Jufang Huang et al., J Clin Exp Ophthalmol 2016, 7:3 (Suppl)
http://dx.doi.org/10.4172/2155-9570.C1.033



# Macroglia derived trombospondin 2 modulates retinal synaptic plasticity following acute high intraocular pressure

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Many researches on retinal injury and repair following High Intraocular Pressure (HIOP) suggested that the survival ratio of Retinal Ganglion Cells (RGCs) have been improved much by kinds of measures. However, the visual function recovery is far lower than expected. Previous studies found that the homeostasis of the synapse in visual signal pathway is the key structural basis for the delivering of visual signals *in vivo*. While, the studies from our group indicated that the complicated synaptic plasticity of retinal neurons occurred much earlier than obvious degeneration of RGCs in rat retinae. We speculate that the retinal synaptic plasticity may be one a key reason for the limited visual function recovery under kinds of RGCs repair means following HIOP. Exploring the modulatory mechanisms of synaptic plasticity after HIOP may provide new ideas for better visual function recovery. Interestingly, macroglia was found activated after HIOP and inhibiting its activation could suppress the alteration of synapse following acute HIOP. Thus, retinal macroglia may participate in the modulation of synaptic plasticity in rat retina after HIOP. From *in vivo* and *in vitro* study, we proved that macroglia modulated retinal synaptic plasticity through delivering Trombospondin 2 (TSP2), which bounds to its neuronal receptor α2δ1 to promote synaptogenesis. These results may provide new evidences to retinal repair strategies for better visual function recovery on intervention time points and targets after HIOP.

## Biography

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