This Week in The Journal

Cellular/Molecular

Spiking Reduces IPSP Amplitude in Neurogliaform Cells

Gengyu Li, Robert Stewart, Marco Canepari, and Marco Capogna

(see pages 1280–1292)

The hippocampus contains more than 20 types of inhibitory interneurons that express different proteins and impinge on different regions of pyramidal cells to regulate spatiotemporal integration of EPSPs and define temporal windows for spiking. Neurogliaform cells (NGFCs) form synapses on the distal tufts of pyramidal cell apical dendrites alongside excitatory inputs from the entorhinal cortex. NGFCs express neuronal nitric oxide synthase (nNOS), are often synaptically coupled, and fire during theta oscillations *in vivo*. Li et al. found that when theta-associated activity patterns were evoked in NGFCs in rat hippocampal slices, the cells showed a transient reduction in unitary IPSP amplitude. This "firing-induced suppression of inhibition" (FSI) required back-propagation of action potentials, calcium influx through L-type calcium channels, nNOS activity, and activation of NO-sensitive guanylyl cyclase (NO-sGC) receptors, which are present on presynaptic terminals. FSI also indirectly increased the amplitude of EPSPs. Thus FSI may enhance spatial and temporal summation of excitatory inputs to NGFCs, thus regulating their inhibition of pyramidal cells.

Development/Plasticity/Repair

LTP at Inhibitory Synapses Blocks LTP at Excitatory Synapses

Lang Wang and Arianna Maffei

(see pages 1083–1093)

Long-term potentiation (LTP) can occur at both excitatory (e) and inhibitory (i) synapses, but far less is known about the mechanisms and functions of LTPi than of LTPe. Wang and Maffei have begun to close this gap. Fast-spiking inhibitory interneurons (FSIs) contact many pyramidal cells in layer IV of rat primary visual cortex, but FSI bursting induced LTPi only at the synapses where the postsynaptic cell was also depolarized. LTPi required activation of $GABA_B$ receptors and $G_{i/o}$ proteins, and it was not associated with a change in the paired-pulse ratio, suggesting it occurred by postsynaptic mechanisms. But LTPi did not require postsynaptic calcium, metabotropic glutamate receptors, or phosphatases. Interestingly, LTPi prevented coincident induction of LTPe onto the postsynaptic neuron. In fact, stimulation that normally elicited LTPe produced long-term depression (LTDe) when delivered within a few seconds of LTPi induction. Furthermore, monocular deprivation induced LTPi and caused LTPe-inducing stimuli to produce LTDe, suggesting that LTPi-induced suppression of LTPe has physiological roles.

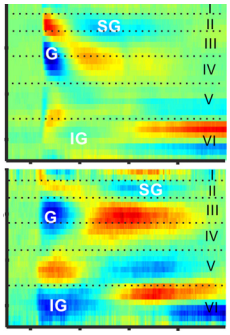
Systems/Circuits

Dopamine Prolongs Corticoefferent Feedback in Auditory Cortex

Max F. K. Happel, Matthias Deliano, Juliane Handschuh, and Frank W. Ohl

(see pages 1234–1247)

Dopamine modulates neural circuits throughout the brain and has important roles in motor control, reward, attention, and learning. In the primary auditory cortex (A1), dopamine is required for long-term memory formation. To investigate how dopamine modulates auditory circuits, Happel et al. recorded extracellularly in gerbil A1 and used current-source-density (CSD) analyses to compare synaptic activation patterns evoked by auditory stimulation in the presence and absence of a D1/D5 dopamine receptor agonist. These analyses suggested that activation of D1/D5 receptors, which are expressed primarily in the infragranular (corticoefferent) layers, increased the amplitude of synaptic currents in this layer, prolonged activity in granular (thalamocortical input) layers, and subsequently increased synaptic currents involving horizontal (corticocortical) inputs. Analyses of activity patterns evoked by intracortical microstimulation in different layers before and after local polysynaptic connections were silenced indicated that the sustained activity in granular layers likely stemmed from enhanced positive recurrent feedback from infragranular layers relayed polysynaptically through areas outside A1, most likely the thalamus.



CSD profiles showing time course (250 ms) of activity in A1 evoked by an auditory stimulus in the absence (top) or presence (bottom) of dopamine D1/D5 receptor agonist. Cortical sinks (blue) reflect synaptic current flow. See the article by Happel et al. for details.

Behavioral/Cognitive

Required and Actual Actions Are Encoded Independently

Lucie Charles, Jean-Rémi King, and Stanislas Dehaene

(see pages 1158–1170)

When attempting to act as quickly as possible, people frequently make mistakes. Often one realizes a mistake was made before receiving any feedback. To explain this phenomenon, Charles et al. propose that two processing streams tie sensory input to motor output: a rapid, unconscious pathway and a slower, more accurate conscious pathway. When speed is essential, the unconscious pathway commands action before the conscious pathway is completed. To test this model, the authors obtained magnetoencephalographic (MEG) and electroencephalographic (EEG) recordings from people who quickly assessed variably masked stimuli, then asked whether multivariate pattern analysis could identify neural activity patterns representing stimulus positions, participants' responses, required responses, and response accuracy. When participants reported seeing the stimulus, all four components could be decoded, indicating that the required response was discernable in neural activity even when the wrong response was made. In contrast, when stimuli were successfully masked (subjects did not consciously perceive them) representations of required response and response accuracy could not be discerned.