This Week in The Journal

● Cellular/Molecular

Tracking Single Synaptic Vesicles

Edward A. Lemke and Jurgen Klingauf

(see pages 11034–11044)

The static images of synaptic vesicles from the "slam-freeze" studies of Heuser, Reese and colleagues in the 1970s are imbedded in the minds of most neurobiologists. In the 30 years hence, many imaging tricks have been used to capture live the life cycle of the synaptic vesicle. In this week's *Journal*, Lemke and Klingauf used another one. To watch single vesicle movements in hippocampal synaptic boutons, they labeled the entire recycling pool of vesicles with the red dye FM5-95. Ten minutes later, they stained "early endocytosed" vesicles during a brief stimulation in the presence of the green dye FM1-43. The dual staining allowed them to track individual vesicles while excluding samples in which the synapse itself moved. To label "late endocytosed" vesicles, they stimulated synapses for 30 s before dye perfusion. Surprisingly, early, late, and spontaneously released vesicles were similar, being nearly immobile at rest with slightly increased activity during stimulation.

▲ Development/Plasticity/Repair

Reversing Maternal Programming of Stress

Ian C. G. Weaver, Frances A. Champagne, Shelley E. Brown, Sergiy Dymov, Shakti Sharma, Michael J. Meaney, and Moshe Szyf

(see pages 11045–11054)

How we respond to stress has some very early roots. For example, the response to stress in adult rats is affected by epigenetic imprinting resulting from early maternal behavior patterns. Nurturing dams show increased licking/grooming and arched back nursing (high LG-ABN; otherwise known as "good moms"). Their offspring show reduced methylation of exon 1_7 of the glucocorticoid receptor (GR) promoter, elevated expression of hippocampal GRs, and damped stress responses of the hypothalamic–pituitary–adrenal axis, compared with rats raised by low LG-ABN mothers. Weaver et al. explore the reversibility of this imprinting by increasing brain methionine levels in high LG-ABN-reared rats. The treatment led to methylation of the promoter site, reduced binding of the nerve growth factor-inducible protein-A transcription factor, decreased hippocampal GR expression, and a heightened response to stress. The results suggest that the epigenomic marking associated with early behavioral programming is potentially reversible into adulthood.

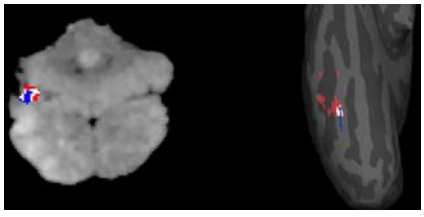
■ Behavioral/Systems/Cognitive

Face and Body Recognition Areas

Rebecca F. Schwarzlose, Chris I. Baker, and Nancy Kanwisher

(see pages 11055–11059)

Areas of the fusiform gyrus respond to images of faces or bodies. In this week's *Journal*, Schwarzlose et al. ask whether these regions of the ventral visual pathway process specialized classes of visual stimuli. Recent functional magnetic resonance imaging (fMRI) studies indicate that the fusiform face area (FFA), which is strongly activated by faces compared with other objects, also responds to images of bodies. The authors confirmed that the FFA responded more strongly to headless bodies or body parts than to cars but not as strongly as to faces. However, when they used higher-resolution fMRI, the authors were able to discern separate but overlapping and adjacent FFA and fusiform body area (FBA) regions of interest in most subjects. Although FFA can respond to nonface objects, this appears necessary for general object recognition. The specialization of FFA and FBA, however, may be the exception rather than the rule for visual processing in other cortical regions.



Examples of the face (FFA, blue) and body (FFB, red) areas from the high-resolution MRI studies of Schwarzlose et al. are shown. Areas of overlap are white. The left image is shown with the cerebellum at the top, right-left reversed. The right panel shows a map of the inflated ventral temporal surface of the right hemisphere. See the article by Schwarzlose et al. for details.

◆ Neurobiology of Disease

Activity-Dependent Dispersion of α-Synuclein

Doris L. Fortin, Venu M. Nemani, Susan M. Voglmaier, Malcolm D. Anthony, Timothy A. Ryan, and Robert H. Edwards

(see pages 10913–10921)

α-Synuclein accumulates in the intracellular inclusions of Parkinson's disease (PD), and mutant forms or overexpression can cause PD. Although α-synuclein lacks a transmembrane domain or lipid anchor, its synaptic localization requires association with lipid rafts. In this week's *Journal*, Fortin et al. investigated the dynamic movements of EGFP-tagged human α-synuclein expressed in dissociated hippocampal neurons with fluorescence recovery after photobleaching. Despite its synaptic localization, α-synuclein was highly mobile, recovering rapidly at a single synapse after photobleaching as a result of movement from neighboring synapses. Neural stimulation also dispersed α-synuclein from the synapse. The dispersion required exocytosis but was more rapid than synaptic vesicle markers, indicating that α-synuclein was not tightly bound to vesicles. The kinetics suggested that the entire pool of α-synuclein was mobile, and that α-synuclein rapidly dissociated from vesicles and the membrane after exocytosis. Surprisingly, reaccumulation did not occur in the first 10 min after stimulation.