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"Targeting adenosine receptors in retinal degenerative diseases: from neuroinflammation to neuroprotection"

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RESUMEN

Glaucoma is a progressive retinal degenerative disease and the second cause of vision loss in the world. It is characterized by damage of the optic nerve and retinal ganglion cell (RGC) loss. The degeneration of RGCs in glaucoma is accompanied by an increased neuroinflammatory response involving retinal microglial cells and RGCs. The etiology of glaucoma is unknown, but elevated intraocular pressure (IOP) and aging have been identified as risk factors. IOP is currently the only modifiable risk factor and the main target for therapeutic interventions. However, glaucomatous progression continues in as many as half of glaucoma patients undergoing an IOP-lowering regimen. Therefore, new and more effective treatments are necessary, and neuroprotection of RGCs is considered to offer potential as an alternative therapy.

Adenosine is a neuromodulator in central nervous system (CNS) acting on metabotropic adenosine receptors (A1, A2A, A2B and A3). It has been claimed that adenosine is one of the most promising neuroprotective systems in CNS.

Our results show that, in experimental models of glaucoma, the blockade of A2A receptors prevents retinal neuroinflammation and the activation of A3 receptors confers neuroprotection to RGCs. These results may open the possibility for new pharmacologic strategies for the treatment of retinal degenerative diseases, like glaucoma.

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