This Week in The Journal

● Cellular/Molecular

A Cool Receptor in the Spinal Cord?

Kenzo Tsuzuki, Hong Xing, Jennifer Ling, and Jianguo G. Gu
(see pages 762–771)

The recently cloned cold- and menthol-sensitive receptor, TRPM8, is a member of the large and diverse transient receptor potential (TRP) family. This nonselective cation channel is permeable to calcium and is expressed in some primary afferent neurons. One might expect that the cold receptor would be expressed primarily on peripheral nerve endings. However Tsuzuki et al. report a possible central action of the cold receptor. They examined synapses in cocultured DRG and dorsal horn (DH) neurons. Both cooling and menthol application increased the frequency of miniature EPSCs without affecting the amplitude. Menthol also enhanced evoked release from DRG neurons, consistent with a presynaptic site of action. These actions appeared to depend on intracellular calcium stores but not on extracellular calcium or conventional intracellular signaling pathways. Thus the authors propose that the cold receptor is expressed on intracellular membranes causing direct release of calcium stores.

▲ Development/Plasticity/Repair

Maternal Separation, Fear, and the Amygdala

M. D. Bauman, P. Lavenex, W. A. Mason, J. P. Capitanio, and D. G. Amaral
(see pages 711–721)

The amygdala has a well accepted role in fear-related behaviors. However its role in social interactions is not as clear. Bauman et al. test the relationship of fear and social interactions in an interesting situation: the relationship between mother and infant. They studied infant macaque monkeys after bilateral lesions of the amygdala or hippocampus. Amygdala-lesioned monkeys had increased physical contact time with their mothers, but otherwise showed normal maternal interactions. However after weaning at 6 months, amygdala-lesioned monkeys did not seek out their mothers or display distress signals in a "maternal preference test." This test seemingly measures the equivalent of separation anxiety familiar to human moms. The authors attribute the disrupted behavior as an impaired ability to perceive danger. The results suggest that the amygdala is not essential for the development of social behavior; rather, it mediates responses to dangerous or fear-provoking situations.

■ Behavioral/Systems/Cognitive

Nociceptive Inputs to Rat Cortex

Caroline Gauriau and Jean-François Bernard
(see pages 752–761)

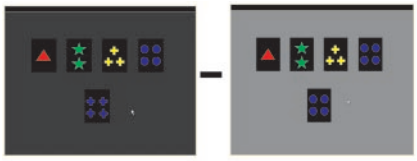
The thalamic and cortical pain-processing areas are not as well mapped as in other sensory systems. Several cortical areas are activated by pain, but their thalamic connections are still in question. In this issue, Gauriau and Bernard take a look at the posterior triangular thalamus (PoT) as a potential relay between nociceptive sensory neurons in lamina 1 of the spinal cord and the cortex. The authors recorded the sensory modalities of rat PoT neurons using extracellular recording. Approximately one-half of the neurons responded to tactile or nociceptive stimulation. Of these, one-half were nociceptive-specific (NS), whereas the other half responded to both stimuli [nociceptive nonspecific (NNS)] or only to tactile stimuli. The cells were then labeled with biotin–dextran to map their cortical projections. Interestingly, the pain-processing neurons innervated different sites according to their sensory fingerprint. NNS and tactile-responsive neurons projected primarily to the insular cortex and amygdala, whereas NS neurons terminated exclusively in somatosensory cortex S2. The results suggest distinct cortical components in pain processing.

◆ Neurobiology of Disease

Probing Cognitive Deficits in Parkinson's Disease

Oury Monchi, Michael Petrides, Julien Doyon, Ronald B. Postuma, Keith Worsley, and Alain Dagher
(see pages 702–710)

Among the cognitive deficits that accompany Parkinson's disease (PD) are difficulties in "set shifting," or adjusting one's behavior to match changing circumstances. This deficit can be viewed as a slowing of mental processes analogous to the slow initiation of movement (bradykinesia) that is one of the hallmarks of PD. Similar set-shifting problems also occur in patients with lesions of the prefrontal cortex (PFC), as detected by the Wisconsin Card Sorting Task (WCST). This task requires subjects to sort objects according to constantly changing criteria. In this week's *Journal*, Monchi et al. used functional magnetic resonance imaging to examine PFC activity during the set-shifting task. Negative feedback or matching after negative feedback on the WCST caused coactivation of the striatum and specific regions of the PFC. This localized PFC activation was decreased in PD, suggesting that depletion of nigrostriatal dopamine may be responsible for the set-shifting deficit. Other subregions of the PFC actually showed increased activity during WCST in PD subjects, which the authors attribute to decreases in intracortical dopamine.



In the WCST, the subject is asked to match test cards (bottom) to reference cards (4 top cards) according to one of three rules (color, number, shape). An unannounced change in the classification requires set shifting. See the article by Monchi et al. for details.