ENDOSOMAL TRAFFICKING IN DAMAGED OR SEVERED AXONS IN *C. ELEGANS*

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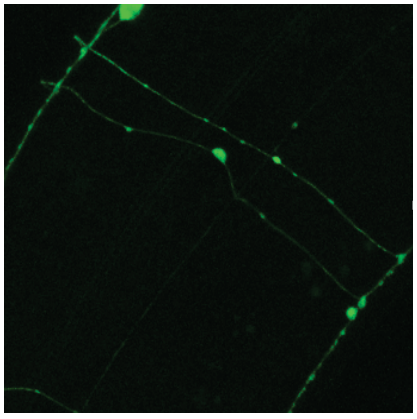
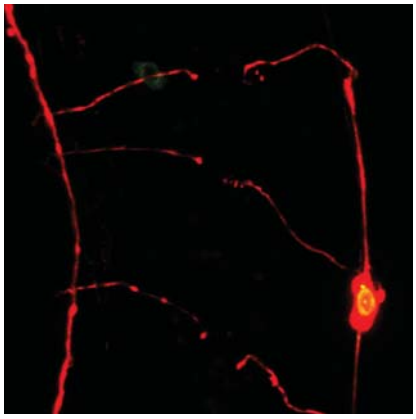
When an axon is subject to severe damage, the neuron must compensate by engaging in a series of stress-induced biochemical responses. These responses are aimed at up-regulating transcription factors to assist the axon with mounting a successful regenerative response. This response must include increased synthesis of materials for the growth cone and newly formed axon which must be transported to the growing tip of the regenerating axon. This up-regulation of transport can be visualized by the increase in organelles trafficking from the cell body to the axon. This includes organelle such as the Golgi apparatus, endoplasmic reticulum, lysosomes, and endosomes. A recent RNAi screen completed in 2014 by Nix et. al. identified at least 50 genes of *Caenorhabditis elegans* that contain either growth-promoting or growth inhibiting functions. One of these genes was *unc-16*, which encodes the homolog of mammalian JIP3, a JNK-interacting protein that has been found to bridge the activity of JNK-1 and JNK kinases (Byrd et al., 2001). It has been proposed that the *unc-16* gene could serve as a 'gatekeeper' for organelles in the axon initial segment (AIS). The AIS serves as a regulatory junction between the axon and the neuron cell body. It plays a vital role in maintaining the axon organelle composition that could support the axon during stressful biological events such as axonal injury. A study using *unc-16* loss-of-function mutants showed that axons lacking this gene accumulated endosomal organelles at a rate that was 5-7 times higher than the wild type worms (Edwards et al., 2013)[1]. In this study we addressed the relationship between the quantity of endosomes transported to the site of injury and the likelihood of a successful axonal regeneration. We used still-imaging and time-lapse imaging techniques to determine if there was a significant difference between endosomal count in wild type and unc-16 worms and if increased organelle trafficking contributes to successfully regenerating an axon. Our findings suggest that there is a substantial increase in endosomal accumulation in the AIS of severed axons in unc-16 mutants compared to willd-type. However, although there is a strong improvement in regeneration in unc-16 animals compared to wild-type animals (90% compared to 70%), we could not correlate this improved regeneration with increased endosomal trafficking in each specific wild type and unc-16 regenerating axon.





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