



The Glaucoma Foundation

2008 Annual Report

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Message from the President

Dear Friends:

2008 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. We annually provide our own awards as well as funding an American Glaucoma Society (AGS) Research Grant in the amount of \$40,000. Of the 9 Grants designated by the AGS, we are proud to be the only non-AGS and non-pharmaceutical company donor.

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 2008, we hosted an award-worthy 15th Annual International Think Tank in New York City. Fifty one participants from around the world gathered to address: "Current Status of Translational Nano-Medicine and Tissue Bioengineering in the Eye." Enormous positive progress was demonstrated throughout the session, with the hope being that the same exciting report will be forthcoming from the 16th Annual Think Tank which will be held in October, 2009 once more in New York City.

Thanks to your generosity and commitment to us, revenue flows remained strong in most categories of gifts. The only area that was well below historical averages was our legacy and bequest income. The Black and White Ball honored New York Governor, David Paterson, attracted 345 guests and raised nearly \$700,000 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.

Sincerely yours,

A handwritten signature in black ink, appearing to read 'Scott R. Christensen', with a long horizontal line extending to the right.

Scott R. Christensen
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Director of Molecular Ophthalmic
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University of Louisville School of
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Ophthalmic Consultant
Pfizer, Inc.

2008 RESEARCH GRANTS

TOM GLASER, MD, PhD

University of Michigan Medical School, Ann Arbor

ATOH7 (Math5) Mutations in Optic Nerve Aplasia

Retinal ganglion cell (RGC) neurons and their axons in the optic nerve are the targets of glaucoma disease pathology. This project studies ATOH7, a major gene discovered by the project team that controls the first step in the formation of RGC's from embryonic retinal stem cells. The project explores how mutations, identified within or near ATOH7, cause congenital absence of the optic nerve in two families. In one, they will compare the molecular properties of normal and mutant ATOH7 protein products. In the other, they will find the exact DNA change that causes this disease by high-resolution genomic analysis. Complementary studies will test whether halving the ATOH7 gene dosage affects the number of optic nerve axons. The results should help to guide future studies on RGC regeneration and optic nerve disease.

GARETH R. HOWELL, Ph.D.

The Jackson Laboratory, Bar Harbor, ME

Assessing Glial Activation in a Mouse Model of Glaucoma

Glaucoma is characterized by the degeneration of the optic nerve, which disrupts neurotransmission between the eye and the brain, leading to blindness. Glial cells are thought to play an important role in glaucoma. In a resting state, glial cells are supportive to neurons, but in response to stress, can become activated and damaging. It has been shown that glial cells in the optic nerve become activated in early stages of glaucoma. However, it is not known whether this is a primary cause of the disease, or occurs later as the disease progresses. Due to the experimental limitations imposed with human studies, mice are valuable complementary organisms both to study the complex mechanisms of glaucoma and to develop improved therapeutics. Utilizing a mouse model that reproduces important aspects of human glaucomas, we propose to determine the timing and extent of glial activation in relation to glaucomatous damage using a combination of gene and protein expression analyses. This will be one of the most wide-ranging investigations of the role of glial cells in glaucoma to date.

ALBERTO IZZOTTI, MD, PhD
University of Genoa, Italy

Analysis of Mitochondrion Involvement in the Pathogenesis of Primary Open-Angle Glaucoma

Glaucoma patients might have a genetic predisposition, rendering them more susceptible to free radical-induced damage. However, the source of oxidative stress remains to be identified. The aim of the study is to identify the relationship between oxidative stress and mitochondrial damage. In this study, mitochondrion-related molecular endpoints will be tested in the trabecular meshwork, the tissues involved in the regulation of aqueous humor outflow from the anterior chamber. Obtained data will be useful to clarify the interplay among different processes during primary open-angle glaucoma pathogenesis with particular reference to the sources of endogenous oxidative stress.

