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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,

Scott R. Christensen
President
Chief Executive Officer
Board of Directors

Gregory K. Harmon, M.D.
Chairman
New York, NY

Robert Ritch, M.D.
Medical Director, Vice President,
Secretary & Founder
Professor of Clinical Ophthalmology
Chief, Glaucoma Service
The New York Eye & Ear Infirmary
New York, NY

Joseph M. La Motta
Chairman Emeritus
Pound Ridge, NY

William C. Baker
New York, NY

Debora K. Grobman, Esq.
New York, NY

Stephen D. Barkin
Stephen D. Barkin Real Estate
New York, NY

Barbara W. Hearst
Charleston, SC

Joseph M. Cohen
J.M.Cohen & Company
New York, NY

Chuck F.V. Imhof
American Airlines Inc.
New York, NY

Peter J. Crowley
CIBC World Markets
New York, NY

Gerald Kaiser, Esq.
Old Westbury, NY

David Cushman
Orvis/Cushman & Wakefield of California, Inc.
Los Angeles, CA

Paul Kaufman, M.D.
University of Wisconsin-Madison
Madison, WI

Rutledge Ellis-Behnke, PhD
Massachusetts Institute of Technology
Cambridge, MA

Theodore Krupin, M.D.
Northwestern Medical School
Chicago, IL

Barbara W. Hearst
Charleston, SC

Susan A. Murphy
Santa Fe, NM

David Cushman
Orvis/Cushman & Wakefield of California, Inc.
Los Angeles, CA

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University of Wisconsin-Madison
Madison, WI

Susan LaVenture
National Association for Parents of Children with Visual Impairments
Watertown, MA

Kenneth Mortenson
New York, NY

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Northwestern Medical School
Chicago, IL

Mary Jane Voelker
Pueblo, CO

Maurice H. Luntz, M.D.
The Mount Sinai School of Medicine
Manhattan Eye, Ear & Throat Hospital
NYU School of Medicine
New York, NY

Kenneth Mortenson
New York, NY

Jeffrey M. Liebmann, M.D.
The New York Eye & Ear Infirmary
New York, NY

Sheldon M. Siegel
Boca Raton, FL

Murray Fingeret, O.D.
St. Albans VA Medical Center
Hewlett, NY

James C. Tsai, MD
Yale School of Medicine
New Haven, CT

Ilene Giaquinta
New York, NY

Mary Jane Voelker
Pueblo, CO

Jeffrey M. Liebmann, M.D.
The New York Eye & Ear Infirmary
New York, NY

Irving Wolbrom
New York, NY

Maurice H. Luntz, M.D.
The Mount Sinai School of Medicine
Manhattan Eye, Ear & Throat Hospital
NYU School of Medicine
New York, NY

Alcon Laboratories, Inc.
Kevin Buehler
Fort Worth, TX

Pfizer, Inc.
Dennis Kowalski
New York, NY

Allergan, Inc.
Julian Gangolli
Irvine, CA

Kevin Buehler
Fort Worth, TX
Robert Ritch, M.D.  
Chairman  
Professor of Clinical Ophthalmology  
Chief, Glaucoma Service  
Surgeon Director  
New York Eye & Ear Infirmary

Terete Borrás, Ph.D.  
Professor of Ophthalmology  
University of North Carolina

Claude F. Burgoyne, M.D.  
Senior Scientist and Research Director  
Optic Nerve Head Research Laboratory  
Devers Eye Institute & Research Laboratories

Adriana DiPolo, Ph.D.  
Associate Professor  
Department of Pathology & Cell Biology  
University of Montreal

Rutledge Ellis-Behnke, Ph.D.  
Principle Investigator  
Department of Brain & Cognitive Sciences  
Massachusetts Institute of Technology

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Associate Professor  
Department of Ophthalmology & Visual Sciences  
Carver College of Medicine, University of Iowa

Jeffrey L. Goldberg, M.D., Ph.D.  
Research Assistant Professor  
Department of Ophthalmology  
McKnight Vision Research Center  
Bascom Palmer Eye Institute

John W. Grunden, Pharm.D.  
Senior Director, Team Leader  
Ophthalmology  
Global Medical Organization  
Pfizer, Inc.

