The Glaucoma Foundation

2015 Annual Report
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Message from the President

Dear Friends:

2015 was a challenging year for everyone, and The Glaucoma Foundation was certainly not immune to the meaningful economic uncertainties we were all presented. However, as the year ended, we were able to cite meaningful and measurable accomplishments 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 2015, we hosted an award-worthy 22\textsuperscript{nd} Annual International Think Tank in New York City. Fifty-one participants from around the world gathered to address: “Regenerative Medicine for Cornea and Trabecular Meshwork”. Enormous positive progress was demonstrated throughout the session, with the hope being that the same exciting report will be forthcoming from the 23\textsuperscript{rd} Annual Think Tank that will be held in June, 2016 once more in New York City.

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
President
Chief Executive Officer
Board of Directors

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New York, NY

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Chairman Emeritus
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Secretary & Founder
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Albert Einstein School of Medicine
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J.M. Cohen & Company
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Vistakon
Jacksonville, FL

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St. Albans VA Medical Center
Hewlett, NY

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New York, NY

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Huntington, NY

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Madison, WI

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Martin R. Lewis Associates
New York, NY

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Columbia University Medical Center
New York, NY

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New York, NY

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Boston, MA

Kumar Mahadeva
Greenwich, CT

Kenneth Mortenson
New York, NY

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Boca Raton, FL

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Pueblo, CO

Irving Wolbrom
New York, NY

Alcon Laboratories, Inc
Gary Menichini
Fort Worth, TX
Scientific Advisory Board

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Chairman
Shelley and Steven Einhorn
Distinguished Chair, Professor of Ophthalmology
Chief, Glaucoma Services
Surgeon Director
New York Eye & Ear Infirmary

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Director, Glaucoma Service
Duke University

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Carver College of Medicine, University of Iowa

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Washington University School of Medicine

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Devers Eye Institute & Research Laboratories

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Vice Chair for Research
Director of Glaucoma Service
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Carver College of Medicine, University of Iowa

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Byers Eye Institute at Stanford University

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Chief of Glaucoma
University of Toronto, Canada
Director, Glaucoma Unit
Li Ka Shing Knowledge Institute
St. Michael’s Hospital, Canada

Simon John, PhD
Principal Investigator
Howard Hughes Medical Institute
The Jackson Laboratory

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Department Chair Emeritus
Department of Ophthalmology & Visual Sciences
School of Medicine & Public Health
University of Wisconsin-Madison

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Department of Pharmaceutical Sciences
University of Colorado Denver

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SVM Professor of Nanomedicine
Professor of Basic Medical Sciences and Biomedical Engineering
Purdue University

Jeffrey M. Liebmann, MD
Shirlee and Bernard Brown Professor of Ophthalmology
Vice Chair, Department of Ophthalmology
Director, Glaucoma Service
Harkness Eye Institute
Columbia University Medical Center

Carlo D. Montemagno, PhD
CRC Chair in Intelligent Nanosystems
Director, Ingenuity Lab
Director, Biomaterials Program
NRC-CNRC Nat. Inst Nanotechnology
Professor of Chemical and Materials Engineering
University of Alberta

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University of Wisconsin-Madison

Colm O’Brien, FRCS, MD
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Mater Misericordiae University Hospital, Ireland

Louis Pasquale, MD, FARVO
Professor of Ophthalmology
Harvard Medical School
Director, Glaucoma Service
Massachusetts Eye & Ear Infirmary

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Faculty of Medicine
Department of Anatomy and Cell Biology
McGill University

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Professor of Epidemiology
W.K. Kellogg Eye Center
University of Michigan
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Professor of Human Molecular Genetics
Director of Molecular Ophthalmic Genetics Laboratory
Divisions of Ophthalmology & Surgical Research
Department of Surgery
University of Connecticut Health Center

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Harkness Eye Institute
Columbia University Medical Center

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Distinguished Professor of Ophthalmology
Chairman, Department of Ophthalmology
Director, Shiley Eye Center
Director, Hamilton Glaucoma Center
Morris Gleich Chair
University of California San Diego

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President
Wayne State University

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Investigator and Professor
The Stowers Institute for Medical Research
Department of Anatomy and Cell Biology
University of Kansas Medical Center

Michael Joseph Young, PhD
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Schepens Eye Research Institute
Associate Professor
Department of Ophthalmology
Harvard Medical School

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Vice President, Glaucoma Development, R&D (Acting)
Alcon Research, Ltd.

