An antibody against Semaphorin 3A for treatment of degeneration diseases of the optic nerve (Ramot)

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Glaucoma is a term which covers different pathological conditions leading to neuropathy, degeneration of the vulnerable optic nerve axons and cell bodies (Retinal Ganglion Cells = RGC). The most common glaucoma is associated with high intraocular pressure (IOP). The treatment today is based on lowering the IOP but is inefficient in preventing RGC loss. Thus there is an urgent need for treatment that protects the RGCs . From other side the worldwide prevalence of glaucoma is increasing. This is in part due to the rapidly aging population. The number of people with glaucoma worldwide will increase to 111.8 million in 2040, disproportionally affecting people residing in Asia and Africa. In 2010, 8.4M people worldwide were blind from primary open-angle glaucoma with incident growing to an estimated 11 M by 2020.

Semaphorin 3A (Sema3A) is a cell secreted protein that participates in the axonal guidance pathways. We were the first to show that Sema3A is also capable of inducing neuronal cell death. It was further shown that Sema3A is mediating vast RGC apoptosis following optic nerve injury. Importantly, marked inhibition of RGC loss was achieved when eyes with axotomized optic nerves were co-treated by intravitreous injection of antibodies against Sema3A providing the proof of concept for the therapeutic approach for neuroprotection via inhibiting the Sema3A pathway.

Many of the pathological mechanisms in glaucoma are apparent in acute optic nerve neuropathy which is characterized by neuronal death following stroke (an age related disease). Thus, this project goal is to develop a therapy for glaucoma and acute optic nerve neuropathy using the same therapeutic approach, i.e. inhibiting further death of vision related neural cells by prolonged inhibition of Sema3A apoptotic pathway.

We developed a Sema3A blocking human antibody using a phage library, to produce recombinant Fabs and IgGs and test their potential to antagonize sema3a in vitro and in an animal model.

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