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Optogenetic dissection of neural circuits in *C. elegans*

Barry Bentley, Marios Chatzigeorgiou, Ithai Rabinowitch, William Schafer

MRC Laboratory of Molecular Biology, United Kingdom

Presenter: Barry Bentley

Despite knowing the connectivity of the *C. elegans* nervous system, its computational properties remain poorly understood. This is especially true for gap junctions, which while forming 890 electrical synapses in *C. elegans*, have been largely unexplored in terms of processing.

Previous research has demonstrated a role for gap junctions in coincidence detection [1]; however, the mechanisms underlying this function have yet to be fully elucidated. Our hypothesis is that gap junctions facilitate shunting inhibition by leaking current to inactive neurons in the network, thus requiring concomitant activation. To test this hypothesis, we have expressed a light-activated cation channel (channelrhodopsin) in two classes of electrically-coupled neurons, OLQ and CEP, which are known to modulate FLP-mediated reversals via RIH. Our hypothesis predicts that simultaneously activating these neurons should induce reversals, whereas the stimulation of an individual neuron should not. We are now performing experiments to test this.

Combining the controlled-illumination system of Stirman *et al.* [2], with Q binary expression [3], and brainbow recombination [4], we are also developing a system for cell-specific targeting of optogenetic reagents, with the aim of surveying further circuits in *C. elegans* at the level of individual neurons.

References:

[1] Chatzigeorgiou, M. & Schafer, W.R. *Neuron* 70, 299-309 (2011).

[2] Stirman, J.N., *et al. Nat Meth* 8, 153-158 (2011).

[3] Wei, X., *et al. Nat Meth* 9, 391-395 (2012).

[4] Hampel, S., *et al. Nat Meth* 8, 253-259 (2011).