This Week in The Journal

● Cellular/Molecular

Mitral Cells Receive Direct and Indirect Input from OSNs

Marion Najac, Didier De Saint Jan, Leire Reguero, Pedro Grandes, and Serge Charpak

(see pages 8722–8729)

Olfactory sensory neurons (OSNs) transmit information to glomerular tufted and mitral cells and juxtaglomerular external tufted cells in the olfactory bulb. Surprisingly, whether OSNs synapse directly onto mitral cells or excite them indirectly remains uncertain. Najac et al. provide evidence that mitral cells receive both direct and indirect input from OSNs. Electrically stimulating axons converging on a glomerulus elicited biphasic EPSCs in mitral cells. The fast component of EPSCs occurred with short, constant latency similar to that of EPSCs in nearby external tufted cells, suggesting that it was monosynaptic. Both components were blocked by AMPA receptor antagonist, and when mitral cells were held at positive membrane potentials, OSN stimulation elicited biphasic outward current, suggesting that both components of the response were mediated by glutamatergic synapses rather than electrical coupling. Stimulation of fewer OSN axons often evoked only a slow, variable-latency EPSC in mitral cells. This component was likely mediated by feedforward excitation from nearby external tufted cells.

▲ Development/Plasticity/Repair

Astrocyte Ca^{2+} Elevation Requires Pannexin Hemichannels

Yann Bernardinelli, Chris Salmon, Emma V. Jones, W. Todd Farmer, David Stellwagen, et al.

(see pages 8905–8919)

Astrocytes regulate neuronal activity by removing or limiting the diffusion of extracellular ions and neurotransmitters and by releasing gliotransmitters. Neuronal activity promotes Ca^{2+} release from astrocytic intracellular stores, which triggers release of gliotransmitters that can excite multiple nearby neurons. Ca^{2+} signaling spreads between astrocytes via gap junctions, creating a network that can transmit information through a neuronal population. Little is known about how activity patterns evolve in neuron–astrocyte networks, however. To visualize these patterns, Bernardinelli et al. photostimulated channelrhodopsin-expressing neurons in mouse hippocampal slice cultures and monitored astrocytic Ca^{2+} with a fluorescent indicator. Spike trains caused Ca^{2+} elevation in subsets of nearby astrocytes, primarily those surrounding the proximal apical dendrite. Blocking synaptic vesicle release did not affect Ca^{2+} responses, but blocking action potentials or pannexin hemichannels—which are thought to mediate nonsynaptic release of glutamate and ATP from dendrites—prevented astrocytic Ca^{2+} elevation, suggesting that it is evoked by molecules released following backpropagating action potentials.

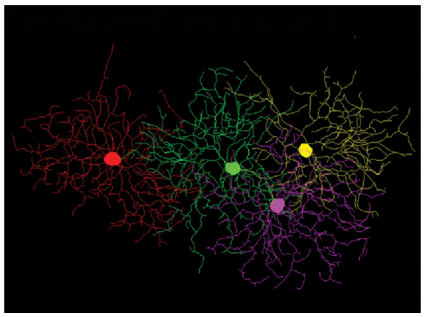
■ Behavioral/Systems/Cognitive

Receptor Expression Distinguishes Posterior-Motion-Tuned RGCs

Michal Rivlin-Etzion, Kaili Zhou, Wei Wei, Justin Elstrott, Phong L. Nguyen, et al.

(see pages 8760–8769)

The retina contains a remarkable number of cell types. Each of the major cell classes has multiple subtypes, which usually can be further subcategorized. Retinal ganglion cells (RGCs) are broadly classified as On-center, Off-center, or On-Off, and further divided by morphology, projection pattern, and functional properties, such as direction sensitivity. Direction-selective RGCs are subcategorized as anterior, posterior, superior, or inferior motion preferring. The advent of genetic tools for labeling specific cell types has facilitated examination of unique electrophysiological properties and has led to the discovery of additional subtypes. Rivlin-Etzion et al. report that RGCs that express thyrotropin-releasing hormone receptor (TRHR) are On-Off cells that respond preferentially to posterior motion, but they are distinct from posterior-tuned On-Off cells that express dopamine receptor D4 (DRD4). The former respond to a broader range of directions than the latter, and they have distinct axonal projections, most notably one to the zona incerta in the forebrain.



Four neighboring TRHR-expressing RGCs. See the article by Rivlin-Etzion et al. for details.

♦ Neurobiology of Disease

Tic-Associated Activity in GPi Suggests It Does Not Drive Movement

Maya Bronfeld, Katya Belelovsky, and Izhar Bar-Gad

(see pages 8713–8721)

Motor tics are brief, involuntary muscle contractions that occur in several neurological conditions. Tics are probably produced by abnormal activity in circuits involving cortex and basal ganglia, but their site of origin remains controversial. To address this question, Bronfeld et al. induced orofacial tics in primates by injecting GABA antagonist into the dorsal putamen, the input structure of the basal ganglia, and recording activity in other areas. Tic-related bursting occurred in presumptive medium spiny neurons (MSNs) in the ventral putamen, and activity modulation was recorded throughout the globus pallidus externus (GPe), GP internus (GPi) (the main motor output nucleus of the basal ganglia), and in primary motor cortex (M1). Tic-related activity in presumptive MSNs generally preceded modulations in M1, which usually preceded those in GPe and GPi. The relative timing and lack of regional specificity of pallidal modulation suggests that, contrary to some models, GPi does not initiate tics or determine which muscles they involve.