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Message From The President

Dear Friends:

2007 was another outstanding year for The Glaucoma Foundation. 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-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 2007, we hosted an award-worthy 14th Annual International Think Tank in New York City. Fifty five participants from around the world gathered to address: “High Resolution Imaging of the Eye: Advanced Optics, Microtechnology and Nanotechnology.” Enormous positive progress was demonstrated throughout the session, with the hope being that the same exciting report will be forthcoming from the 15th Annual Think Tank which will be held in September, 2008 once more in New York City.

Revenue flows remained strong in all categories of gifts. A significant portion of our legacy and bequest income was deposited into our Endowment account and coupled with an outstanding investment performance allowed our Endowment to grow to over $2.9 million. The Black and White Ball attracted 325 guests and raised nearly $700,000 in revenue. On the other hand, expenses are analyzed continually for their value to the organization. They 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

Joseph M. La Motta
Chairman Emeritus
Pound Ridge, NY

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

William C. Baker
New York, NY

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

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

Peter J. Crowley
CIBC World Markets
New York, NY

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

Donald Engel
New York, NY

David Fellows
Vistakon
Jacksonville, FL

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

Ilene Giaquinta
New York, NY

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New York, NY

Barbara W. Hearst
Charleston, SC

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

Gerald Kaiser, Esq.
Old Westbury, NY

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

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

Kenneth Mortenson
New York, NY

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Santa Fe, NM

Sheldon M. Siegel
Boca Raton, FL

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Yale School of Medicine
New Haven, CT

Mary Jane Voelker
Pueblo, CO

Irving Wolbrom
New York, NY

Alcon Laboratories, Inc.
Kevin Buehler
Fort Worth, TX

Allergan, Inc.
Julian Gangolli
Irvine, CA

Pfizer, Inc.
Dennis Kowalski
New York, NY
Scientific Advisory Board

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

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

Neeru Gupta, M.D., Ph.D.
Dean
Associate Professor
Ophthalmology & Vision Science, Laboratory Medicine & Pathobiology
University of Toronto

M. Rosario Hernandez, DDS
Professor of Ophthalmology
Feinberg School of Medicine, Northwestern University

Simon John, Ph.D.
Assistant Investigator
Howard Hughes Medical Center
The Jackson Laboratory

Chris Johnson, Ph.D.
Director of Diagnostic Research & Senior Scientist
Devers Eye Institute

Paul L. Kaufman, M.D.
Professor of Ophthalmology & Visual Sciences Director of Glaucoma Services

University of Wisconsin-Madison Medical School, Hospital & Clinics
Yoshiaki Kitazawa, M.D., Ph.D.
Professor Emeritus
Director
Akasaka Kitazawa Eye Clinic
Kitazawa Glaucoma Research Laboratories, Japan

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

Robert Nickells, M.D.
Associate Professor
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

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.

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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

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.
2007 Research Grants

NADEAN L. BROWN, Ph.D.
Children’s Hospital Research Foundation
Cincinnati, OH

**In vivo Investigation of Optic Nerve Formation and Connectivity within the Mouse Brain**

The goal of this project is to understand how retinal neurons grow out of the mammalian eye, assemble into functional nerves and establish the correct connections with the brain. Each of these steps is essential for the images an eye sees to be properly interpreted by the brain. To accomplish this, the researchers created a transgenic mouse model in which the developing optic nerve is labeled in living mouse embryos. This project will place the growing retina from these embryos in culture, by itself, or with the appropriate brain tissues and study optic nerve formation. Using this system, the project will test the ability of the factor oncomodulin, which stimulates adult optic nerve regeneration, to direct embryonic optic nerve formation. It will also test the ability of oncomodulin to restore mutant optic neuron outgrowth into the brain. These studies will provide crucial information about the requirements for initially creating the optic nerve versus regenerating it.

