# *This Week in The Journal*

## ● Cellular/Molecular

### *Dancing with the Astrocytes*

Hideko Nishida and Shigeo Okabe

(see pages 331–340)

Time-lapse imaging has revealed that virtually all aspects of neuronal migration and synaptic function involve dynamic interactions with surrounding glial processes. This week, Nishida et al. looked at the role of astrocyte-neuronal contact in the stabilization and maturation of dendritic spines. The authors used two-photon time-lapse imaging of cultured hippocampal slices to visualize fluorescently labeled dendrites and green fluorescent protein-expressing astrocytes. Dendritic protrusions stabilized after making contact with astrocytes, such that protrusion lifetimes were longer if contact occurred. Viral expression of a mutant Rac1 decreased astrocyte motility and increased the length of dendritic filopodia but not their motility or lifetimes. Viral expression of chimeric molecules interfered with the association between the dendritic EphA4 receptor and its astrocytic ephrin-A3 ligand and shortened the lifetime of protrusions that contacted astrocytes. The results are consistent with contact-dependent signaling between dendritic protrusions and astrocyte processes during the formation of dendritic spines.

## ▲ Development/Plasticity/Repair

### *Guiding GnRH-1 Neurons*

Paolo Giacobini, Andrea Messina, Susan Wray, Costanza Giampietro, Tiziana Crepaldi, Peter Carmeliet, and Aldo Fasolo

(see pages 431–445)

Gonadotropin hormone-releasing hormone-1 (GnRH-1) neurons begin life with a long trek from their birthplace in the nasal placode along olfactory nerve tracts to their permanent home in the hypothalamus. This week, Giacobini et al. make a case for the cytokine hepatocyte growth factor (HGF) as a guidance molecule for these neurons. Nasal explant cultures expressed HGF and its receptor Met in a spatiotemporal pattern that fitted GnRH-1 neuronal migration *in vivo*. Tissue-type plasminogen activator (tPA), which cleaves and activates pro-HGF, was also expressed by migrating GnRH-1 neurons. In explants treated with an HGF-neutralizing antibody, the number of GnRH-1 neurons was unaffected, but migration was considerably restricted. Likewise, treatment with exogenous HGF expanded the migration pattern in a manner that was sensitive to the gradient created by exogenous HGF. In mice lacking tPA, the adult GnRH-1 neuron population was reduced by about one-third, perhaps indicative of a migratory failure.

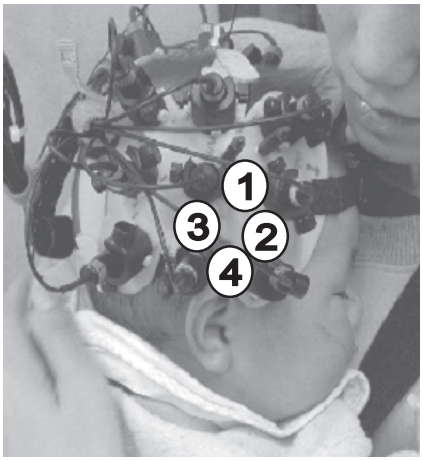
## ■ Behavioral/Systems/Cognitive

### *Japanese Infants Listening to Native Vowel Contrasts*

Yasuyo Minagawa-Kawai, Koichi Mori, Nozomi Naoi, and Shozo Kojima

(see pages 315–321)

Human acoustic processing becomes attuned to the sounds of our native language within the first year of life. Minagawa-Kawai et al. used near-infrared spectroscopy (NIRS) to track acoustic processing in Japanese infants. Subjects were gently fitted with headgear containing near-infrared laser emission and detection probe arrays over the lateral auditory areas. Absorption of light by hemoglobin was used to estimate blood flow changes, providing a spatial resolution of 2–3 cm. NIRS measures only small vessels and thus may be less sensitive to systemic circulatory changes than functional magnetic resonance imaging. The stimuli consisted of two pairs of speech sounds or phonemes, in this case Japanese vowel sounds. The pairs had identical physical structure, but only one contained linguistic information. At 6 months, infants showed phonemic specificity, but this was no longer apparent by 10–11 months. After 12 months, the language specificity was again detectable but now was left lateralized, a pattern similar to adults.




Emission and detection probes were fitted in a 2 × 2 square lattice on the lateral side of the head for near-infrared spectroscopy measurement of auditory responses. See the article by Minagawa-Kawai et al. for details.

## ◆ Neurobiology of Disease

### *Mitochondrial Trafficking and CMT2 Neuropathy*

Robert H. Baloh, Robert E. Schmidt, Alan Pestronk, and Jeffrey Milbrandt

(see pages 422–430)

Heriditary neuropathies come in many shapes and sizes. Charcot-Marie-Tooth type 2 (CMT2) causes degeneration of peripheral sensory and motor neurons, particularly at the ends of these long axons. Many cases of CMT2 arise from mutations in a mitochondrial fusion protein mitofusin 2 (MFN2). This week, Baloh et al. propose that MFN2 mutations cause disease by altering mitochondrial trafficking. The authors expressed mutant MFN2 in cultured dorsal root ganglion neurons. Fragmented mitochondria clustered in cell bodies and proximal axons of these neurons. Time-lapse fluorescence imaging revealed disrupted mitochondrial trafficking, although ATP production and mitochondrial oxidative function remained intact. Likewise, oxidative activity was not disrupted in a muscle biopsy specimen from a patient with CMT2A. The transport failure may result from reduced attachment of mitochondria to the microtubule transport apparatus, thus limiting delivery of energy supplies to long axons.