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Glioprotection Versus Neuroprotection

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Description

Neuroprotection outlined as relative preservation of neuronal structure and/or function. Includes neuroprotective agents that secure from neuronal injury following acute diseases or neurodegeneration in brain following chronic neurodegenerative diseases. Most common forms of brain injury during neurosurgical procedures are brain retraction, incising and take out brain tissue, and temporary vascular occlusion. For instance, eliminating pathological brain tissue, brain retraction will necessarily guide to injury of normal brain structures. Moreover, clamping of a carotid artery during carotid endarterectomy or temporary clipping of intracerebral arteries can create unilateral global ischemia or acute ischemic stroke, respectively. *In vitro* model of TBI, dexmedetomidine had shown a significant neuroprotective cause. Activation of extracellular signal-regulated kinases might be connected in mediating the neuroprotective effect of dexmedetomidine.

Glia modulate neuronal excitability, seizure sensitivity by conserving potassium and water homeostasis. A SIK3 regulated gene expression program handles glial ability to buffer K+ and water, however upstream regulatory mechanisms strange. Here we recognize an octopaminergic circuit connecting neuronal activity to glial ion and water buffering. Under basal conditions, octopamine functions through inhibitory octopaminergic GPCR OctβR to upregulate glial buffering ability, while under pathological K+ stress, octopamine signals through stimulatory octopaminergic GPCR OAMB1 to downregulate glial buffering program. Prosaptide stimulation of cells transduced with GPR37 or GPR37L1 caused phosphorylation of ERK in a pertussis toxin-sensitive manner, stimulated (35)S GTPγS binding, and promoted prohibition of forskolin-stimulated cAMP production. Prosaptide is active fragment of secreted neuroprotective, glioprotective factor prosaposin (sulfated glycoprotein-1), we purified full-length prosaposin and found that it stimulated GPR37, GPR37L1 signaling. In addtion, both prosaptide, prosaposin were found to secure primary astrocytes against oxidative stress, with these protective effects being reduced by siRNA-mediated knockdown of endogenous astrocytic GPR37 or GPR37L1.

For immunocytochemical experiments, atleast of ten larvae were assessed for all genotype and condition. For electrophysiology experiments, data were pooling from at least seven independent cells derived from a minimum of four different larvae. Here, every NMJ is consider as n of 1, as each motor axon is controlled to its own muscle cell. For behavioral studies, a minimum of fifty and hundred flies assessed per genotype, respectively. Elaborated information on n used for every experiment included in figure legends. Male, female animals were used in comparable numbers exclude for behavioral and lifespan assays, in which male adult flies are used. There are no statistical differences in results betwixt the two groups. All data are display as mean standard error of mean. Data had passed D'Agostino Pearson and Shapiro Wilk test for normality earlier being evaluated for statistical importance. Statistical analyses were executed using Prism, including one way ANOVA test with Tukey's multiple comparisons, two way ANOVA test with Tukey's multiple comparisons and unmatched two tail Student's test with unequal variance. Results shown represent data pooled from at least two independent trails executed on larvae or adult flies derived from different crosses. Researchers were blinded to genotype, treatment condition during all experiments, data analyses.