



The Glaucoma Foundation

2010 Annual Report

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Message from the President

Dear Friends:

2010 was a difficult and challenging year for everyone, and The Glaucoma Foundation was certainly not immune to the meaningful downturn in the economy. However, as the year ended, we were able to cite meaningful and measurable accomplishment in all key areas.

Our mission continues to embrace the funding of cutting-edge research that is being performed around the world by the best and the most talented investigators. They each offer a vision coupled with an idea, that if validated and achieved, may stand to make a meaningful difference in the diseases that we call glaucoma.

The second component of our core purpose is to provide educational outreach to all, relative to proper eye care and awareness about glaucoma. As we all understand, proper and timely diagnosis is essential to arresting the progress of this disease. We are continually reminded that our efforts have made a huge impact on behalf of the populations of the world.

During the year 2010, we hosted an award-worthy 17th Annual International Think Tank in New York City. Fifty four participants from around the world gathered to address: "The Complex Genetics and Genomics of Glaucoma." Enormous positive progress was demonstrated throughout the session, with the hope being that the same exciting report will be forthcoming from the 18th Annual Think Tank which will be held in September, 2011 once more in New York City.

Thanks to your generosity and commitment to us, revenue flows remained strong in most categories of gifts. The Black and White Ball honored glaucoma patient and world-renowned photographer, Elliott Erwitt, attracted over 300 guests and raised over \$500,000 in revenue. Expenses are analyzed continually for their value to the organization and are deemed by the Board to be well under control.

We are very proud of our Foundation and its accomplishments. We are also extremely excited about the future service that will be provided to all of our constituencies. We thank you for your support of and interest in The Glaucoma Foundation. You and we, as partners, can make a significant difference to the world in which we operate.

Scott R. Christensen
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Dean
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Director of Molecular Ophthalmic
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Weizmann Institute of Science

Gulgun Tezel, MD

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Ting Xie, PhD
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Stowers Institute

Michael Joseph Young, PhD
Director
Minda de Gunzburg Center for
Ocular Regeneration
Schepens Eye Research Institute
Associate Professor
Harvard Medical School

2010 RESEARCH GRANTS

AMERICAN GLAUCOMA SOCIETY

BRIAN CHRISTOPHER SAMUELS, MD, PhD

American Glaucoma Society

Development of a new experimental model of glaucoma in rats via reduction of intracerebral pressure

Glaucoma is one of the leading causes of blindness worldwide, yet we are still trying to develop a fundamental understanding of the pathophysiologic mechanisms underlying the retinal ganglion cell loss that originates at the lamina cribrosa.

KATALIN CSISZAR, PhD

John A. Burns School of Medicine, Honolulu, Hawaii

LOXL1-Associated pathomechanisms in pseudoexfoliation glaucoma

This study aims to uncover the association between the LOXL1 gene that results in the development of exfoliation syndrome (XFS) and exfoliative glaucoma (XFG). LOXL1 is a major genetic risk factor for XFS and XFG, but its exact role remains unknown. We will test the hypothesis that disease risk alleles of LOXL1 affect interactions of the LOXL1 protein with two regulatory proteases, BMP1 and Cathepsin B, adversely influencing LOXL1 activation or degradation with the consequent development of XFG. It is anticipated that the new data will identify the mechanism responsible for the development of XFS and advance the development of novel therapeutic approaches for the treatment of XFG.

FRANZ GRUS, MD, PhD

University Medical Center of the Johannes Gutenberg University, Mainz, Germany

Detailed analysis of the autoimmune component of normal-tension glaucoma via microarray screening

The immune system of glaucoma patients can attack some of the body's own ocular proteins. We will attempt to detect antigens specifically affected by antibodies in normal-tension glaucoma (NTG) patients. We will conduct a highly precise microarray approach analyzing the antibody patterns and especially the reactivities of different antibody subclasses in study groups from Germany and the U.S. Results of this study will give more detailed insights on antibody classes involved and draw

conclusions on further components of the immune system in glaucoma pathogenesis.