TATJANA C. JAKOBS, MD
Massachusetts General Hospital, Boston, MA

Single-Cell Imaging of Optic Nerve Astrocytes in Glaucoma

Ganglion cells are the only neurons in the retina that send axons to the brain via the optic nerve. Glaucoma leads to a progressive and irreversible loss of these cells, thereby severing the connection of an otherwise functional retina with the brain. Recent evidence suggests that a non-neural cell type in the optic nerve, astrocytes, might play an active role in the disease. Using a transgenic mouse strain in which astrocytes are labeled with a fluorescent protein and IOP has been increased, this project will follow damage in the optic nerve, especially during early stages of the disease. The goal is to visualize individual astrocytes in more detail than has been possible before.

PAULO D. KOEBERLE, Ph.D.
University of Toronto, Ontario, Canada

The Role of Extracellular Matrix Interactions in Retinal Ganglion Cell Survival and Growth Factor Neuroprotection

Glaucoma is a progressive disease that results in the programmed cell death of retinal ganglion cells (RGCs). A number of naturally occurring proteins known as neurotrophic factors have been shown to promote RGC survival and regeneration. The therapeutic use of neurotrophic factors has been limited due to a number of factors, including the loss of effectiveness when they are delivered for prolonged periods. Dr. Koeberle's research suggests that one factor contributing to the loss of effectiveness is the activation of enzymes that degrade the extracellular matrix surrounding nerve cells. This study will identify those critical matrix components and the signaling cascades that help promote cell survival in concert with signaling pathways that are activated by

neurotrophic factors. This will lead to the development of new avenues for using neurotrophic factors as effective therapeutics for glaucoma.

MARKUS H. KUEHN, Ph.D.

The University of Iowa, Iowa City

Genetic Characterization of a Novel Canine Model of Heritable Angle Closure Glaucoma

In primary angle closure glaucoma (PACG), the iris blocks the drainage of fluid from the eye through the trabecular meshwork. In the U.S., PACG accounts for about 10 percent of glaucoma, but in other countries, particularly in Asia, it represents the majority of cases. To date, genes associated with PACG have not been identified. The researchers recently identified a pedigree of Basset hounds afflicted with hereditary PACG, with features similar to those observed in humans. Preliminary genetic studies point to small regions of their genome which most likely contain the disease – causing mutation. The proposed project seeks to identify this mutation. Discovery of the responsible gene will enhance understanding of how this disease develops and may aid in early detection of at-risk persons and improve the ability to evaluate the effectiveness of treatment regimens.

CHRISTOPHER KAI SHUN LEUNG, MD, MB ChB, BMedSc, MSc

University Eye Center, Hong Kong Eye Hospital

In Vivo Imaging of Retinal Ganglion Cells – A New Model to Study Neuroprotection in Glaucoma

The goal of this project is to investigate the use of a novel in vivo imaging technique to monitor the longitudinal profile of retinal ganglion cell (RGC) damage in glaucoma and to study their response to a neuroprotectant, brain-derived neurotrophic factor (BDNF). An experimental model of glaucoma is induced in a strain of transgenic mice (Thy-1 CFP) that express cyan fluorescent protein (CFP) under the control of a Thy-1 promoter. Using a modified confocal scanning laser ophthalmoscope, RGC damage is detected as loss of fluorescent signals. BDNF is considered to be neuroprotective if it could either prevent the decrease of Thy-1 CFP expression or increase the expression of Thy-1 in fading RGCs. This imaging model offers a unique opportunity to monitor RGCs longitudinally and non-invasively, and will provide a new paradigm to study neuroprotection in glaucoma.

KEITH RG MARTIN, MA, DM, MRCP, FRCOphth
Cambridge Center for Brain Repair, United Kingdom

Does Tau Dysfunction Play a Role in Glaucoma?

Exactly how and why neurons die in glaucoma is not yet fully understood. Previous work suggests that blockage of the transport of survival factors from the brain to retinal neurons contributes to cell death in glaucoma. Similar transport problems occur in other neurodegenerative conditions such as Alzheimer's and multiple sclerosis. In these diseases, dysfunction of a protein called tau contributes to disrupted cellular transport. Tau is a small protein that stabilizes the tracks along which motor proteins transport their cargo (e.g. neuronal survival factors), much like cross ties keep railroad tracks firmly in place. There is strong preliminary evidence that tau dysfunction occurs in experimental glaucoma. This is exciting because drugs that modulate tau are available, including lithium and also newer agents with more favorable side-effect profiles. Investigators will test whether these drugs reduce neuron death in glaucoma and help to preserve sight.