ROBERT O. DUNCAN, Ph.D.
Hamilton Glaucoma Center
University of California, San Diego

**Functional Magnetic Resonance Imaging (fMRI) of Function – Specific Vision Loss in Glaucoma**

If left untreated, glaucoma eventually results in the death of cells in the eye that relay visual information to the brain. Animal studies have shown that the loss of these cells, in turn, has detrimental consequences for the cells in the brain. There are three primary pathways that relay different aspects of the visual scene from the eye to the brain: the magnocellular, the parvocellular, and the koniocellular pathway. This study aims to determine if any of the three primary visual pathways is affected differentially by glaucoma. Functional magnetic resonance imaging (fMRI) will be used to compare cortical responses to visual stimuli that differentially stimulate one of these three pathways. The experiments should demonstrate which, if any, of these functionally distinct neural pathways is most affected by human glaucoma. Understanding how the visual pathway from the optic nerve to the brain is affected by glaucoma will provide insights into the pathology of the disease, which may guide future research for neuroprotective, genetic, and molecular therapies.
CD44-Osteopontin Interaction in Axonal Outgrowth of Retinal Ganglion Neurons

Progressive irreversible blinding diseases collectively called glaucoma cause damage to the optic nerve. To design strategies to rescue the injured nerve, it is necessary to understand the underlying processes in nerve growth. Therefore, it is essential to identify proteins that are involved in axonal growth of retinal ganglion cells (RGCs) during development as well as in the mature central nervous system (CNS). The laboratory uses a completely new approach that involves the design of a computer program suite collecting protein names from literature relevant to “nerve regenerations.” They identified two proteins that interact with each other that have never been investigated regarding their importance in axonal outgrowth of RGCs and were able to demonstrate the role of these two proteins in neurite growth of embryonic RGCs. Further experiments will investigate the expression pattern of these two proteins in embryonic as well as mature brains. We will also use genetically modified mice, so-called knock-out mice, to perform additional studies on the biological function of these proteins in the visual system.

Developmental Determinants of Retinal Ganglion Cell Regenerative Ability (Renewal)

In glaucoma, axons of mature retinal ganglion cells (RGC) do not regenerate into the optic nerve. The vast majority of regenerative research has focused on identifying extrinsic glial-associated inhibitors of regeneration. This has been fruitful, yet overcoming the inhibitory environment leads to only a small fraction of regenerative response. In this proposal Dr. Goldberg will continue to investigate the molecular basis for the developmental loss of intrinsic axon growth ability in RGR in vitro and in vivo, screening developmentally-regulated RGC genes for an ability to improve axon growth. This approach has the opportunity to open a conceptual breakthrough into the failure of RGR regeneration, and to lead to entirely new molecular insights and thus to new strategies to “revert” mature RGR to their greater embryonic axon growth ability.
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.

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. It is hoped that this will lead to the development of new avenues for using neurotropic factors as effective therapeutics for glaucoma.
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 US, 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.

Intranasal Application of Neuroprotective Agents in Rats with Glaucoma

There is evidence to suggest that disruption of the blood flow to the retina and optic nerve in patients with glaucoma may in part explain the loss of the cells of the retina in these patients. The researchers have characterized a model of retinal and optic nerve injury that is caused by hypoxia to these tissues, which is loss of oxygen due to temporary disruption of the blood supply. They will test a novel method of drug administration, intranasal application, to determine whether this method of treatment can rescue the retinal cells and optic nerve axons that had been exposed to a short-lived disruption of blood flow resulting in ischemia. They will examine the potential efficacy of insulin growth factor-1, a hormone known with neuroprotective effects in stroke, retinal and spinal cord injury, but whose systemic side effects from high doses are not acceptable for patient use. The study will also test erythropoietin, another neuroprotective candidate molecule. Intranasal drug application results in higher effective doses to the tissues of the nervous system than systemic applications.