Neeru Gupta, M.D., Ph.D.  
Dean  
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Ophthalmology & Vision Science, Laboratory Medicine & Pathobiology  
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Feinberg School of Medicine, Northwestern University

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Howard Hughes Medical Center  
The Jackson Laboratory

Chris Johnson, Ph.D.  
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Devers Eye Institute

Paul L. Kaufman, M.D.  
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Director of Glaucoma Services  
University of Wisconsin-Madison Medical School, Hospital & Clinics

Theodore Krupin, M.D.  
Professor of Ophthalmology  
Northwestern University Medical School  
University Eye Specialists

James F. Leary, Ph.D.  
Professor of Biomedical Engineering, SVM Professor of Nanomedicine  
Weldon School of Biomedical Engineering  
Purdue University

Leonard A. Levin, M.D., Ph.D.  
Associate Professor of Ophthalmology & Visual Sciences, Neurology, & Neurological Surgery  
University of Wisconsin Medical School

Jeffrey M. Liebmann, M.D.  
Clinical Professor of Ophthalmology  
New York University, School of Medicine  
Director, Glaucoma Service – New York University Medical Center & Manhattan Eye, Ear, Throat Hospital

Carlo D. Montemagno, Ph.D.  
Dean  
College of Engineering  
University of Cincinnati

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Department of Ophthalmology & Visual Science  
University of Wisconsin Medical School

Julia E. Richards, Ph.D.  
Associate Professor  
Department of Epidemiology  
Department of Ophthalmology & Visual Sciences;  
W.K. Kellogg Eye Center  
University of Michigan

Mansoor Sarfarazi, Ph.D.  
Professor of Human Molecular Genetics  
Director of Molecular Ophthalmic Genetics Lab  
University of Connecticut Health Center

Ursula Schlötzer-Schrehardt, Ph.D.  
Professor  
Department of Ophthalmology  
University of Erlangen-Nurnberg

Joel Schuman, M.D.  
Eye and Ear Foundation Professor and Chairman, Department of Ophthalmology  
University of Pittsburgh

Michal Schwartz, Ph.D.  
Professor of Neuroimmunology  
Weizmann Institute of Science, Israel

Michael A. Walter, Ph.D.  
Associate Professor  
Ocular Genetics Laboratory  
University of Alberta

Martin B. Wax, M.D.  
Vice President  
Research & Development  
Alcon Laboratories, Inc.

Robert N. Weinreb, M.D.  
Professor & Vice Chairman, Department of Ophthalmology  
University of California-San Diego  
Shiley Eye Center

Larry Wheeler
Senior Vice President, Biological Sciences
Allergan, Inc.

M. Roy Wilson, M.D., M.S.
President
Texas Tech University Health Sciences Center

Michael Joseph Young, Ph.D.
Assistant Professor of Ophthalmology
Harvard Medical School
Schepens Eye Research Institute

Ting Xie, Ph.D.
Investigator
Stowers Institute

Thom J. Zimmerman, M.D., Ph.D.
Emeritus Professor & Chairman, Department of Ophthalmology & Visual Sciences
Kentucky Lions Eye Center
University of Louisville School of Medicine
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 neurotropic 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 never cells dies 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.
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.

Micro & Nanotechnology-Based Bioplatforms 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 bioplatform, 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 bioplatforms with the ultimate aim of using these devices to analyze cellular communication between retinal axons and glial cells.

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

### ASSETS

<table>
<thead>
<tr>
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<th>2008</th>
<th>2007</th>
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<tbody>
<tr>
<td><strong>CURRENT ASSETS</strong></td>
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<tr>
<td>Cash and cash equivalents</td>
<td>$ 569,603</td>
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<td>Accounts receivable</td>
<td>208,694</td>
<td>157,723</td>
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<td>Prepaid expense</td>
<td>7,607</td>
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<td><strong>Total current assets</strong></td>
<td>785,904</td>
<td>1,074,935</td>
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<tr>
<td><strong>EQUIPMENT, NET</strong></td>
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<td>9,399</td>
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<tr>
<td><strong>OTHER ASSETS</strong></td>
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<tr>
<td>Assets restricted for permanent endowments</td>
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<tr>
<td>Cash</td>
<td>26,082</td>
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<tr>
<td>Investments - equity securities</td>
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<tr>
<td>Investments - money market</td>
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<td><strong>Total other assets</strong></td>
<td>1,748,372</td>
<td>2,980,206</td>
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<tr>
<td><strong>TOTAL ASSETS</strong></td>
<td>$2,541,477</td>
<td>$4,064,540</td>
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### LIABILITIES AND NET ASSETS