BRUCE R. KSANDER, PhD

Harvard Medical School, Boston, Massachusetts

Fas/FasL is a critical regulator of apoptosis and retinal degeneration in glaucoma

Retinal ganglion cells are the cells that transmit visual images through the optic nerve to the brain and that die in glaucoma. It is unclear to scientists exactly why these cells die. One of the signals that causes cells to die (called Fas Ligand or FasL) can be expressed in two different forms. The first triggers the cells to die, and the second prevents the cells from dying. Whether the retinal cells die ultimately depends upon which form of this signal prevails. We have developed mice that are genetically altered so that they only produce only the signal that triggers cell death. If these predictions are correct, then these mutant mice will display an accelerated and more severe form of glaucoma. Future studies would then be directed at developing mutant mice that only express the signal that blocks cell death. These mice should be resistant to glaucoma.

ERIN LAVIK, SB, SM, ScD

Case Western Reserve University, Cleveland, Ohio

A minimally invasive drug delivery approach to modify the ECM and promote neural regeneration in a model of glaucoma

Vision loss associated with glaucoma arises as a result of the loss of retinal ganglion cells and degeneration of the optic nerve. The neural degeneration in glaucoma is accompanied by extensive remodeling of the extracellular matrix, the environment of the optic nerve that inhibits repair. The degeneration also includes the loss of retinal ganglion cells. We propose to make the environment permissive for repair and replace the retinal ganglion cells. We will alter the environment by delivering a drug, AG1478, that has been shown to alter the environment and promote regeneration of the optic nerve. We will deliver the drug over 4 months, the time we estimate will be needed to promote repair, from injectable microspheres that deliver the drug as they degrade. We will replace the lost retinal ganglion cells with neural progenitor cells. We hypothesize that the combination of neural progenitor cells to replace lost retinal ganglion cells along with sustained delivery of AG1478 will promote robust regeneration. To test this approach, we will use an optic nerve crush model. The optic nerve crush model is an excellent first model for studying methods to promote regeneration in glaucoma because it causes similar changes in the environment and loss of cells and is very reproducible leading to clear results.

RICHARD T. LIBBY, PhD
University of Rochester, New York

JNK signaling is critical for retinal ganglion cell death after axonal injury

Loss of vision in glaucoma is caused by the death of a specific type of neuronal cell, the retinal ganglion cell (RGC, the neuron that sends information to the brain). Presently there are no widely available treatments aimed at neuroprotection. Unfortunately, this means that in many cases, physicians are left with no treatment options to prevent their patients from going blind. This project aims to determine the molecular signaling pathways responsible for killing RGCs in glaucoma. Identifying these molecules will provide important information about the complexity of the signaling pathways active in glaucoma, indicate which pathways could be targeted for glaucoma therapies, and identify potential genes that could account for the variability in susceptibility to glaucoma in different people.

YUTAO LIU, PhD
Duke University, Durham, North Carolina

Roles of regulatory variants for LOXL1 in pseudoexfoliation glaucoma

Exfoliation syndrome is the single most identifiable cause of open-angle glaucoma in the world. Coding variants in the lysyl oxidase-like 1 (LOXL1) gene are associated with the increased risk of exfoliation glaucoma across many different populations. New evidence suggests that the coding variants currently known are not the major cause of exfoliation glaucoma. This project will study the part of the LOXL1 gene that regulates its activity and may be responsible for causing exfoliation glaucoma. This is a crucial step in understanding how exfoliation glaucoma develops and will lead the way to new treatment approaches.

SHANNATH MERBS, MD, PhD
Johns Hopkins University, Baltimore, Maryland

DNA methylation changes associated with ganglion cell injury

Alterations in the expression of genes, caused by changes such as a gene mutation, can lead to disease. Another way to alter gene expression, without changing the DNA sequence of a gene, is to chemically modify the DNA by methylation. Abnormal DNA methylation has been shown to lead to some cancers. It is quite possible that DNA methylation changes could also contribute to the development of non-neoplastic diseases like glaucoma. To our knowledge, no one has ever looked at how DNA methylation changes might contribute to blinding eye diseases. This proposal will use an animal model to begin to look for DNA methylation changes that

are associated with ganglion cell death (the cells that die in glaucoma). A greater understanding of the role DNA methylation plays in eye diseases could lead to new treatment strategies. For example, drugs to manipulate DNA methylation have been used in the treatment of some cancers. These same strategies might someday prove useful as an adjunct to traditional eye pressure lowering approaches for the treatment of glaucoma.