Three Dimensional Reconstruction of the Lamina Cribrosa using Second Harmonic Imaging Microscopy

Advancing age and increasing intraocular pressure (IOP) are risk factors for progression of glaucoma. Experimental studies have demonstrated that the initial injury in glaucoma
is in the lamina cribrosa or scleral portion of the optic nerve head where nerve cells from the retina form the optic nerve and ascend toward the brain. Though numerous studies have examined the lamina cribrosa, detailed knowledge as to the effects of IOP on its organization and structure are very limited. This project will use a new technology to visualize the three-dimensional structure of the lamina cribrosa at a very high resolution using non-invasive second harmonic imaging microscopy (SHIM). This technique allows for direct measurement of the structural changes in the lamina caused by IOP that avoid many of the problems and artifacts of past methods. These data should provide critically important insights as to how IOP causes vision damage.

DEREK MURPHY, Ph.D.
Centre for Human Proteomics
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.

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

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.

VALERY SHESTOPALOV, Ph.D.
Bascom Palmer Eye Institute
University of Miami, FL

The role of glial NF-kappaB in retinal ganglion cell loss in glaucoma

This project aims to investigate the effect of the cellular environment, specifically the neural glia, on the survival of RGC. The death of these neurons, which communicate visual information to the brain, causes blindness in glaucoma. Utilizing a transgenic mouse strain possessing genetically inactivated nuclear factor-kappaB (NF-kB), this research will test the hypothesis that NF-kB plays a key role in converting the normally supportive neuronal environment into a noxious, reactive one. The mouse strain will allow the researchers to examine whether the genetic inactivation of this complex will protect these neurons. Comparing neuronal death rates in normal and transgenic mice will determine the effect of NF-kB activation directly in animal retinas. This knowledge may provide novel targets for both prevention and molecular therapy of glaucoma.

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 the 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.

ANDREI SURGUCHOV, Ph.D.
Kansas University Medical Center
Kansas City, MO

New Mechanism of MMP-9 Regulation and its Role in Glaucoma

Metalloproteinase-9 (MMP-9) is an enzyme that is implicated in retinal damage and alterations in the optic nerve in glaucoma. Despite the important functions of MMP-9 in glaucoma, its role in pathology is not completely understood. Recent data suggest that defects in MMP-9 production leading to its excessive accumulation may be a key step in
glaucoma and probably other eye diseases. In previous studies, the researchers found new potent activators of MMP-9 production that may play a significant role in ocular diseases. This upregulation is induced by synucleins, proteins that are expressed in the retina and the optic nerve, but their exact role in these illnesses is not known. This study will investigate the mechanism of increased MMP-9 production caused by synucleins and elucidate the implication of this mechanism in eye pathologies. The results will provide important insights into understanding the mechanisms and developing treatments for these ocular pathologies.

DOUGLAS E. VOLLRATH, M.D., Ph.D
Stanford University School of Medicine
Stanford, CA

Chemical Genetic Screen for Compounds that Enhance Secretion of Mutant Myocilin

This project investigates an inherited form of glaucoma that affects thousands of Americans. Unlike more common forms of glaucoma, the mutant gene that causes this disorder is known. The study’s goal is to understand how the mutant protein encoded by the gene causes the disease, and by solving this tractable problem, to gain insight into some of the causes of more common forms of glaucoma. Current results show that the mutant protein has an abnormal shape and is not properly released from cells. When cells derived from the front of the eye make the mutant protein, they become sick and die. These particular cells are known to be important in draining fluid from the eye, so their loss could well explain how the mutant gene/protein causes glaucoma. When these cells are grown at temperatures a little below body temperature, the mutant protein is released from the cells and the cells no longer die. The research team proposes to find drugs that stimulate release of the mutant protein from cells at body temperature and hopes that identification of such compounds will lead to development of new forms of therapy for this type of glaucoma and encourage similar investigations into other forms of glaucoma.

