This Week in The Journal

● Cellular/Molecular

An Anti-Apoptotic Action of Glia Lipoproteins

Hideki Hayashi, Robert B. Campenot, Dennis E. Vance, and Jean E. Vance

(see pages 1933–1941)

Apolipoprotein E (apoE), an apolipoprotein contained in lipoproteins secreted by CNS glia, is perhaps best known because its ε4 allele is a risk factor for late-onset Alzheimer's disease. This week, Hayashi et al. outline a signaling pathway by which these apoE-containing lipoproteins can inhibit apoptosis. The authors induced apoptosis in cultured rat retinal ganglion cells by removing trophic factors from the media. Glia-conditioned media or glia-secreted lipoproteins protected neurons from apoptosis. Protection specifically required apoE and was blocked either by receptor-associate protein, which competes with ligands of low-density lipoprotein receptors (LDLrs), or by inhibition of the apoE receptor LDLr-related protein (LRP). Downstream of LRP, signaling involved protein kinase Cδ, possibly by phosphorylating and inactivating the pro-apoptotic glycogen synthase kinase-3β. ApoE3 was more effective in preventing apoptosis than apoE4.

▲ Development/Plasticity/Repair

Myelination and Remyelination in the Pregnant Mouse

Christopher Gregg, Viktor Shikar, Peter Larsen, Gloria Mak, Andrew Chojnacki, V. Wee Yong, and Samuel Weiss

(see pages 1812–1823)

This week, Gregg et al. report that pregnancy in rats increased myelin-forming oligodendrocytes and myelin formation, an effect the authors attribute to prolactin. In corpus callosum and spinal cord, oligodendrocyte precursor cells (OPCs) increased in early pregnancy, followed by an increase in oligodendrocytes. Postpartum, pregnancy-generated oligodendrocytes extended processes. There was also an associated increase in myelination of callosal axons. To test the effects of pregnancy on the repair of demyelinating lesions, the authors injected lysolecithin into the spinal cord. Pregnant rats displayed smaller lesions, fewer demyelinated axons, and more remyelinated axons. The prolactin (PRL) receptor, expressed by OPCs, was required for pregnancy-induced OPC proliferation. Furthermore, exogenous PRL administered to virgin rats increased OPC generation and remyelination similar to pregnant rats. The authors suggest that such changes might affect axon function and/or remyelination in situations such as multiple sclerosis.

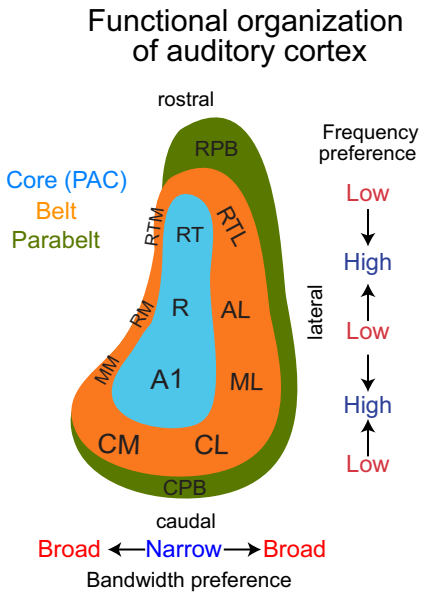
■ Behavioral/Systems/Cognitive

Multisensory Modulation of Auditory Processing

Christoph Kayser, Christopher I. Petkov, Mark Augath, and Nikos K. Logothetis

(see pages 1824–1835)

Putting it all together when it comes to combining information from our senses is usually attributed to "higher" centers in association cortex, yet recent studies suggest that multisensory integration can also occur in primary sensory cortex. This week, Kayser et al. used functional magnetic resonance imaging in macaques to examine visual modulation of auditory receptive fields. The authors used sounds of various frequency and bandwidth to stimulate "core" (primary auditory) and "belt" (nonprimary auditory) fields. In anesthetized animals, most fields were activated only by auditory stimuli, but some areas responded just to visual stimuli. Combined auditory and visual stimuli produced enhanced responses particularly in caudal fields. In awake animals, too, visual and auditory stimuli activated overlapping fields including those in the core. Visual activation and audiovisual enhancement also occurred in the caudal parts of the so-called parabelt. Characteristic of sensory integration, enhancement was stronger for less effective stimuli.



The functional organization of the auditory cortex is shown in this schematic with the core, belt, and parabelt regions (blue, orange, and green, respectively). The differential preferences for sound frequency and bandwidth were used to obtain a functional parcellation of auditory fields. See the article by Kayser et al. for details.

♦ Neurobiology of Disease

Reelin and the Hippocampus, after Status Epilepticus

Chao Gong, Tsu-Wei Wang, Holly S. Huang, and Jack M. Parent

(see pages 1803–1811)

Temporal lobe epilepsy in humans is associated with abnormal lamination of the dentate gyrus and ectopic hilar location of some dentate granule cells (DGCs). Similar changes are seen in rodent models of hippocampal epilepsy. This week, Gong et al. provide evidence that this abnormal distribution of DGCs results from deficient signaling by the secreted guidance factor Reelin. Reelin immunoreactivity in the dentate gyrus was reduced after pilocarpine-induced status epilepticus in adult rats. The authors ascribed this loss to the death of Reelin-expressing inhibitory interneurons, a principal source of Reelin in the adult. In mouse dentate gyrus explants cultured in a gel matrix, progenitor cells migrated in chains. Exogenous Reelin caused cell detachment and disruption of the migratory chains, consistent with a direct effect of Reelin on the migration of DGC progenitors.