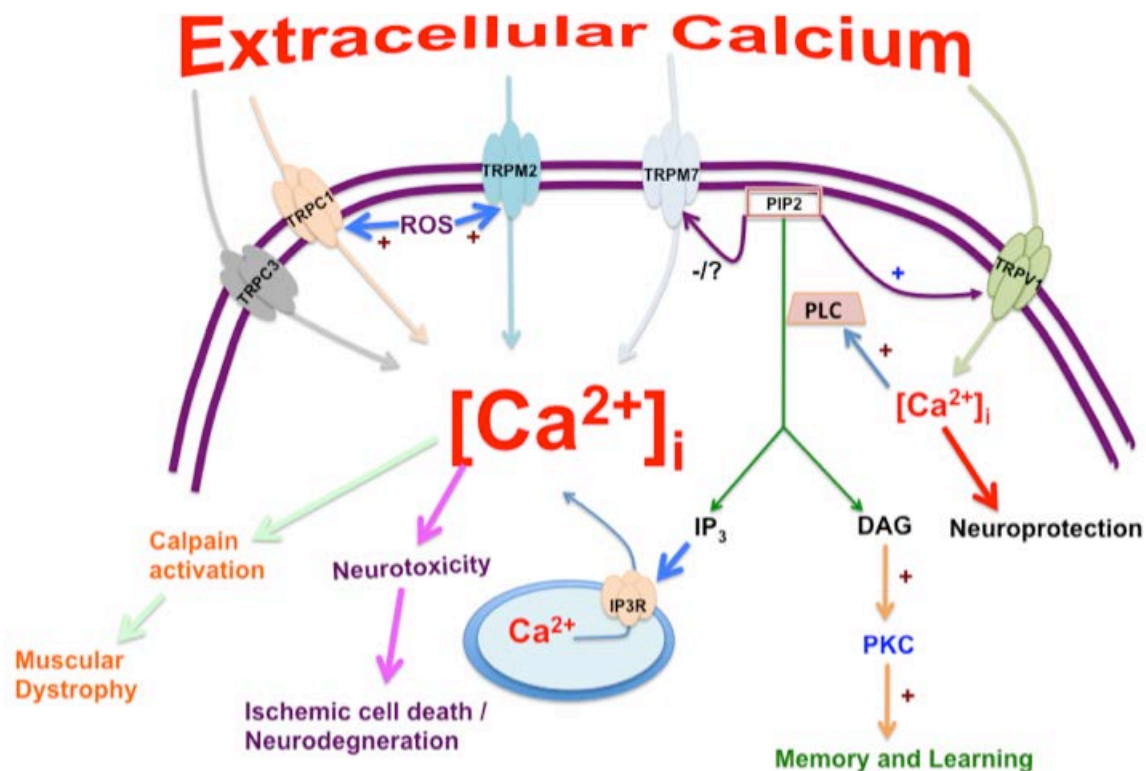


The major goal of our research is to understand the implications of membrane 4,5-phosphatidylinositol 4,5-bisphosphate (PIP2) and Transient Receptor Potential (TRP) ion channel activity in the pathophysiology of neurodegenerative diseases. We employ electrophysiological, intracellular Ca^{2+} imaging, optogenetic tools, confocal microscopy, biochemical, cell and molecular biological techniques and *in vivo* approaches to answer exciting research questions to understand the complex mechanisms that are involved in the cause and regulation of neuromuscular and neurodegenerative diseases. We use *in vivo* animal models, isolated nerve-muscle preparations, heterologous expression systems in cell lines, primary cells and unrestrained worms (*C. elegans*) to study and discover novel mechanistic pathways as well as drugs/tools to treat such diseases.

To expand our knowledge of synaptic diversity in the expression and interactions among TRP channel proteins, PIP2, neurotoxins and lipid microdomains, we aim to uncover the potential role of novel partners and mechanisms. This will eventually contribute for the development of TRP channels as therapeutic targets. The hypothetical model described below signifies the role of TRP channel proteins in the pathophysiology of neurodegenerative diseases, muscular dystrophy, and for neuroprotection. Further research to elucidate these effects of TRP channels is in progress.



TRP, transient receptor potential proteins; TRPC, TRP canonical; TRPM, TRP melastatin; TRPV, TRP vanilloid; PIP2, phosphatidylinositol 4,5-bisphosphate; PLC, phospholipase C; IP₃, inositol 1,4,5-trisphosphate; DAG, diacylglycerol,

$[Ca^{2+}]_i$, intracellular Ca^{2+} ; + activation/stimulation; - inhibition or suppression; ?
effect unknown.