Summary of Article

Neuronal Regeneration in C. elegans Requires Subcellular Calcium Release by Ryanodine Receptor Channels and Can Be Enhanced by Optogenetic Stimulation

Calcium signals play a huge role in neuronal repair. Calcium that is released from the endoplasmic reticulum via the ryanodine receptor (RyR) channels is critical in stimulating initial regeneration following traumatic cellular damage *in vivo*. Mutation of *unc-68*/RyR greatly impedes the growth of regenerating neurons. *Caenorhabditis elegans* is a useful model organism to study neuronal regeneration. In this experiment, it was addressed how cellular calcium signals might stimulate new growth in a damaged neuron. The results led to the conclusion that RyR channels mediate sustained subcellular calcium signals in a damaged neuron that are critical in the initial stages of regeneration. This is an indication that mutation of the RyR2 can lead to increased activity of the ryanodine receptor.