Abstract/Session Information for Program Number 452B

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Abstract Content

Program Nr: **452B**

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

The egg-laying behavior of C. elegans is modulated by the environment and experience. When a well-fed hermaphrodite is removed from a food source, the animal stops laying eggs. We have observed that upon return to food, the frequency of egg-laying events by food-deprived animals increases over that of animals left on food, and the magnitude of this increase is proportional to the duration of time spent away from food.

To characterize this modulation of egg-laying behavior, we have examined egg-laying behavior by mutants defective in various sensory pathways and neurotransmitter signaling. Our results suggest a role for chemosensory neurons and for the inhibitory neurotransmitters octopamine, tyramine, and GABA in the inhibition of egg-laying behavior in the absence of food.

We have also scored Egl mutants for defects in the modulation of egg-laying behavior, in particular class C, D, and E Egl mutants (Trent et al., 1983), 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. It therefore seems that in these mutants there are no defects in either the release of serotonin from the HSNs or in the ability of the egg-laying neuromusculature to respond to serotonin.

One possibility is that mutations underlying the egg-laying defects in serotonin- and imipramine-responsive Egl mutants inappropriately activate or alter the properties of pathways that normally inhibit egg-laying behavior. Our survey of serotonin- and imipramine-responsive Egl mutants suggests that such mutants exist. While most Egl mutants appropriately modulate egg-laying behavior, *egl-6(n592)* and *unc-31(e928)* mutants, in addition to having low rates of egg-laying, fail to inhibit egg-laying in the absence of food. We have also found that *egl-7(n595)* mutants fail to up-regulate egg-laying after a period of food deprivation. *unc-31* has been cloned by others and encodes a CAPS-like protein implicated in the exocytosis of dense-core granules. It is therefore possible that regulated secretion of neurotransmitters or neuromodulators is required both to stimulate egg-laying behavior in the presence of food and inhibit egg-laying behavior in the absence of food. We are pursuing the cloning and further characterization of *egl-6* and *egl-7*.

Session Information

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Abstract Information

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