

Genetic and Neural Pathways Underlying Light-induced Inhibition of Pharyngeal Pumping by *C. elegans*

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C. elegans moves away from ultraviolet and blue light, and this avoidance is dependent on LITE-1, a putative light sensor, as well as the uncloned genes *lite-2* and *lite-3* (Edwards..Miller, *PLoS Bio* 2008). We found that worms also stop pharyngeal pumping in response to ultraviolet and blue light. For example, worms immediately stop pumping when exposed to blue light (the “acute response”) and maintain reduced pumping for many seconds after blue light is removed (the “sustained response”).

Mutations in *lite-1*, *lite-2* or *lite-3* have little effect on the acute response but lead to a completely defective sustained response, such that pumping immediately recovers to pre-light levels after light is removed. After testing neurotransmission-related mutants, we found that *eat-4*, which encodes a vesicular glutamate transporter, is required for the acute but not the sustained response to light. *unc-13*, which encodes a regulator of neurotransmitter release, is required for both responses. Since *eat-4*; *lite-1* double mutants lack both the acute and sustained responses, *eat-4* and *lite-1* likely act in parallel genetic pathways, indicating that an additional light sensor upstream of *eat-4* might be present in the worm. Pumping in mutants of *gur-3* and *gur-4*, paralogs of *lite-1*, respond normally to light.

The *C. elegans* nervous system consists of 2 anatomical networks connected near the tip of the nose by a pair of gap junctions between the main network’s RIP neurons and the pharyngeal network’s I1 neurons. Consistent with the hypothesis that the main network is involved in the pumping response to light, laser ablation of both I1s yielded worms that lacked the acute but not the sustained light response, similar to *eat-4* mutants. To investigate whether *eat-4* acts downstream of the I1s, we ablated individual classes of *eat-4*-expressing pharyngeal neurons (M3s, NSMs, and I5). We found that these ablations had no effect on the worm’s light response, indicating that *eat-4* likely acts outside the pharynx to control the acute response. We also tested available mutants of other known glutamate transporters as well as glutamate receptors, but all responded normally to light.

We plan to identify the site-of-action of *lite-1* and *eat-4* via cell ablation and cell-specific rescue experiments. As we continue ablations and identify more neurons required for the pumping response to light, we plan to image calcium to measure the physiological sequence in which these neurons are activated. We also plan to do a mutagenesis screen for additional mutants defective in the pumping response to light to seek the light sensor upstream of *eat-4*, the glutamate receptor downstream of *eat-4*, and the signaling molecules downstream of *lite-1*. We hope that such analysis will elucidate important principles about neural communication that are relevant more generally across species.

Poster

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