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

Development of a Functional Assay for WDR36

The WD40 repeat 36 (WDR36) gene has recently been identified as a new primary open angle glaucoma locus. However, the function of WDR36 and its role in glaucoma pathogenesis are unknown. One of the important challenges presented by an adult-onset disease such as glaucoma is deciding if a DNA change seen in a patient causes the disease or is instead a normal variation that is not associated with the disease. We plan on developing a test that will determine if changes of the WDR36 gene found in glaucoma patients have a functional consequence. This will allow us to determine if WDR36 causes glaucoma. Understanding the actual function of WDR36 could also
provide insight into a new cellular pathway to which novel glaucoma therapies can be targeted.

2007 American Glaucoma Society Fellowship Grant

JOHN H. FINGERT, M.D., Ph.D.
University of Iowa Hospitals and Clinics

Molecular Genetics of Pigment Dispersion Syndrome

Glaucoma is a leading cause of irreversible blindness in the United States, and glaucoma associated with pigment dispersion syndrome (PDS) is of particular scientific interest for several unique reasons. PDS is a common condition affecting as many as 2.5% of Americans and 10-50% of those with this condition develop high intraocular pressure (IOP) and a form of glaucoma (pigmentary glaucoma). In comparison to other forms of glaucoma, pigmentary glaucoma has an early average age of onset and consequently a relatively high potential cost to society.

PDS has a strong hereditary component. Although no PDS-causing genes have been discovered to date, several lines of evidence suggest that heredity has a significant role in PDS and pigmentary glaucoma. First, a PDS-like condition has been detected in laboratory mice and in dogs that is clearly heritable and is caused by defects in known genes in the case of the laboratory mice. It is likely that similar genetic abnormalities also cause PDS in human patients. Family studies and comparisons of the prevalence of PDS in different ethnic groups have also suggested that this condition is heritable and may be caused by specific genes. These observations suggest that PDS and pigmentary glaucoma are important public health problems that may be best studied with molecular genetic approaches.
## COMPARATIVE FINANCIAL SUMMARY

### ASSETS

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<tr>
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<th>2007</th>
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<tr>
<td><strong>CURRENT ASSETS</strong></td>
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<td>Cash and cash equivalents</td>
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<td>Accounts receivable</td>
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<td>Prepaid expense</td>
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<td><strong>EQUIPMENT, NET</strong></td>
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<tr>
<td><strong>OTHER ASSETS</strong></td>
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<td>Assets restricted for permanent endowments</td>
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<td>Investments - equity securities</td>
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<td>Investments - money market</td>
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<tr>
<td><strong>Total other assets</strong></td>
<td>2,952,410</td>
<td>2,393,487</td>
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<tr>
<td><strong>TOTAL ASSETS</strong></td>
<td>$4,064,540</td>
<td>$3,345,862</td>
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### LIABILITIES AND NET ASSETS

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<th>2007</th>
<th>2006</th>
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<tr>
<td><strong>CURRENT LIABILITIES</strong></td>
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<tr>
<td>Accounts payable and accrued expenses</td>
<td>$ 318,911</td>
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<td>Charitable gift annuity-current portion</td>
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<td><strong>Total current liabilities</strong></td>
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<tr>
<td><strong>LONG-TERM LIABILITIES</strong></td>
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<td>Charitable gift annuity-long term portion</td>
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<td><strong>TOTAL LIABILITIES</strong></td>
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<td><strong>NET ASSETS</strong></td>
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<td>Unrestricted</td>
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<td><strong>Total net assets</strong></td>
<td>3,740,144</td>
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**TOTAL LIABILITIES AND NET ASSETS**

<table>
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<tr>
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<th>2007</th>
<th>2006</th>
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<tbody>
<tr>
<td><strong>TOTAL LIABILITIES AND NET ASSETS</strong></td>
<td>$4,064,540</td>
<td>$3,345,862</td>
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### Comparative Statement of Activities