XIUQIAN MU, MD, PhD

University at Buffalo, New York

Math5 target genes in retinal ganglion cell formation

The underlying cause for optic nerve damage and retinal ganglion cell death in glaucoma is not known. This hinders the development of efficient therapies. By understanding how retinal ganglion cells, the cell type that send out axons to form the optic nerve, form during embryonic development, we can find clues regarding why ganglion cells die in glaucoma and how to prevent it. This proposal focuses on the function of a transcription factor named Math5 in retinal ganglion cell formation. Transcription factors are a type of proteins that regulate the activities of other genes. Genetic studies have shown that Math5 is absolutely required for retinal ganglion cell formation, but how it functions is not known. In another word, we don't know what genes are turned on and off by Math5. Finding out these genes is the objective of this proposal. We will use a recently-developed technology, ChIP-seq, to achieve this goal. This technology uses specific antibody and ultrahigh-throughput DNA sequencing to identify the genes a transcription factor controls. The knowledge obtained from this study will guide us in future efforts trying to generate retinal ganglion cells in cell culture for cell replacement therapy for glaucoma.

VINCENT RAYMOND, MD, PhD (renewal)

Université Laval Hospital Research Center, Quebec City, Canada

Characterization of modifiers for open-angle glaucoma by candidate gene screening and genome wide linkage study

Genetic factors play a major role in the etiology of glaucoma. Fourteen chromosomal regions encode genes for primary open-angle glaucoma (POAG), the most common form of glaucoma, but only three of these genes have been identified: myocilin, optineurin and WDR36. The surprising occurrence of older individuals with healthy vision, despite the fact that they are carriers of myocilin mutations, raises the possibility that "good" genes, named protective modifier genes, maintain healthy vision by counteracting the effects of "bad" genes. The investigators recently found evidence for at least one of these modifier genes in the world's largest known glaucoma family. The goal of this study is to discover these modifier genes. Their

identification should offer novel and powerful approaches for discovering drugs to treat and perhaps prevent glaucoma.

MICHAL SCHWARTZ, PhD

Weizmann Institute of Science, Rehovot, Israel

Searching for a molecular mechanism to awaken dormant retinal stem cells: A therapeutic approach to glaucoma

Glaucoma is a major cause of blindness in the elderly. While treatments are available to lower pressure in the eye, thereby prevent continued damage, there is currently no cure for glaucoma, nor any therapy capable of inducing cell renewal in the damaged tissue. It has been suggested that stem cells, which can differentiate to form numerous cell types, could be used to replace the nerve cells in the retina that were damaged by the disease. Stem cells exist in the mammalian eye but are dormant. The present proposal aims to explore the reasons that the ocular stem cells are unable to divide and form new nerve cells, and to use this information as a basis for therapy aimed at awakening this quiescent stem cell population. We believe that activating these dormant stem cells is a promising therapy that would circumvent the need for donor stem cells, and their potential complications.

DEEPAK SHUKLA, PhD (renewal)

University of Illinois, Chicago

Novel peptides to understand herpetic damage to human trabecular meshwork via actin rich nanotubular structures

The cells of the trabecular meshwork help regulate the normal intraocular pressure. Herpes virus can infect and destroy these cells and also the optic nerve, causing serious damage. Our goal is to understand how the virus infects these cells and then design new agents to block that process. To achieve this goal we have identified certain cellular receptors that help the virus invasion process by forming nano-size structures for virus spread from cell to cell. We plan to destroy the ability of the virus to form such structures, using small but highly potent peptides that will affect multiple pathways in virus spread process. These peptides will be isolated by a specialized process and then tested for their ability to prevent the damage to the trabecular meshwork cells by ocular Herpes infection.

