This Week in The Journal

Development/Plasticity/Repair

Mitochondria Limit Dendritic Branching

Toshiya Kimura and Fujio Murakami

(see pages 6938 – 6951)

Mitochondria generate ATP and buffer calcium, making them essential in all cell types. They are particularly important in neurons, because synaptic activity and maintaining the resting membrane potential require large amounts of ATP and because neurotransmission requires influx of calcium, which is sequestered by mitochondria. By releasing calcium, mitochondria also influence synaptic plasticity. To perform these roles, mitochondria are transported to and enriched at synapses in mature neurons. Because axonal and dendritic growth and branching also consume ATP and rely on calcium-dependent signaling, one might expect mitochondrial transport to be important during development as well. Indeed, Kimura and Murakami found that transport of mitochondria into dendrites was required for normal development of these processes in mouse neocortical pyramidal neurons. Contrary to expectations, however, the presence of mitochondria appeared to inhibit dendritic branching *in vivo*. Preventing mitochondria transport into dendrites caused exuberant branching of proximal dendrites, increasing the number of branch points and total dendritic length while reducing the linear extent of the dendritic arbor.

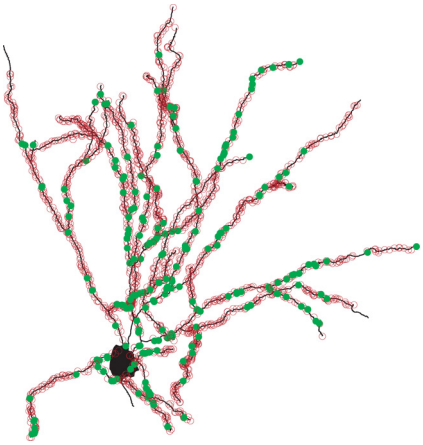
Systems/Circuits

Thalamocortical EPSPs Are No Larger Than Corticocortical

Carl E. Schoonover, Juan-Carlos Tapia, Verena Schilling, Verena Wimmer, Richard Blazeski, et al.

(see pages 6746 – 6758)

Most excitatory inputs to neurons in layer 4 of primary sensory cortex originate in the cortex; although only ~10% of inputs come from thalamus, these inputs exert a strong influence on cortical neurons. It has therefore been hypothesized that individual thalamocortical inputs produce larger EPSPs than corticocortical



Distribution of spine synapses on a layer 4 spiny stellate neuron. Spines apposed to synaptophysin-EGFP signal (filled green circles) were designated as thalamocortical synapses, whereas unapposed spines (open red circles) were assumed to have corticocortical synapses. See the article by Schoonover et al. for details.

inputs because they have more releases sites or because they are located closer to the soma. Data from neocortical slices support this hypothesis: thalamocortical synapses were several-fold stronger than corticocortical synapses. This does not appear to be true *in vivo*, however. By mapping synapses across the entire dendritic arbor of layer 4 cells in rat barrel cortex, Schoonover et al. confirmed that thalamocortical synapses comprised ~6–15% of the spine synapses and tended to be closer to the soma than other inputs. Nonetheless, the amplitude of thalamocortical and corticocortical unitary EPSPs measured *in vivo* were not significantly different. The authors therefore propose that synchronous activity among thalamocortical inputs underlies their effectiveness in driving cortical neurons.

Behavioral/Cognitive

Zebra Finch NCM Might Store Tutor Song

Alessandro Canopoli, Joshua A. Herbst, and Richard H.R. Hahnloser

(see pages 7018 –7026)

Zebra finches learn to sing by memorizing a tutor song heard early in life. They then adjust their own song until it matches the tutor. The adult song remains flexible, however, and birds can be trained to alter the pitch of a syllable if a white noise stimulus is played when the syllable is sung. Nevertheless, the birds revert to their original song within two weeks after the white noise is discontinued. Canopoli et al. hypothesized that the memory of the original song allowing this reversion is stored in the caudal medial nidopallium (NCM), a secondary auditory brain area comparable to human auditory association cortex. Indeed, bilateral ablation of NCM after white noise training impaired the reemergence of the original song. Although the pitch of the altered syllable moved closer to the original pitch, it never fully recovered and the song remained variable for several weeks after white noise discontinuation. NCM lesions did not affect the original song without white noise training, however.

Neurobiology of Disease

α-MSH Prevents Cognitive Decline in TgCRND8 Mice

Keran Ma and JoAnne McLaurin

(see pages 6736 – 6745)

Disruption of the balance between excitation and inhibition in the CNS has been proposed to contribute to cognitive deficits in Alzheimer's disease (AD). One cause of this imbalance may be loss of GABAergic interneurons, particularly those expressing somatostatin. Because α-melanocyte stimulating hormone (α-MSH) has neuroprotective effects and its levels are reduced in AD brain, Ma and McLaurin asked if α-MSH treatment would increase survival of GABAergic neurons and improve cognitive function in TgCRND8 mice, which express an AD-linked form of human amyloid precursor protein. Although amyloid pathology was present in 20-week-old TgCRND8 mice, spatial memory was comparable to that of controls. Memory performance declined over the next 4 weeks, as did the number of GABAergic somatostatin-expressing neurons in hippocampal area CA1. Daily injections of α-MSH during this period prevented neuron loss and cognitive decline without affecting levels of soluble or insoluble β-amyloid, the number of β-amyloid plaques, or other GABAergic neurons, suggesting loss of somatostatin-expressing neurons was a principal cause of cognitive decline.