219. Modulation of C. elegans egg-laying behavior by the environment and experience
Niels Ringstad, Bob Horvitz
HHMI, Dept. Biology, MIT, Cambridge, MA 02139 USA

The egg-laying behavior of *C. elegans* provides a powerful system for the study of neural circuits at a cellular and molecular level. While the egg-laying neuromusculature is simple and well-characterized, little is known about how egg-laying behavior is regulated by the environment or experience. We are interested in characterizing two types of modulation of egg-laying behavior. First, when a well-fed hermaphrodite is removed from a food source, the animal stops laying eggs. More recently, we have also observed that upon return to food, the frequency of egg-laying events in food-deprived animals increases over that of animals left on food, and the magnitude of this increase is a function of the duration of time spent away from food.

To identify mutants defective in the modulation of egg-laying, we have begun to examine the existing set of Egl mutants, in particular class C, D, and E Egl mutants, which have HSNs with apparently normal morphology, have functional sex muscles, and have a normal vulva, yet lay fewer eggs than wild-type animals. These mutants, like wild-type animals, lay eggs in response to exogenous serotonin and in response to the serotonin reuptake inhibitor imipramine, which is thought to potentiate the signaling from the serotonergic HSNs to the sex muscles. The mutations underlying the egg-laying defects in serotinin- and imipramine-responsive Egl mutants may constitutively activate or alter the properties of pathways whose normal role is to modulate egg-laying.

Our preliminary survey of serotonin- and imipramine-responsive Egl mutants has identified *egl-6(n592)* and *unc-31(e928)* as mutants that fail to inhibit egg-laying in the absence of food. We also identified *egl-7(n595)* as a mutant that fails to up-regulate egg-laying after a period of food deprivation.*unc-31* has been cloned by others and encodes a CAPS-like protein that is implicated in the exocytosis of dense-core granules. *egl-6* and *egl-7* have not been molecularly characterized. We are pursuing the cloning and further characterization of these two genes.