

### This Week in The Journal

**Cellular/Molecular**

**Thermotactic Memory in C. elegans**

Synaptic Activity of the AFD Neuron in *Caenorhabditis elegans* Correlates with Thermotactic Memory

Aravindhan D. T. Samuel, Ruwan A. Silva, and Venkatesh N. Murthy

(see pages 373–376)

It has been possible to identify synaptic transmission molecules underlying simple behaviors in *Caenorhabditis elegans* by screening mutants and working backward to identify the culprit genes. However, direct study of cellular events in *C. elegans* has proven difficult because of the small size of the neurons. Samuel et al. use a clever strategy to investigate the role of the AFD neuron in thermotactic behavior (i.e., the ability of the worm to detect spatial thermal gradients and move toward the temperature at which it was cultivated). The worms can remember their cultivation temperature for up to 4 hr, then they “forget.” The authors used AFD-specific promoters to restrict expression of a synaptic vesicle protein [vesicle-associated membrane protein (VAMP)], along with a pH-sensitive indicator (pHluorin), specifically to this neuronal type. The 20 presynaptic terminals of AFD appeared as fluorescent puncta. The fluorescent recovery after photobleaching (FRAP) of selected synapses was used as a measure of synaptic release. The authors found that AFD was active when the ambient temperature differed from the cultivation temperature. AFD was inactive in worms that had been starved for 4 hr (i.e., when they had forgotten the cultivation temperature). The authors conclude that the AFD neuron is part of a comparator circuit for temperature detection. Imaging strategies such as these may provide a critical bridge between molecular and behavioral studies in *C. elegans*.

**Development/Plasticity/Repair**

**Patterned Spontaneous Activity in Spinal Cord Development**

Characterization of the Circuits That Generate Spontaneous Episodes of Activity in the Early Embryonic Mouse Spinal Cord

M. Gartz Hanson and Lynn T. Landmesser

(see pages 587–600)

Patterned neural activity, before input from the environment, has been documented throughout the developing nervous system. Spontaneous activity, thought to play an important role in the establishment and refinement of connections, has been studied most extensively in the visual system. In this issue, Hanson and Landmesser used an isolated spinal cord–hindlimb preparation in the mouse to identify the circuitry that generates local and distributed rhythmic, patterned activity in the spinal cord. Activity was present before and during the time at which motor neurons reach their peripheral targets and was dependent on cholinergic and glycnergic transmission as well as electrical coupling. AMPA/kainate or NMDA receptor-mediated responses were not responsible for rhythmic activity. Rather, nicotinic neurotransmission mediated by dihydro-β-erythroidine hydrobromide (DHβE)-sensitive receptors provided the main excitatory drive. Inhibition of glycnergic excitatory responses also completely abolished spontaneous patterned activity. Antidromic activation and retrograde labeling of motor axons revealed an extensive network of motor axon collaterals that arborize within motor columns, proximate spinal cord regions, and even in developing fiber tracts of the spinal cord. These collaterals contribute to an extensive, developing network that can evoke both local and propagated rhythmic activity. Together, these data provide a new conceptual framework for distinctive local and propagating circuits in the spinal cord. In this scheme, motor neurons are the central elements that drive the spontaneous activity that is likely to influence the development and maturation of central and peripheral motor function.

**Behavioral/Systems/Cognitive**

**Trace Conditioning and Hippocampal Spine Formation**

Associated Memory Formation Increases the Presence of Dendritic Spines in the Hippocampus

Benedetta Leuner, Jacqueline Falduto, and Tracey J. Shors

(see pages 659 – 665)

Dendritic spines are one of the most striking structural features of excitatory synapses on pyramidal neurons in the hippocampus. These protuberances come in a variety of sizes and shapes and can alter their shape within minutes to a variety of stimuli. Given the role of the hippocampus in memory and synaptic plasticity, changes in dendritic spine shape or number have long been considered as a potential cause of increased synaptic efficacy. This idea has gained support from studies of intact animals as well as *in vitro* studies of synaptic plasticity. In this issue, Leuner et al. provide additional evidence for changes in spine density in association with learning. They analyzed hippocampal spine density on CA1 pyramidal cells after a trace eyelink conditioning paradigm, a task known to require the hippocampus, and after training with delay conditioning, a task that does not require the hippocampus but does cause increased activity in hippocampal neurons. Both resulted in increases in spine density. Interestingly, the increase was specific to the basal dendrites of CA1 cells, whereas no changes occurred in the apical dendrites. So is the increase in spines necessary for learning or does it simply represent activity in the hippocampus? The authors argue that the increased spine density accompanies associative memory, but that the increase is not necessary for learning to occur.