited postsynaptic spikes only after occasional periods of silence or when two presynaptic terminals were stimulated simultaneously.



Simultaneous expression of mOrange dye in thalamic neurons (red) and GFP in axons and endings of cortical L5B and L6 neurons (green) reveals presynaptic giant terminals (arrowheads) and postsynaptic dendrites. See the article by Groh et al. for details.

## ▲ Development/Plasticity/Repair

### *TGF-β1, a Schwann-Cell-Derived Synaptogenic Molecule*

Zhihua Feng and Chien-Ping Ko

(see pages 9599–9609)

Extensive research on the neuromuscular junction has produced a detailed understanding of the interactions that occur between neurons and muscles during synaptic development. One essential step in synaptogenesis is secretion of agrin by nerve terminals, which induces clustering of acetylcholine receptors (AChRs) in the muscle. Nonmyelinating, perisynaptic Schwann cells also contribute to formation of the neuromuscular junction, by guiding axonal growth and by secreting another factor that promotes AChR clustering. This week, Feng and Ko identify the Schwann-cell-derived synaptogenic factor as transforming growth factor β1 (TGF-β1). In frog nerve–muscle cocultures, TGF-β1 replicated the ability of Schwann-cell-conditioned media to increase AChR clustering in muscles. Furthermore, depletion of TGF-β1 abolished the synaptogenic effect of the conditioned media. TGF-β1 did not induce AChR clustering in pure muscle cultures—which is unsurprising, because muscle lacks TGF-β1 receptors. Instead, TGF-β1 appears to exert its synaptogenic effect by inducing agrin secretion by the growing nerve.

## ■ Behavioral/Systems/Cognitive

### *Precise and Imprecise Olfactory Coding*

Derek J. Hoare, Catherine R. McCrohan, and Matthew Cobb

(see pages 9710–9722)

How olfactory information is processed by the nervous system remains a mystery. *Drosophila* larvae provide a good model system for studying olfactory processing, because they have only 25 odor receptor (OR) genes and 21 pairs of olfactory sensory neurons (OSNs), and *Drosophila* OSNs, like those in other organisms, express a single OR gene. It has long been assumed that OSNs respond in a characteristic way to particular odors. To test this hypothesis, Hoare et al. generated several strains of *Drosophila* larvae that each expressed a single functional OSN. As expected, most OSNs responded in a qualitatively consistent, characteristic manner (excitation, inhibition, or no response) to different odors. Surprisingly, however, ~20% of OSNs had variable responses to particular odors, responding only sometimes, independent of concentration or duration of the stimulus. Such stochastic responses were also detected in wild-type larvae. How such stochastic coding might aid olfactory discrimination is a new puzzle to be solved.

## ◆ Neurobiology of Disease

### *Damage Response in the Retinal Pigment Epithelium*

Amir Rattner, Leila Toulabi, John Williams, Huimin Yu, and Jeremy Nathans

(see pages 9880–9884)

The retinal pigment epithelium (RPE), which lies adjacent to the outer segments of photoreceptors, is essential for retinal health and function, providing nutrients, maintaining ionic balance, and converting all-*trans* retinol back to 11-*cis* retinal during the visual cycle. Local degradation of the RPE is a common cause of vision loss in age-related macular degeneration, and mutations in RPE proteins can lead to retinal degeneration and congenital blindness. To understand the changes that occur in the RPE in response to damage, Rattner et al. examined gene expression changes that occur following bright light exposure and retinal detachment. These two types of damage produced largely overlapping patterns of gene expression changes. Notably, genomic changes in the RPE appeared to be triggered by a signal from the retina, because the changes did not occur in an *ex vivo* preparation after the retina had been removed.