Abstract:

Caenorhabditis elegans, a small nematode with its genome being sequenced, well-defined nervous system and pre determined cell fate has been serving the scientific community to study various cellular and biological processes. More the 60% of *C. elegans* genes have human counterparts, which makes it an excellent model system to study diverse biological processes such as neurobiology, aging, apoptosis, gene regulation and developmental biology.

In my work I have used *C. elegans* to understand two different phenomena; synaptic functioning and RNA splicing.

In first part of my talk, I will discuss characterization of the function of a claudin-like protein, HPO-30, and its role in maintaining the levamisole sensitive nicotinic acetylcholine receptors (LAChRs) at the neuromuscular junction (NMJ). Using pharmacological and electrophysiological approaches we establish that in *hpo-30* mutants, the LAChR levels are compromised at the NMJ. HPO-30 localizes at the NMJ and shows genetic and physical interaction with the LAChRs. Finally, we show that HPO-30 functions through another cell adhesion molecule neuroligin (NLG-1) to maintain the postsynaptic receptor levels.

The second part of my talk involves understanding the function of a ubiquitin like protein, Hub1 in *C. elegans.* In this work, we identify the interaction of *Ce*Hub1 with splicosomal protein *Ce*SNU66. Further, using microarray analysis we assay for splicing specific function of Hub1 in *C. elegans* and establish that Hub1 function is quite conserved across species and plays an important role in mRNA splicing in *C.elegans.*