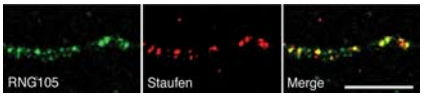
This Week in The Journal

● Cellular/Molecular

A New Player in Dendritic RNA Granules

Nobuyuki Shiina, Kazumi Shinkura, and Makio Tokunaga
(see pages 4420–4434)

Translation of dendritic mRNAs provides a potentially important mechanism for rapid and local control of synaptic efficacy. Dendritic mRNAs are associated with granules that contain clusters of ribosomes and colocalize with other critical molecules for mRNA transport and translation. This week, Shiina et al. report a new RNA-binding protein that could act as an on–off switch for translation at RNA granules. RNA granule protein 105 (RNG105) contains a highly conserved RNA-binding motif. Overexpression of RNG105 tagged with green fluorescent protein induced cytoplasmic ribosome-containing granules in *Xenopus* A6 cells. In rats, RNG105 also was concentrated in granules in hippocampal dendrites and colocalized with mRNAs, including Ca^{2+}/calmodulin-dependent kinase IIα, cAMP response element-binding protein, and tyrosine receptor kinase B. RNG105 bound directly to mRNA and prevented translation *in vitro* and in A6 cells. Considering RNG105 as a repressor of translation, the authors propose a model in which brain-derived neurotrophic factor stimulation leads to dissociation of RNG105 from granules, allowing an increase in local translation.



Cultured hippocampal neurons were immunostained with anti-RNG105 antibody (green) and with antibodies against staufen (red), a component of RNA granules in dendrites. Their colocalization is apparent in the overlay (right). See the article by Shiina et al. for details.

▲ Development/Plasticity/Repair

Foxg1 and Cortical Arealization

Luca Muzio and Antonello Mallamaci
(see pages 4435–4441)

The areas of the cortex are laid out under the control of transcription factors expressed in gradients that direct cortical neuroblasts. These cells then differentiate and migrate into the substructures of the cortex. This week, Muzio and Mallamaci examine *Foxg1* that is expressed in a gradient from high in rostral/lateral areas to low in caudal/medial areas. Mouse *Foxg1* expression begins at embryonic day 9.5 and is necessary for events such as the development of the basal ganglia. In *Foxg1*$^{-/-}$ mice, recent studies suggest that all neurons express Reelin, a marker for preplate (Cajal–Retzius) neurons, perhaps indicating that *Foxg1* is necessary for cortical lamination. However, based on the pattern of expression of transcription factors in *Foxg1*$^{-/-}$ mice, the authors suggest that this effect arises from lateral-to-medial repatterning of the cortical primordium. Without *Foxg1*, nearly the entire cortex developed as archicortex, with ectopic development of hippocampal plate and dentate-blade-like structures in place of the absent neocortical plate.

■ Behavioral/Systems/Cognitive

Synergy Meets Redundancy in the Motor Cortex

Nandakumar S. Narayanan, Eyal Y. Kimchi, and Mark Laubach
(see pages 4207–4216)

One can imagine advantages to both synergy and redundancy. With synergy, ensemble behavior is more than the sum of its parts. With redundancy, removal of an individual part has little effect on the outcome, but ensemble behavior is less noisy and error-prone. Narayanan et al. examine redundancy and synergy in neuron ensembles in rat motor cortex. They made simultaneous single-unit recordings in motor cortex during a reaction time task in which rats were trained to press a level and then release it quickly at the time of a stimulus. A statistical pattern recognition approach was used to examine features in spike trains that predicted behavior or the ensemble response. More than one-half of the neurons showed some task-related features. The ensembles were highly redundant, because removal of individual neurons did little to degrade ensemble performance or predictability. Pairs and triplets of neurons were the exception, showing synergy at times. It seems that for these neurons, like committees, you have more of a voice in a small group.

♦ Neurobiology of Disease

Good News for Runners

Paul A. Adlard, Victoria M. Perreau, Viorela Pop, and Carl W. Cotman
(see pages 4217–4221)

We have all heard about the benefits of exercise, but if you need more evidence, here's some. This week, Adlard et al. show that exercise reduces extracellular amyloid-β (Aβ) plaques in the TgCRND8 mouse model of Alzheimer's disease. Starting at 1 month of age, mice with running wheels in their cages exercised voluntarily (~1.5–3 miles/night) each night for 5 months. These mice had lower Aβ loads in the cortex and hippocampus and were better than controls in some measures of the Morris water maze. Steady-state levels of total amyloid precursor protein (APP), its C-terminal fragments (CTFs), and secretase activity remained constant, as did neprilysin and insulin-degrading enzyme expression. But in mice that exercised for only 1 month, the proteolytic fragments (α- and β-CTFs) were reduced. The authors suggest that exercise-induced neuronal activity may cause metabolic changes in APP processing independent of classical Aβ degradation pathways.