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University of Erlangen-Nurnberg, Germany

Joel S. Schuman, MD, FACS
Professor and Chairman of Ophthalmology
NYU Langone Medical Center
New York University School of Medicine

Barbara Wirostko, MD
Clinical Adjunct Associate Professor
University of Utah
Moran Eye Center

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New York University School of Medicine

Barbara Wirostko, MD
Clinical Adjunct Associate Professor
University of Utah
Moran Eye Center
In the frontal anterior part of the eye there is constant formation of a nutrient fluid. The fluid then moves out through the eye providing nutrition to the different intraocular cells and then exits the eye through a filtering mesh-like area. Sometimes the mesh gets blocked and the fluid cannot exit. This causes increased pressure inside the eye. This extra pressure chokes the blood capillaries that feed the retina, as a result retinal cells start to die, which leads to blindness. This disease is called glaucoma. In exfoliation syndrome (XFS), frontal interior parts of the eye start accumulating white fluff deposits composed of protein aggregates. The aggregates eventually float away with the fluid flow and block the fluid exit, causing glaucoma. We have discovered that cells obtained from these eyes may have a problem degrading these protein aggregates inside the cell leading to a buildup of "cellular trash" that becomes toxic to the cells. In this proposal we will test methods to improve the degradation of this cellular waste to improve the health of the XFS cells.

Validation of Pseudoexfoliation Glaucoma Biomarkers by MRM

Glucoma is a leading cause of blindness worldwide that encompasses a complex group of disorders that are multigenic and multifactorial in origin. Primary open angle glaucoma (POAG) and pseudoexfoliation glaucoma (PEXG) are among the most prevalent forms of glaucoma in developed countries. It is clinically observed that by the time a typical glaucoma patient is diagnosed, it has already lost 35-40% of their retinal ganglion cells. Therefore, a much more sensitive and specific method of early detection is crucial to improve treatment of glaucoma. The study of discovery and validation of diagnostic markers in blood serum for the early detection of glaucoma presents more advantages than accessing to surgically invasive intraocular biofluids or tissues. We propose a novel technology that is based on the simultaneously quantitative determination of many biomarkers in many blood samples. This approach will permit a rapid and accurate assay to determine significant differences of a small parcel of markers among PEXG and POAG patients, that will be able to diagnose, classify, predict therapeutic responses and monitor their treatment. We expect from this work to provide insight on both the pathogenesis of GPXE and the overall systemic involvement in the disease.
Determining the Genetic Basis of Exfoliation Syndrome in a Large Pedigree Using Next-Generation Sequencing

Determining the genetic basis of exfoliation syndrome in the general population has proven challenging similar to other complex, age-related diseases. This study aims to determine the genetic basis of exfoliation syndrome in a large family with a strong pattern of inheritance. Affected family members genomes likely contain a causal or high risk disease variant(s). Genetic signatures will be obtained by sequencing the DNA of affected and unaffected individuals. Analyzing these signatures will produce a small list of gene variants and chromosomal regions that segregate with affected individuals. Knowledge of how exfoliation syndrome is inherited can help with diagnoses and may provide novel approaches to treatment.