DEREK MURPHY, Ph.D
Royal College of Surgeons in Ireland, Dublin

Evaluation of PEX Glaucoma-Associated Autoantigens as Disease Biomarkers and the Role of their Antigenic Targets in Retinal Neurodegeneration

Exploitation of the immune response of glaucoma patients has identified molecules that are of importance for diagnosis, disease development and potentially new therapies for the disease. We have established a unique collaboration between ophthalmologists and molecular biologists to develop protein arrays for the discovery of novel disease markers in glaucoma, and so contribute to the fields of diagnosis and molecular characterization of this disease. To this end, we have profiled the humoral immune responses in pseudoexfoliation syndrome (PEX) glaucoma patients, identifying disease associated autoantibodies in patients' sera. This project can contribute enormously to providing panels of unique markers for the development of a biochip assay to help in the correct diagnosis of this disease. These markers may also provide novel therapeutic targets for the specific prevention of retinal neural degeneration in glaucoma patients.

VINCENT RAYMOND, M.D., Ph.D.
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.

MANSOOR SARFARAZI, Ph.D.
University of Connecticut Health Center

Genome-Wide Association Study of Normal-Tension Primary Open-Angle Glaucoma

While elevated intraocular pressure (IOP) is the most important known risk factor for glaucoma, approximately 30 percent of primary open-angle glaucoma in the United States can be accounted for by non-IOP dependent risk factors, most commonly referred to as normal tension glaucoma (NTG). Dr. Sarfarazi’s group previously identified a defective gene that is primarily involved with the inherited forms of NTG. But for the majority of cases no specific gene is known. This study will use a subgroup of NTG cases and a similar number of matched control subjects and scan the genome with over 1.8 million land marked DNA markers. It is anticipated that a specific DNA marker will be identified that is highly associated with the NTG phenotype. Identification of such a DNA marker will lead the researchers to a specific gene or a known biological pathway, providing an early method of detection for NTG and promoting subsequent development of an effective medical therapy.

MICHAL SCHWARTZ, M.S.
Weizmann Institute of Science, Rehovot, Israel

Searching for a Molecular Mechanism to Awaken Dormant Retinal Stem Cells: A Therapeutic Approach to Glaucoma

While treatments are available to lower pressure in the eye, and thereby prevent continued damage from glaucoma, there is currently no cure for glaucoma nor any therapy capable of inducing cell renewal in the damaged tissue. Stem cells, which can differentiate to form numerous cell types, might be used to replace nerve cells in the retina that have been lost to glaucoma. Stem cells exist in the human eye but are dormant. Dr. Schwartz will explore the reasons why 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 these stem cells in order to circumvent the need for donor stem cells.

DEEPAK SHUKLA, Ph.D.
University of Illinois at Chicago

Novel Peptides to Understand Herpetic Damage to Human Trabecular Meshwork via Actin Rich Nanotubular Structures

The infection of human trabecular meshwork (TM) cells with herpes simplex virus leads to elevated intraocular pressure (IOP) and may contribute to the development of glaucoma, which is the second most common cause of permanent blindness in the United States. HSV-1 infection into TM is mediated by HVEM receptor in which long actin rich nanotubular structures (LARS) plays a major role during viral spread from one cell to another. Here, we plan to isolate peptides against HVEM to prevent virus from using HVEM receptors to invade cells and to understand virus interaction with LARS during viral spread. Our study will allow us to develop novel strategies to reduce the risk of glaucoma and prevent blindness.

DAVID W. SRETAVAN, M.D., Ph.D.
University of California, San Francisco, CA

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

Better understanding of the causes of damage to the axons of retinal ganglion cells should lead to improved treatment of glaucoma. This project will 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. With this bioplayers, researchers will be able to conduct high-throughput experimentation simultaneously on a hundred axons, providing the amount of data that currently might require several dozen rounds of experimentation. This project will fabricate and test this new generation of micro/nano research bioplayers with the ultimate aim of using these devices to analyze cellular communication between retinal axons and glial cells.

