

Neuroprotection for glaucoma: Requirements for clinical translation

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Within the field of glaucoma research, neuroprotection is defined as slowing the functional loss in glaucoma by a mechanism independent of lowering of intraocular pressure. There is currently a great potential for research surrounding neuroprotection as it relates to glaucoma. Anatomical targets for neuroprotection should focus on upstream rather than downstream factors, and could include any part of the retinal ganglion cell, the glia, especially astrocytes or Muller cells, and vasculature.

The great number of anatomical targets is exceeded only by the number of possible biochemical pathways and potential treatments. Successful treatment may be accomplished through the targeting of one or even a combination of multiple pathways. Once a treatment is shown effective in vitro, it should be evaluated in vivo with carefully chosen animal models and studied in sufficient numbers to detect statistically and clinically significant effects. Such a drug should have few systemic side effects and its delivery should be optimized so as to encourage compliance.

There are still a multitude of possible screens available to test the efficacy of a neuroprotective drug and a single gold standard is ideal for the accurate assessment and comparison of new drugs. Future studies in neuroprotection should investigate the genetic component of the disease, novel pharmaceutical agents for new or known pathways, modulations of scleral biomechanics, and relation to research of other complex disorders of the central nervous system.

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