<table>
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<td>Individual and corporate donations</td>
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<td>$ —</td>
<td>$ 74,894</td>
<td>$1,480,964</td>
<td>$ 947,871</td>
<td>$ —</td>
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<td>Board designated restrictions</td>
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<td>Net assets released from restrictions</td>
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<tr>
<td><strong>Total support, contributions, and other revenue</strong></td>
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<td>558,923</td>
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<tr>
<td>Interest and dividend income</td>
<td>53,219</td>
<td>—</td>
<td>—</td>
<td>53,219</td>
<td>35,087</td>
<td>—</td>
<td>—</td>
<td>35,087</td>
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<tr>
<td>Investment management fees</td>
<td>(47,157)</td>
<td>—</td>
<td>—</td>
<td>(47,157)</td>
<td>(35,261)</td>
<td>—</td>
<td>—</td>
<td>(35,261)</td>
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<tr>
<td>Net unrealized and realized gains on investments</td>
<td>403,387</td>
<td>—</td>
<td>—</td>
<td>403,387</td>
<td>292,473</td>
<td>—</td>
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<tr>
<td><strong>Total investment income</strong></td>
<td>409,449</td>
<td>—</td>
<td>—</td>
<td>409,449</td>
<td>292,299</td>
<td>—</td>
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<td>292,299</td>
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<tr>
<td><strong>Total revenue</strong></td>
<td>2,015,496</td>
<td>—</td>
<td>558,923</td>
<td>2,574,419</td>
<td>1,212,612</td>
<td>—</td>
<td>—</td>
<td>808,336</td>
</tr>
<tr>
<td><strong>Expenses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operating expenses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Program services</td>
<td>1,390,204</td>
<td>—</td>
<td>—</td>
<td>1,390,204</td>
<td>1,175,602</td>
<td>—</td>
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<td>1,175,602</td>
</tr>
<tr>
<td>Management and general</td>
<td>54,849</td>
<td>—</td>
<td>—</td>
<td>54,849</td>
<td>76,900</td>
<td>—</td>
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<td>76,900</td>
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<tr>
<td>Fundraising</td>
<td>219,707</td>
<td>—</td>
<td>—</td>
<td>219,707</td>
<td>258,052</td>
<td>—</td>
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<td>258,052</td>
</tr>
<tr>
<td><strong>Total operating expenses</strong></td>
<td>1,664,760</td>
<td>—</td>
<td>—</td>
<td>1,664,760</td>
<td>1,510,554</td>
<td>—</td>
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<tr>
<td><strong>Fundraising benefit expenses</strong></td>
<td>233,062</td>
<td>—</td>
<td>—</td>
<td>233,062</td>
<td>250,874</td>
<td>—</td>
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<td>250,874</td>
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<tr>
<td><strong>Total expenses</strong></td>
<td>1,897,822</td>
<td>—</td>
<td>558,923</td>
<td>1,761,428</td>
<td>808,336</td>
<td>—</td>
<td>—</td>
<td>1,761,428</td>
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<tr>
<td><strong>Change in net assets</strong></td>
<td>117,674</td>
<td>—</td>
<td>558,923</td>
<td>(548,816)</td>
<td>—</td>
<td>808,336</td>
<td>—</td>
<td>259,520</td>
</tr>
<tr>
<td><strong>Net assets, beginning of year</strong></td>
<td>670,060</td>
<td>—</td>
<td>2,393,487</td>
<td>3,063,547</td>
<td>1,218,876</td>
<td>—</td>
<td>—</td>
<td>1,585,151</td>
</tr>
<tr>
<td><strong>Net assets, end of year</strong></td>
<td>$ 787,734</td>
<td>$ —</td>
<td>$ 2,952,410</td>
<td>$ 3,740,144</td>
<td>$ 670,060</td>
<td>$ —</td>
<td>—</td>
<td>$ 2,393,487</td>
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