MICHAEL WALTER, Ph.D.
University of Alberta, Edmonton, Canada

Development of a Functional Assay for WDR36 (Renewal)

Finding the genes that cause glaucoma is the first step in improving early diagnosis and treatment. WDR36 has been proposed as a new primary open-angle glaucoma gene, but its role in the disease is controversial. While a number of nucleotide changes of WDR36 have been found in elevated frequency in glaucoma patients, proof that these alterations are disease-causing mutations awaits demonstration that these alterations

result in actual defects in WDR36 function. This group developed an assay to test the consequences of these DNA sequence changes and found that WDR36 mutations alter cellular processes, but only when a second gene is also mutated. They will now test if mutations of this second gene also cause glaucoma, and will investigate the cellular processes in which both genes are involved to determine the role of such processes in glaucoma.

XIANJUN ZHU, Ph.D.

The Jackson Laboratory, Bar Harbor, ME

Characterizing Microglial Activation in a Mouse Model of Glaucoma

Mice provide valuable models for molecular and mechanistic studies of glaucoma pathogenesis and for the rational development of neuroprotective therapy. DBA/2J mice provide an inherited glaucoma model that accurately reproduces many hallmarks of human glaucoma. Microglia are cells that appear to play an important role in glaucoma. However, their role is not clearly defined. This project aims at determining how the expression of various microglial genes change during DBA/2J glaucoma and to assess the relationship of these changes to glaucomatous damage. The researchers will also assess the role of a microglial enzyme in DBA/2J glaucoma. This will be one of the first experiments to functionally test the role of a specific microglial molecule in glaucoma.

2008 American Glaucoma Society Fellowship Grant

PRADEEP Y. RAMULU, M.D., Ph.D.

Wilmer Eye Institute, Baltimore, Maryland

Reading Impairment in Subjects with Bilateral Glaucoma

The impact of glaucoma on task performance has mainly been defined through questionnaire-based research, with few studies observing how individuals with glaucoma function. Here, we propose to:

- 1.) Evaluate whether reading is impaired in subjects with bilateral glaucoma through direct evaluation of reading performance. Reading performance will be primarily evaluated by measuring the reading speed of newspaper-sized text, and reading impairment will be defined as a reading speed of less than 90 words per minute, generally regarded as the speed necessary for fluent reading. Secondary reading tasks such as skimming will also be evaluated.
- 2.) Define conditions under which reading performance worsens for bilateral glaucoma patients. We will test reading abilities under different lighting conditions, with lower-contrast reading materials, with distraction, and over longer time durations.

COMPARATIVE FINANCIAL SUMMARY

	<u>ASSETS</u>	
	<u>2008</u>	<u>2007</u>
CURRENT ASSETS		
Cash and cash equivalents	\$ 569,603	\$ 894,206
Accounts receivable	208,694	157,723
Prepaid expense	<u>7,607</u>	<u>23,006</u>
Total current assets	<u>785,904</u>	<u>1,074,935</u>
EQUIPMENT, NET	<u>7,201</u>	<u>9,399</u>
OTHER ASSETS		
Assets restricted for permanent endowments		
Cash	26,082	-
Investments - equity securities	1,325,371	2,883,751
Investments - money market	<u>369,123</u>	<u>68,659</u>
	1,720,576	2,952,410
Security Deposit	<u>27,796</u>	<u>27,796</u>
Total other assets	<u>1,748,372</u>	<u>2,980,206</u>
TOTAL ASSETS	<u>\$2,541,477</u>	<u>\$4,064,540</u>
	<u>LIABILITIES AND NET ASSETS</u>	
CURRENT LIABILITIES		
Accounts payable and accrued expenses	\$ 343,230	\$318,911
Charitable gift annuity-current portion	<u>840</u>	<u>840</u>
Total current liabilities	344,070	319,751
LONG-TERM LIABILITIES		
Charitable gift annuity-long term portion	<u>3,805</u>	<u>4,645</u>
TOTAL LIABILITIES	<u>347,875</u>	<u>324,396</u>
NET ASSETS		
Unrestricted	473,026	787,734
Permanently restricted	<u>1,720,576</u>	<u>2,952,410</u>
Total net assets	<u>2,193,602</u>	<u>3,740,144</u>
TOTAL LIABILITIES AND NET ASSETS	<u>\$2,541,477</u>	<u>\$4,064,540</u>