DAVID W. SRETAVAN, MD, PhD

University of California, San Francisco, CA

Micro & Nanotechnology-Based Bioplatfoms for High-Throughput Analysis of Axon-Glial Interactions in Glaucomatous Neuropathy

Improved clinical management of glaucoma requires a deeper understanding of disease mechanisms that damage the elongated processes (axons) of retinal nerve cells at the optic nerve head. We propose to develop a new type of highly versatile microplatform for glaucoma research that incorporates advances in micro and nanotechnology to provide researchers with unprecedented control over key experimental parameters such as reagent and pressure application to nerve cell bodies versus axons, and the spatial relationships between axons and glial cells. With this bioplatfom, researchers will be able to conduct high-throughput experimentation simultaneously on a hundred axons, and thus each experiment can provide the amount of data that currently might require several dozen rounds of experimentation. This technology has the potential of greatly accelerating glaucoma research towards a cure. In this application for TGF funding, we will fabricate and test this new generation of micro/nano research bioplatfoms with the ultimate aim of using these devices to analyze cellular communication between retinal axons and glial cells.

IRINA SURGUCHEVA, PhD

VA Medical Center, Kansas City, Missouri

Protein aggregation in glial cells of the optic nerve role in glaucoma

Vision loss in glaucoma is caused by damage to the optic nerve. This nerve acts like an electric cable with over a million wires. It is responsible for carrying images from the eye to the brain. This electric cable is composed of the endings of cells called retinal ganglion cells and another type of cell called astrocytes. Astrocytes support normal functions of neuron ending in the optic nerve. In glaucoma patients astrocytes die in the optic nerve because of excessive accumulation of abnormal proteins. In this application we propose to reveal why these abnormal proteins accumulate in cells. We also propose to unveil the mechanisms of death of astrocytes due to the accumulation of these abnormal proteins and find substances which may prevent their death.

COMPARATIVE FINANCIAL SUMMARY

| | <u>ASSETS</u> | |
|--|--------------------|--------------------|
| | <u>2010</u> | <u>2009</u> |
| CURRENT ASSETS | | |
| Cash and cash equivalents | \$ 542,514 | \$ 479,485 |
| Accounts receivable | 82,780 | 104,757 |
| Prepaid expense | <u>19,857</u> | <u>23,333</u> |
| Total current assets | <u>645,151</u> | <u>607,575</u> |
| EQUIPMENT, NET | <u>2,627</u> | <u>4,890</u> |
| OTHER ASSETS | | |
| Assets restricted for permanent endowments | | |
| Cash | 33,746 | 20,943 |
| Investments - equity securities | 2,650,409 | 2,198,628 |
| Investments - money market | <u>85,212</u> | <u>29,669</u> |
| Total other assets | <u>2,769,367</u> | <u>2,249,240</u> |
| Security deposit | <u>27,796</u> | <u>27,796</u> |
| Total other assets | <u>2,797,163</u> | <u>2,277,036</u> |
| TOTAL ASSETS | <u>\$3,444,941</u> | <u>\$2,889,501</u> |
| <u>LIABILITIES AND NET ASSETS</u> | | |
| CURRENT LIABILITIES | | |
| Accounts payable and accrued expenses | \$ 225,471 | \$ 203,363 |
| Grants payable | 64,994 | 87,500 |
| Charitable gift annuity-current portion | <u>840</u> | <u>840</u> |
| Total current liabilities | <u>291,305</u> | <u>291,703</u> |
| LONG-TERM LIABILITIES | | |
| Charitable gift annuity-long term portion | <u>2,125</u> | <u>2,965</u> |
| TOTAL LIABILITIES | <u>293,430</u> | <u>294,668</u> |
| NET ASSETS | | |
| Unrestricted | 382,144 | 345,593 |
| Permanently restricted | <u>2,769,367</u> | <u>2,249,240</u> |
| Total net assets | <u>3,151,511</u> | <u>2,594,833</u> |
| TOTAL LIABILITIES AND NET ASSETS | <u>\$3,444,941</u> | <u>\$2,889,501</u> |

