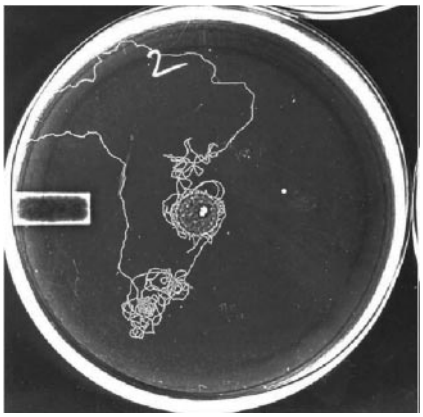
This Week in The Journal

● Cellular/Molecular

A Mating Game in the Worm

Jonathan Lipton, Gunnar Kleemann, Rajarshi Ghosh, Robyn Lints, and Scott W. Emmons
(see pages 7427–7434)

Although the motivation behind mate-seeking behaviors has long been fodder for popular literature, little is known of these complex drives at the most basic level. In this week's *Journal*, Lipton et al. delve into this behavior in worms using measures we can all understand, food and companionship. *Caenorhabditis elegans* males differed markedly in their behavior from hermaphrodites (the mate in the case of *C. elegans*). Sexually mature males, when left alone on a food source (a nice plate of *Escherichia coli* if you're interested), wandered off, presumably in search of a mate. When a potential mate was present on the food source, though, they remained. Hermaphrodites, in contrast, do not require a mate to reproduce, and did not leave the food source. However, in male–female nematode species, both sexes left food in search of a mate. Mutations in the serotonin and insulin signaling pathways that affect food seeking also influenced mate seeking, as did genes involved in gonadal signaling.



Tracks of a wild-type male *C. elegans* isolated on a food source away from mating partners. The male was placed initially in the central ring of food and his path was followed for 5 hr. See the article by Lipton et al. for details.

▲ Development/Plasticity/Repair

Wiring in a Cone-Only Retina

Enrica Strettoi, Alan J. Mears, and Anand Swaroop
(see pages 7576–7582)

In the retina, rods and cones form a distinct wiring diagram with specific second-order and third-order neurons. Although the pathways eventually overlap at the ganglion cell layer, they are segregated at their initial inputs onto rod-specific and cone-specific bipolar cells. But what happens when rods, normally constituting >90% of photoreceptors, do not form and thus all photoreceptors are cones? Strettoi et al. address this question using Nrl (neural retinal leucine zipper)-deficient mice. Absent this transcription factor required for rod cell differentiation, the retina generates extra cones. The authors asked whether rod bipolar neurons remain functional and whether the enlarged population of cones transmits signals to ganglion cells. Amazingly, the morphology, connectivity, and transmission of rod bipolar neurons as well as horizontal and AII amacrine cells that normally receive rod input appeared to be neatly maintained when rods were replaced with cones. The cone-only retinas were also light-sensitive.

■ Behavioral/Systems/Cognitive

An Additional Role for Orexin

Susan G. Walling, David J. Nutt, Margaret D. Lalies, and Carolyn W. Harley
(see pages 7421–7426)

The orexin (hypocretin) peptides were discovered in the hypothalamus and soon found to be involved in feeding and sleep behaviors. However, the distribution of nerve fibers containing orexins is quite widespread, perhaps suggesting broader actions. This week, Walling et al. explored the actions of orexin-A (ORX-A) in the locus ceruleus (LC), which receives a dense projection of ORX-A neurons. As their assay, they used noradrenaline-induced long-term potentiation (NE-LTP) in the hippocampus, which results from activation of β-adrenergic receptors in dentate granule cells. They found that ORX-A infused into the LC of an anesthetized rat induced NE-LTP. When the LC was silenced with an α_2-adrenergic agonist, ORX-A lost its effect. Microdialysis experiments confirmed that the ORX-A input increased NE release from LC neurons in dentate gyrus. These experiments may provide a pathway linking sleep disorders such as narcolepsy to attention and memory deficits that can accompany such disorders.

◆ Neurobiology of Disease

Bystander Cell Death of Motoneurons

Jessica Darman, Stephanie Backovic, Sonny Dike, Nicholas J. Maragakis, Chitra Krishnan, Jeffrey D. Rothstein, David N. Irani, and Douglas A. Kerr
(see pages 7566–7575)

Glutamate-mediated excitotoxic cell death has been implicated as a partner in several forms of selective motoneuron death. Darman et al. now investigate this issue in an interesting model system. They infected mice and rats with neuroadapted Sindbis virus (NSV), a neurotropic virus that causes hindlimb paralysis and motoneuron loss. They found that motoneuron death could be prevented by blocking calcium-permeable AMPA receptors, but degenerating neurons were not necessarily infected with NSV. Thus, bystander cell death must account for at least some of the cell loss. Astrocytic glutamate transport contributed to the susceptibility to NSV-induced damage as transporter blockers increased motoneuron loss *in vivo*. The main glutamate transporter GLT-1 was also downregulated during infection, perhaps because of a cytokine-mediated mechanism. In addition, the susceptibility of different strains correlated with their basal glutamate transport. The authors suggest that this sequence of events may occur in other conditions with acute or chronic motoneuron loss.