COMPARATIVE STATEMENT OF ACTIVITIES

	2008			2007				
	<u>2008 Unrestricted</u>	<u>Temporarily Restricted</u>	<u>Permanently Restricted</u>	<u>2008 Totals</u>	<u>2007 Unrestricted</u>	<u>Temporarily Restricted</u>	<u>Permanently Restricted</u>	<u>2007 Totals</u>
<i>Revenue</i>								
<i>Support, contributions, and other revenue</i>								
<i>Individual and corporate donations</i>	\$1,081,517	\$ —	\$ 65,913	\$1,147,430	\$ 1,406,070	\$ —	\$ 74,894	\$1,480,964
<i>Fundraising benefit</i>	698,498	—	—	698,498	684,006	—	—	684,006
<i>Board designated restrictions</i>	1,297,747	—	(1,297,747)	—	(484,029)	—	484,029	—
<i>Net assets released from restrictions</i>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>
<i>Total support, contributions, and other revenue</i>	<u>3,077,762</u>	<u>—</u>	<u>(1,231,834)</u>	<u>1,845,928</u>	<u>1,606,047</u>	<u>—</u>	<u>558,923</u>	<u>2,164,970</u>
<i>Investment income</i>								
<i>Interest and dividend income</i>	59,979	—	—	59,979	53,219	—	—	53,219
<i>Investment management fees</i>	(47,805)	—	—	(47,805)	(47,157)	—	—	(47,157)
<i>Net unrealized and realized gains on investments</i>	<u>(1,476,933)</u>	<u>—</u>	<u>—</u>	<u>(1,476,933)</u>	<u>403,387</u>	<u>—</u>	<u>—</u>	<u>403,387</u>
<i>Total investment income</i>	<u>(1,464,759)</u>	<u>—</u>	<u>—</u>	<u>(1,464,759)</u>	<u>409,449</u>	<u>—</u>	<u>—</u>	<u>409,449</u>
<i>Total revenue</i>	<u>1,613,003</u>	<u>—</u>	<u>(1,231,834)</u>	<u>381,169</u>	<u>2,015,496</u>	<u>—</u>	<u>558,923</u>	<u>2,574,419</u>
<i>Expenses</i>								
<i>Operating expenses</i>								
<i>Program services</i>	1,423,628	—	—	1,423,628	1,390,204	—	—	1,390,204
<i>Management and general</i>	55,223	—	—	55,223	54,849	—	—	54,849
<i>Fundraising</i>	<u>203,131</u>	<u>—</u>	<u>—</u>	<u>203,131</u>	<u>219,707</u>	<u>—</u>	<u>—</u>	<u>219,707</u>
<i>Total operating expenses</i>	<u>1,681,982</u>	<u>—</u>	<u>—</u>	<u>1,681,982</u>	<u>1,664,760</u>	<u>—</u>	<u>—</u>	<u>1,664,760</u>
<i>Fundraising benefit expenses</i>	<u>245,729</u>	<u>—</u>	<u>—</u>	<u>245,729</u>	<u>233,062</u>	<u>—</u>	<u>—</u>	<u>233,062</u>
<i>Total expenses</i>	<u>1,927,711</u>	<u>—</u>	<u>—</u>	<u>1,927,711</u>	<u>1,897,822</u>	<u>—</u>	<u>—</u>	<u>1,897,822</u>
<i>Change in net assets</i>	(314,708)	—	(1,231,834)	(1,546,542)	117,674	—	558,923	676,597
<i>Net assets, beginning of year</i>	<u>787,734</u>	<u>—</u>	<u>2,952,410</u>	<u>3,740,144</u>	<u>670,060</u>	<u>—</u>	<u>2,393,487</u>	<u>3,063,547</u>
<i>Net assets, end of year</i>	<u>\$ 473,026</u>	<u>\$ —</u>	<u>\$ 1,720,576</u>	<u>\$2,193,602</u>	<u>\$ 787,734</u>	<u>\$ —</u>	<u>\$ 2,952,410</u>	<u>\$3,740,144</u>