COMPARATIVE STATEMENT OF ACTIVITIES

| | 2010 | | | 2010 <u>Totals</u> | 2009 | | | 2009 <u>Totals</u> |
|---|---------------------|-----------------------------------|-----------------------------------|-----------------------|---------------------|-----------------------------------|-----------------------------------|-----------------------|
| | <u>Unrestricted</u> | <u>Temporarily Restricted</u> | <u>Permanently Restricted</u> | | <u>Unrestricted</u> | <u>Temporarily Restricted</u> | <u>Permanently Restricted</u> | |
| Revenue | | | | | | | | |
| Support, contributions, and other revenue | | | | | | | | |
| Individual and corporate donations | \$1,550,534 | — | \$ 39,914 | \$1,590,448 | \$1,252,026 | \$ — | \$ 24,810 | \$1,276,836 |
| Fundraising benefit | 444,740 | — | — | 444,740 | 440,760 | — | — | 440,760 |
| Board designated restrictions | (480,213) | — | 480,213 | — | (503,854) | — | 503,854 | — |
| Net assets released from restrictions | — | — | — | — | — | — | — | — |
| Total support, contributions, and other revenue | <u>1,515,061</u> | <u>—</u> | <u>520,127</u> | <u>2,035,188</u> | <u>1,188,932</u> | <u>—</u> | <u>528,664</u> | <u>1,717,596</u> |
| Investment income | | | | | | | | |
| Interest and dividend income | 41,811 | — | — | 41,811 | 34,756 | — | — | 34,756 |
| Investment management fees | (39,543) | — | — | (39,543) | (28,733) | — | — | (28,733) |
| Net unrealized and realized gains (losses) on investments | 378,799 | — | — | 378,799 | 475,625 | — | — | 475,625 |
| Total investment income | <u>381,067</u> | <u>—</u> | <u>—</u> | <u>381,067</u> | <u>481,648</u> | <u>—</u> | <u>—</u> | <u>481,648</u> |
| Total revenue | <u>1,896,128</u> | <u>—</u> | <u>520,127</u> | <u>2,416,255</u> | <u>1,670,580</u> | <u>—</u> | <u>528,664</u> | <u>2,199,244</u> |
| Expenses | | | | | | | | |
| Operating expenses | | | | | | | | |
| Program services | 1,381,769 | — | — | 1,381,769 | 1,355,059 | — | — | 1,355,059 |
| Management and general | 53,535 | — | — | 53,535 | 52,247 | — | — | 52,247 |
| Fundraising | 201,023 | — | — | 201,023 | 197,433 | — | — | 197,433 |
| Total operating expenses | <u>1,636,327</u> | <u>—</u> | <u>—</u> | <u>1,636,327</u> | <u>1,604,739</u> | <u>—</u> | <u>—</u> | <u>1,604,739</u> |
| Fundraising benefit expenses | <u>223,250</u> | <u>—</u> | <u>—</u> | <u>223,250</u> | <u>193,274</u> | <u>—</u> | <u>—</u> | <u>193,274</u> |
| Total expenses | <u>1,859,577</u> | <u>—</u> | <u>—</u> | <u>1,859,577</u> | <u>1,798,013</u> | <u>—</u> | <u>—</u> | <u>1,798,013</u> |
| Change in net assets | 36,551 | — | 520,127 | 556,678 | (127,433) | — | 528,664 | 401,231 |
| Net assets, beginning of year | <u>345,593</u> | <u>—</u> | <u>2,249,240</u> | <u>2,594,833</u> | <u>473,026</u> | <u>—</u> | <u>1,720,576</u> | <u>2,193,602</u> |
| Net assets, end of year | <u>\$ 382,144</u> | <u>—</u> | <u>\$2,769,367</u> | <u>\$3,151,511</u> | <u>\$ 345,593</u> | <u>—</u> | <u>\$2,249,240</u> | <u>\$2,594,833</u> |