Glaucoma Research Society of Canada

We Support New Ideas

2013 Research Grant Projects

Thanks to Our Donors
Researchers Receive $75,000 in Grants

Dr. Graham Trope, founder of the Glaucoma Research Society of Canada and chair of its Scientific Advisory Committee, recently announced the Society's 2013 research grants for the following five projects:

Studying the influence of retinal astrocytes on retinal ganglion cell survival

Glaucoma has remained a challenge to treat due to a complex combination of risk factors, which ultimately damage specialized neurons in the eye, called retinal ganglion cells.

This retinal damage is accompanied by a response from local astrocytes, which are important support cells that normally help to maintain neural function. In response to injury or disease, astrocytes *activate* secreting signals and enzymes to influence neuronal survival, coordinate remodeling of retinal tissues and producing antioxidants.

Astrocyte activation is a prominent early feature during glaucoma pathogenesis. However, the role of this response in the disease process remains unclear.

We have developed and established a system to isolate and activate retinal astrocytes in order to explore their direct influence on the injured retina, and characterize the molecular signals mediating this effect.

Further experiments will manipulate these pathways, with a goal towards introducing new treatment strategies aimed at moderating retinal astrocyte function.

– Dr. Jeremy Sivak, Dr. Izhar Livne-Bar, Toronto Western Research Institute, Toronto, Ontario

Examining the role of PEA15 in glial cell activation

In this research, we want to elucidate the molecular pathways involved in activating ONH and nerve fibre layer (NFL) glia in the aging eye, and developing glaucomatous optic neuropathy.

We propose to investigate PEA15 and its influence on activating NFL and ONH glia, and ultimately retinal ganglion cell (RGC) death.

We believe that the lamina cribrosa is exposed to physiologically significant, intra-ocular pressure-dependent biomechanical strain that causes ONH and nerve fibre layer (NFL) glia to transform to reactive phenotypes characterized by specific biomarkers, including PEA15. These reactive glia contribute to (RGC) apoptosis and hence the development of glaucomatous optic neuropathy.

Our specific hypothesis is that PEA15 is involved in mediating the astrocyte stress responses that lead to RGC death and glaucomatous optic neuropathy.

These experiments will examine the function of PEA15 in response to biomechanical insult.

We will evaluate studies through their effects on previously established optic nerve head astrocyte markers, cell stress and death, and its effects on cytoskeletal organization and components, such as the cytoskeletal proteins GFAP and actin.

– Dr. John G. Flanagan, Dr. Jeremy Sivak, Toronto Western Research Institute, Toronto, Ontario

Studying self-induced motion in people with glaucoma

Patients with glaucoma have difficulties navigating and are prone to falls. Mobility and balance control require information from visual, vestibular, and somatosensory systems. In certain conditions, an impaired visual input may trigger compensatory responses from the other systems.

The interaction between the visual and vestibular systems can be observed when exposing stationary observers to large moving stimuli. Such stimuli typically induce a sensation of self-motion in stationary observers.

Peripheral vision plays an important role in inducing self-motion and we have previously shown that this response is enhanced in patients with central vision loss. However, it is not yet known whether the response of peripheral vision to large moving stimuli is affected in patients with glaucoma and, if so, whether there are any compensatory responses from the vestibular system.

This research will shed more light into the functionality of peripheral vision of patients with glaucoma, into the interaction between visual and vestibular systems in the presence of peripheral visual field defects, and into whether there are any adaptive mechanisms to the visual loss due to glaucoma.
– Dr. Martin Steinbach, Toronto Western Research Institute, Toronto, Ontario

Determining whether a communication exists between the cerebrospinal fluid and the eye

We believe that cerebrospinal fluid (CSF) surrounding the optic nerve communicates with the eye.

In our current study, we will assess CSF communication to the eye and optic nerve head in normal mice.

We will use hyperspectral imaging in live mice to visualize quantum dots injected into their cerebrospinal fluid at various time points and to calculate the rate of communication to the eye. We will track the intensity of quantum dots drained into the lymph nodes of the mice and take non-invasive intraocular pressure measurements.

We will measure quantum dot intensity in harvested eye, orbit and neck tissue and examine their localization to describe the microanatomy of the communication between CSF compartment and the eye compartments.

Understanding the relationship between CSF and the normal eye will provide valuable insight into the role played by the CSF in eye health, and is highly relevant to further studies in glaucoma models.
– Dr. Yeni Yücel, Dr. Neeru Gupta, St. Michael's Hospital, Toronto, Ontario

Determining the biomechanic response of optic nerve head glial cells to combined stretch and compression

The cells of the lamina cribrosa in glaucomatous eyes experience high levels of compression and stretch as the tissue becomes deformed. We have developed a novel model that allows neural cells from the lamina cribrosa region of human optic nerves to be grown on a flexible surface enabling us to stretch and compress the cells. This causes the cells to respond and become active.

We can harvest the resulting proteins and RNA and analyze them to determine how the cells respond. By understanding their response, we will better understand how the deformation that occurs within the human optic nerve at the earliest stages in the development of glaucoma, may contribute to developing the disease.
– Kenneth Olsen, PhD Candidate, Dr. John G. Flanagan, Toronto Western Research Institute,