Novel therapy for the treatment of optic neuropathies

CSIC and Tel-Aviv University have developed a family of compounds able to inhibit Semaphorin3A fostering nerve regeneration after injury. They can be useful for treatment of ischemic events such as stroke, glaucoma and other optical nerve and retina insults.

Industrial partners from the pharmaceutical industry are being sought to collaborate through a patent licence agreement.

An offer for Patent Licensing

Neural regeneration by inhibition of Sema3A

After a neuronal damage is produced, Semaphorin3A (Sema3A) protein is highly expressed as a response in adult mammalian central nervous system (CNS), preventing axon regeneration and promoting progressive neuronal apoptosis, even once the ischemic event is restored, leading to permanent damage.

Compounds presented are able to modulate neurodegeneration by inhibiting Sema3A pathway, thus stimulating injured axons regeneration and brain repair.

Their therapeutic potential has been proved in pre-clinical (in vitro and in vivo) assays involving injure of the optic nerve. The Sema3A inhibitors were able to prevent loss of retinal ganglion cells (RGC) in a range of harsh optic degenerative insults using in vivo models, such as optical nerve transection in adult rat. Results showed between 30-40% or RGC survival after treatment with the compounds.

Main innovations and advantages

- The small molecules developed act as prolonged inhibitors of Sema3A apoptotic pathway.
- Application for treatment in the prevention of RGC loss associated to different optic neuropathies, currently ineffective, is envisioned thus promoting nerve regeneration.
- Potential application to other neurodegenerative processes associated to CNS disorders.

Patent Status

European patent application filed

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