<table>
<thead>
<tr>
<th></th>
<th>2008</th>
<th>2007</th>
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<tbody>
<tr>
<td><strong>CURRENT LIABILITIES</strong></td>
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<tr>
<td>Accounts payable and accrued expenses</td>
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<td>Charitable gift annuity-current portion</td>
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<td>840</td>
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<tr>
<td><strong>Total current liabilities</strong></td>
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<td>319,751</td>
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<tr>
<td><strong>LONG-TERM LIABILITIES</strong></td>
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<tr>
<td>Charitable gift annuity-long term portion</td>
<td>3,805</td>
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<tr>
<td><strong>TOTAL LIABILITIES</strong></td>
<td>347,875</td>
<td>324,396</td>
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<td><strong>NET ASSETS</strong></td>
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<tr>
<td>Unrestricted</td>
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<td>Permanently restricted</td>
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<tr>
<td><strong>Total net assets</strong></td>
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<td>3,740,144</td>
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<tr>
<td><strong>TOTAL LIABILITIES AND NET ASSETS</strong></td>
<td>$2,541,477</td>
<td>$4,064,540</td>
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## COMPARATIVE STATEMENT OF ACTIVITIES

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<th>Unrestricted</th>
<th>Temporarily Restricted</th>
<th>Permanently Restricted</th>
<th>Totals</th>
<th>Unrestricted</th>
<th>Temporarily Restricted</th>
<th>Permanently Restricted</th>
<th>Totals</th>
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<tr>
<td><strong>Revenue</strong></td>
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<tr>
<td>Support, contributions, and other revenue</td>
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<tr>
<td>Individual and corporate donations</td>
<td>$1,081,517</td>
<td>$ —</td>
<td>$ 65,913</td>
<td>$1,147,430</td>
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<td>$ 1,406,070</td>
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<td>Fundraising benefit</td>
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<td>684,006</td>
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<tr>
<td>Board designated restrictions</td>
<td>1,297,747</td>
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<td>(1,297,747)</td>
<td>648,029</td>
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<td>484,029</td>
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<tr>
<td>Net assets released from restrictions</td>
<td></td>
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<tr>
<td><strong>Total support, contributions, and other revenue</strong></td>
<td>$3,077,762</td>
<td></td>
<td>(1,231,834)</td>
<td>1,845,928</td>
<td></td>
<td>1,606,047</td>
<td></td>
<td>558,923</td>
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<tr>
<td><strong>Investment income</strong></td>
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<tr>
<td>Interest and dividend income</td>
<td>59,979</td>
<td></td>
<td></td>
<td>59,979</td>
<td>53,219</td>
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<tr>
<td>Investment management fees</td>
<td>(47,805)</td>
<td></td>
<td></td>
<td>(47,805)</td>
<td>(47,157)</td>
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<tr>
<td>Net unrealized and realized gains on investments</td>
<td>(1,476,933)</td>
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<td>(1,476,933)</td>
<td>403,387</td>
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<tr>
<td><strong>Total investment income</strong></td>
<td>(1,464,759)</td>
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<td>(1,464,759)</td>
<td>409,449</td>
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<tr>
<td><strong>Total revenue</strong></td>
<td>1,613,003</td>
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<td>(1,231,834)</td>
<td>381,169</td>
<td></td>
<td>2,015,496</td>
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<td>558,923</td>
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<tr>
<td><strong>Expenses</strong></td>
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<td>Operating expenses</td>
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<td></td>
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<td>Program services</td>
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<td>Management and general</td>
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<td>54,849</td>
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<td>Fundraising</td>
<td>203,131</td>
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<td>203,131</td>
<td>219,707</td>
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<tr>
<td><strong>Total operating expenses</strong></td>
<td>1,681,982</td>
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<td>1,681,982</td>
<td>1,664,760</td>
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<td>Fundraising benefit expenses</td>
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<td>245,729</td>
<td>233,062</td>
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<tr>
<td><strong>Total expenses</strong></td>
<td>1,927,711</td>
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<td>1,897,822</td>
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<tr>
<td>Change in net assets</td>
<td>(314,708)</td>
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<td>(1,231,834)</td>
<td>(1,546,542)</td>
<td>117,674</td>
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<td>558,923</td>
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<tr>
<td>Net assets, beginning of year</td>
<td>787,734</td>
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<td>2,952,410</td>
<td>3,740,144</td>
<td>670,060</td>
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<td>2,393,487</td>
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<tr>
<td><strong>Net assets, end of year</strong></td>
<td>$473,026</td>
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<td>$1,720,576</td>
<td>$2,193,602</td>
<td>$787,734</td>
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<td>$2,952,410</td>
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