Novel Therapy Targeting Autoimmunity for the Treatment of Glaucoma and Optic Neuropathy
Technology #14870

Applications

This technology is a method for inhibiting and reducing the impact of heat shock proteins (hsp) in ocular neurodegeneration resulting in glaucoma and associated pathologies.

Problem Addressed

Glaucoma is the second leading cause of irreversible blindness worldwide, affecting one in two hundred people aged fifty and younger, and one in ten people over the age of eighty. The major risk factor for glaucoma is elevated intraocular pressure (TOP), which contributes to significant damage of the optic nerve and loss of retinal ganglion cells (RGCs). When left untreated, glaucoma leads to the permanent damage of the optic nerve and visual field loss, which often progresses into irreversible blindness. Current treatment to lower intraocular pressure involves eye drops or surgical intervention; however, treatment is ineffective in stopping its progression to vision loss. New and effective strategies are necessary for early detection and treatment of glaucoma.

This invention provides a potent and effective therapeutic alternative for the treatment of glaucoma by inhibiting key players in the autoimmune response leading to intraocular pressure (TOP).

Technology

The core of this technology relies on the discovery that autoimmune CD4 T response to heat shock proteins 27 (hsp27) and or (hsp60) in the pathogenesis of glaucoma. The autoimmune response initiated by elevated intraocular pressure (TOP) is a key component in causing progressive retinal ganglion cell (RGC) and axonal degeneration in glaucoma.

This invention provides a method for the early diagnosis and evaluation of treatment efficacy, associated to heat shock proteins (hsp27 or hsp60) by detecting auto-antigen-reactive T cells or auto-antigen-antibodies. The invention also provides a therapeutic treatment of glaucoma, anterior ischemic optic neuropathy (AION), and optic nerve trauma by inhibiting an autoimmune response triggered by elevated TOP, ischemia, trauma or other injuries in a subject.

Advantages

- Effective method in inhibiting or reducing the severity of a heat shock protein (hsp)-mediated ocular neurodegenerative condition.
- Early diagnosis and evaluation of treatment efficacy
- Immunosuppressant agent could be administer to ocular tissue
- Therapeutic treatment of glaucoma, anterior ischemic optic neuropathy

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Categories For This Invention:

Biotechnology
Health
Clinical Applications
Other (Clinical Applications)
Ophthalmology
Diagnostics
Protein
Other (Diagnostics)
Therapeutics
Other (Therapeutics)

Intellectual Property:

Therapies that target autoimmunity for treating glaucoma and optic neuropathy.
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Publications:

Commensal Microflora-induced T cell Responses Mediate Progressive Neurodegeneration in Glaucoma
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Image Gallery: