

PRG2's Effect on LPA Reception in Neurons

How do axons know where to go during development? Plasticity-related genes (PRGs) have previously been shown to play a critical role in the developing brain by controlling memory and learning. Recently, some lysophosphatidic acids (LPAs), which have been shown to be integral in synaptic signaling and neuropathic pain, have been shown to interact with these PRGs. So, how exactly do these two components interact with each other and does it have any significance in axon signaling and developmental guidance? Previous research on other LPAs and PRGs, suggests that PRG2 mediates LPA receptor function by inhibiting its receptive abilities. To test this, we performed a neurite retraction assay on wild-type expressing B103 (rat neuroblastoma) cells and another one after FuGENE transfecting the PRG2 gene into said cells. Cells of each type (two without LPA receptors B103-V/B103 and two with LPA receptors B103-LPA2/B103-LPA4) were plated on coverslips, treated with different concentrations of LPA, and counted. Additionally, we ran RT-PCRs that showed PRG2 is already expressed in the cells. This means the study is actually studying the effects of overexpression. According to other studies, PRGs should inhibit LPAs' ability to trigger a RhoA/ROCK signaling pathway and therefore slow cell rounding. However, our preliminary trials indicate PRG2 expressing cells are just as likely to round cells. Theoretically, these studies can better understand how different molecules interact and communicate in neurons. Further studies could help us better understand certain LPA related disorders like multiple sclerosis and neuropathic pain.