



The Influence of Cold Atmospheric Plasma on *Caenorhabditis elegans* Axon Regeneration

Speaker: Jacob Manjarrez, PhD

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Where: 1st floor conference room, IRP II

Abstract:

It has been shown that the ability of injured axons to regenerate declines with age. The development of a treatment to overcome this decline would improve the viability of damaged axons associated with spinal cord injuries. In recent years, *Caenorhabditis elegans* has emerged as a model for the study of axon regeneration through the development of axotomy. Axotomy is a precise procedure, which uses a laser to sever a single axon. This technique is used to investigate the effect of cold atmospheric plasma (CAP) on axon regeneration through growth cone initiation and complete regeneration. Here we show that axon regeneration in aged *C.elegans* is enhanced with treatment by CAP. In five day old *C.elegans* growth cone formation, the first step in axon regeneration, increases following treatment with CAP by approximately 40%. The treatment also expedites the full regeneration of axons with approximately one-third of axons regenerating in a six hours period. This is accomplished with a small increase in the *C.elegans* average lifespan compared to sham control. The mechanism that enables CAP to overcome the age related decline in axon regeneration points to the conserved neuron related pathways that are currently under investigation.

Biosketch:

Dr. Manjarrez is a post doctoral researcher in Prof Kong and Chen's laboratory in the Research Center for Bioelectricity at Old Dominion University. He received his Ph.D. from Oklahoma State University in the Department of Biochemistry and Molecular Genetics, where he studied the Hsp90 client protein DBC2. In his post-doctoral work, he studied the function of cholinergic signaling in *C.elegans* under James Rand at the Oklahoma Medical Research Foundation. At ODU, his current research focuses on axon regenerative in *C.elegans* using Cold Atmospheric Plasma, which facilitates expedited regeneration of injured axon after CAP exposure.