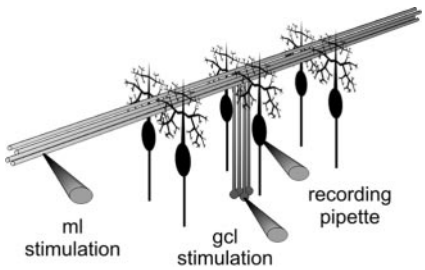
This Week in The Journal

● Cellular/Molecular

LTD-Resistant Cerebellar Synapses

Robert E. Sims and Nicholas A. Hartell
(see pages 3246–3257)

Although it seemed somehow logical to assume that all nerve terminals on an axon have similar properties, studies in several brain regions have found that this is not always the case. This week, Sims and Hartell provide such an example that may have important consequences for the functional organization of the cerebellum. Granule cell (GC) axons pass excitatory information from mossy fibers on to Purkinje cells. GC axons initially extend their axons vertically to the molecular layer, and then they make a sharp turn to extend widely as parallel fibers. The authors report that the properties of synapses on ascending axons differ substantially from parallel fibers. The ascending axon synapses resisted long-term depression induction and displayed a higher release probability and higher mean quantal amplitude than their parallel fiber counterparts. The authors suggest that although ascending axon synapses constitute only 20% of all GC synapses, they carry a stronger synaptic weight. Cerebellar theorists take note.



Cerebellar parallel fiber and ascending axons synapses were independently activated by stimulation in the molecular layer (ml) and granule cell layer (gcl), respectively. See the article by Sims and Hartell for details.

▲ Development/Plasticity/Repair

Assembling Axon Terminals in Zebrafish

Tomoyuki Yoshida and Masayoshi Mishina
(see pages 3067–3079)

Yoshida and Mishina were interested in determining the sequence of events in the differentiation of a nerve terminal. To make these observations *in vivo*, they took advantage of the transparent zebrafish embryo and the olfactory marker protein promoter to specifically label olfactory sensory nerve terminals. They labeled synaptic vesicles and nerve terminals with enhanced green fluorescent protein-tagged vesicle-associated membrane protein 2 and GAP43, respectively. At the time of study, olfactory axons have reached their targets in the olfactory bulb (50–84 h after fertilization). At this stage, the synaptic vesicles gradually accumulated in nerve terminals while the terminals were remodeled from complex shapes with filopodia to simpler shapes. Using dominant-negative constructs and pharmacological tools, the authors found that calcineurin–nuclear factor of activated T cells (NFAT) regulates the shape changes, whereas protein kinase A (PKA)–cAMP response element-binding protein (CREB) is important for synaptic vesicle accumulation. Blocking calcineurin interaction with one of its targets, the transcription factor NFAT, also blocked axon terminal remodeling, suggesting that gene expression is required for this step in synaptic maturation.

■ Behavioral/Systems/Cognitive

Localizing Tasks with fMRI

Giedrius T. Buracas, Ione Fine, and Geoffrey M. Boynton
(see pages 3023–3031)

In response to a task, the cortical area with the maximal functional magnetic resonance imaging (fMRI) response is often considered to be the most relevant area for that task. However, Buracas et al. show that this is not always the case for human subjects in a visual task. The authors compared psychophysical and fMRI responses to moving sinusoidal gratings while subjects performed a speed and a contrast discrimination task. Responses in V1–V3 were larger than MT+ regardless of the task and at all levels of contrast. Yet MT+ is thought to be important in motion perception, including speed discrimination. Why the apparent lack of match with fMRI response size? The authors attribute it to changes in spatial attention with task. However, by using a model in which perceptual thresholds are dependent on the neuronal population mediating the task, the authors were able to identify V1 responses that were consistent with contrast discrimination performance, whereas MT+ responses were consistent with performance on the speed discrimination task.

♦ Neurobiology of Disease

Fish Oil and β-Amyloid in the Mouse

Giselle P. Lim, Frédéric Calon, Takashi Morihara, Fusheng Yang, Bruce Teter, Oliver Ubeda, Norman Salem Jr, Sally A. Frautschy, and Greg M. Cole
(see pages 3032–3040)

Low levels of the omega-3 polyunsaturated fatty acid docosahexaenoic acid (DHA) have been reported in the brains of patients with Alzheimer's disease (AD), as has evidence for an AD protective effect of increased marine fish consumption, a main source of DHA. This week, Lim et al. test whether DHA has a direct effect on amyloid production in a mouse model of AD. They placed aged Tg2576 transgenic mice on a high DHA diet from 17–19 to 22.5 months of age. (Purina breeder chow plus 0.6% DHA if you're interested). Dietary DHA reduced total β-amyloid (Aβ) by 70%, decreased Aβ42 levels, and reduced the overall number of amyloid plaques. DHA depletion also increased amyloidogenic (β-secretase) and nonamyloidogenic (α-secretase) activity. These studies support a role for DHA in reducing amyloid burden even at a relatively late stage in this animal model. Pass the fish oil, please.