This Week in The Journal

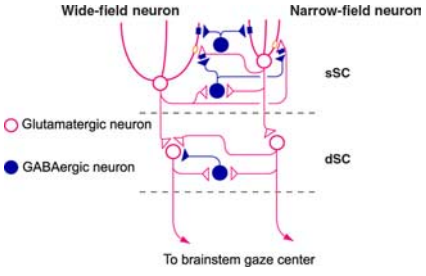
● Cellular/Molecular

Regulation of Burst Duration in the Superior Colliculus

Katsuyuki Kaneda, Penphimon Phongphanphanee, Tomoko Katoh, Kaoru Isa, Yuchio Yanagawa, Kunihiko Obata, and Tadashi Isa

(see pages 816 – 827)

The superior colliculus (SC) regulates orienting behaviors, such as saccades. The superficial layers of the SC (sSC) receive inputs from the retina and visual cortex, whereas the deeper layers of the SC (dSC) send motor commands to the brainstem and spinal cord. Bursts of high-frequency action potentials in dSC trigger express saccades, and these bursts are regulated by inputs from the sSC. To further detail the functional circuitry of the SC, Kaneda et al. examined the role of $GABA_B$ receptors in controlling burst duration. By bath- or puff-applying receptor blockers to mouse SC slices while obtaining whole-cell recordings, they found that high-frequency firing of glutamatergic sSC neurons is likely to cause substantial release of GABA from local GABAergic sSC neurons. GABA then acts presynaptically and postsynaptically on the glutamatergic neurons to limit burst duration of sSC neurons, which in turn determines the burst duration of target neurons in the dSC (see figure).



Simplified schematic of the superior colliculus circuitry that produces express saccades. See the article by Kaneda et al. for details.

▲ Development/Plasticity/Repair

Synaptic Plasticity in the Developing Cerebellum

Laurens W. J. Bosman, Hajime Takechi, Jana Hartmann, Jens Eilers, and Arthur Konnerth

(see pages 798 – 807)

This week, Bosman et al. describe a new form of synaptic plasticity that occurs during development of climbing fiber (CF) inputs to cerebellar Purkinje cells (PCs). In adults, a single CF innervates each PC, whereas during early development, multiple CFs do. To study the changes that occur during synapse elimination in rats, Bosman et al. recorded from PCs while stimulating CFs extracellularly in cerebellar slices. At postnatal day 6, PCs received inputs from multiple CFs, but one CF produced much larger excitatory postsynaptic currents than the others. When high-frequency stimulation of this "strong" CF was paired with depolarization of the PC, NMDA-independent long-term potentiation (LTP) was produced. LTP was also elicited when stimulation of the strong CF was paired with intracellular current injection that produced a normal firing pattern in the PC. Remarkably, stimulating weaker CFs with this same protocol resulted in long-term depression of their synapses.

■ Behavioral/Systems/Cognitive

The Role of Spinal Neurons That Express μ-Opioid Receptors

Robert H. Kline IV and Ronald G. Wiley

(see pages 904 –913)

The effects of opiates are well known in and outside the laboratory, but the function of spinal neurons that express μ-opioid receptors (MORs) is unclear. Studying the function of these neurons is difficult because MORs are expressed presynaptically on nociceptive afferents, as well as on the postsynaptic dorsal horn neurons. To circumvent this obstacle and thus to distinguish the function of presynaptic and postsynaptic MORs in the spinal cord, Kline et al. intrathecally injected rats with a μ-opioid peptide (dermorphin) coupled to a toxin (saporin). This novel technique selectively destroyed many MOR-expressing spinal neurons but spared the nociceptive afferents. Dermorphin–saporin injection did not affect rats' behavioral responses to transient thermal pain, but it increased their response to persistent chemical pain. Dermorphin–saporin injection attenuated the ability of morphine to reduce behavioral responses to both thermal and chemical pain, indicating an essential role for MOR-expressing dorsal horn neurons in the antinociceptive effect of morphine.

♦ Neurobiology of Disease

Suppression of P/Q Calcium Currents by Amyloid β

Volker Nimmrich, Christiane Grimm, Andreas Draguhn, Stefan Barghorn, Alexander Lehmann, Hans Schoemaker, Heinz Hillen, Gerhard Gross, Ulrich Ebert, and Claus Bruehl

(see pages 788 –797)

A possible target for treating the cognitive decline in Alzheimer's disease (AD) patients is identified by Nimmrich et al. in this issue: P/Q calcium channels. Because soluble forms of amyloid β (Aβ) correlate with the degree of cognitive decline in AD patients, the authors applied one such form, $A\beta_{1-42}$ globulomer, to cultured rat hippocampal neurons and measured the effects on synaptic events and ionic currents. Doses as low as 8 nM rapidly reduced the frequency and amplitude of both glutamatergic and GABAergic spontaneous postsynaptic currents (PSCs). The frequency of miniature PSCs was also reduced. By using a variety of channel blockers, the authors demonstrated that $A\beta_{1-42}$ globulomer reduces the current through P/Q channels, which are required for neurotransmitter release. Most promisingly, the effect, and the associated decrease in the frequency of PSCs, was partially reversed by application of roscovitine, which enhances P/Q calcium currents by slowing deactivation of the channel.