Components of the Neural Circuit Implementing Light-induced Inhibition of Pharyngeal Pumping

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C. elegans moves away from short-wavelength light, with strong avoidance of 350 nm UV and weak avoidance of 500 nm blue-green. This avoidance requires LITE-1, a transmembrane protein (Edwards..Miller, PLoS Bio 2008). We found that worms also stop pharyngeal pumping in response to shortwave light. Specifically, worms immediately stop pumping when exposed to 436 nm violet light (the "acute response") and maintain reduced pumping for many seconds after violet light is removed (the "sustained response").

To identify neurons required for this reflex, we used laser microsurgery to systematically kill all 20 neurons in the pharynx. We found that the I1 and I2 neurons are partially redundantly required for the acute response to light. Using the calcium indicator GCaMP3, we observed that light causes a calcium increase in I2 but not I1, with a latency as short as ~300 ms. Depolarization of I2 but not I1 by channelrhodopsin (ChR2) also enhances the light response.

To identify additional components of the light response, we tested candidate mutants. Mutations in *lite-1* partially reduce the acute response and completely abolish the sustained response. *eat-4*, which encodes a vesicular glutamate transporter, is required for the acute response. *eat-4* is expressed in I2 but not I1, and cell-specific expression of *eat-4* in I2 but not I1 partially rescues the defective response of *eat-4* mutants. Strikingly, the *eat-4; lite-1* double mutant is completely unresponsive to light, indicating that the light response is implemented by 2 parallel genetic pathways.

Our results suggest that I2 might sense light as the “eye” of the pharynx and releases glutamate to quickly and briefly inhibit pumping. Our goal is to describe the complete neural circuit underlying this light-induced reflex, such that we can watch the light-dependent signal flow and be transformed from one to cell the next.

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