Unlocking the Hereditable Basis of Exfoliation Syndrome

When we recently contrasted the genetic profiles of 8,000 exfoliation syndrome (XFS) cases and 20,000 controls from 17 countries, we noted a very strong relationship between individuals carrying an alternate form of the CACNA 1A gene and increased predisposition to XFS. This is only the second gene discovered for XFS in the last 7 years, with the other being LOXL1. Until today, investigators were unable to localize the specific genetic variants responsible for XFS due to limited patient collections. We are now in a strong position to address this after building an international XFS genetic consortium across 6 continents (17 countries). This study is part of this global effort to fully understand the hereditable basis of XFS by performing systematic genome-scans on multi-ethnic XFS patient collections. We anticipate this global effort to be able to definitively identify and implicate a robust network of XFS genes which will provide dramatic insights into the disease process of XFS and inform drug discovery.
Pseudoexfoliation syndrome (XFS) is a unique form of ocular condition characterized by accumulation of white dandruff like deposits in various ocular structures like conjunctiva, lens, iris, cornea and ciliary body. It is also known to be deposited in other visceral organs like liver and meninges and is thought to be a systemic disorder with increased incidence of cerebrovascular events, myocardial infarction and hypertension. In the eye, this disease causes laxity of the zonules leading to spontaneous lens subluxation, poor pupillary dilatation, retinal vein occlusions and glaucoma, which forms the leading cause of blindness due to this disease. The pathogenesis of this disease still evades a simple answer since the origin of the deposits remains unclear. Diagnosis of this disease still depends on clinical examination of the unique deposits in the anterior segment, though the other “normal” eye of clinically unilateral cases has been proven to have similar deposits on electron microscopy in several studies. There is no current system for identifying it in the earlier stage before the onset of glaucoma. We have recently characterized the clinical features that may be seen in the earlier stages and those that may be seen at later stages of the disease process. It is still unclear how this disease transits from the unmanifest stage to the clinically manifest stage or the onset of glaucoma. Tear evaluation for markers is known to predict severity of a disease or even presence of a particular condition in the eye. Study of the tears would therefore be a useful non-invasive and easy tool for identifying the earlier stages of the disease as well as predicting progression.

Mechanisms in Exfoliation Glaucoma: Effect of Genetic Risk Variants and Ocular Cell Stressors on LOXL1 Expression

It is known that genetic variants in the gene LOXL1 increase risk for developing a particularly severe form of glaucoma called exfoliation glaucoma. In addition, environmental factors such as UV exposure can influence disease risk in genetically susceptible individuals. However, the precise mechanism for how these genetic and environmental factors contribute to disease development is poorly understood. We are working to better understand how an individual's genetic makeup and environment interact to cause exfoliation glaucoma. With this new knowledge, it will be possible to develop novel therapies to treat and possibly even cure this blinding disease.
DEBORAH WALLACE, PhD
University College, Dublin

To Investigate the Role of Methylation in the Regulation of Lysyl Oxidase Like 1 Expression in Pseudoexfoliation Glaucoma

Genetic studies have identified a gene called lysyl oxidase like 1 (LOXL1) which is thought to be important for an individual's predisposition to developing pseudoexfoliation syndrome. Other studies have shown that levels of LOXL1 can vary between normal and disease patients and also in disease progression. While LOXL1 is of importance other factors also play a role in determining if an individual develops glaucoma, for example levels of oxidative stress and hypoxia (lack of oxygen) in the eye. In this study we wish to address the question of how levels of LOXL1 are altered as glaucoma develops and progresses. We will examine a mechanism of controlling LOXL1 expression called epigenetics. This method of regulation the expression of a gene can be induced in cells exposed to hypoxic environment in glaucoma. We will investigate levels of LOXL1 in cells from donors with and without glaucoma, and in cells from normal donors subjected to hypoxia and investigate the role of epigenetics in controlling LOXL1 expression.
## COMPARATIVE SUMMARY OF ASSETS, LIABILITIES & NET ASSETS

### ASSETS

#### CURRENT ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash and cash equivalents</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available for operations</td>
<td>$811,906</td>
<td>$972,514</td>
</tr>
<tr>
<td>Research reserve - board designated</td>
<td>329,889</td>
<td>329,710</td>
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<tr>
<td>Total cash and cash equivalents</td>
<td>1,141,795</td>
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<td>Contributions receivable</td>
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<td>Prepaid expense</td>
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<td>Total current assets</td>
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#### EQUIPMENT, NET

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<tr>
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<th>2014</th>
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</thead>
<tbody>
<tr>
<td>Total equipment, net</td>
<td>3,883</td>
<td>3,341</td>
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#### OTHER ASSETS

<table>
<thead>
<tr>
<th>Description</th>
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<th>2014</th>
</tr>
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<tbody>
<tr>
<td>Assets permanently restricted for endowments</td>
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<td></td>
</tr>
<tr>
<td>Cash</td>
<td>573</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>368,128</td>
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<tr>
<td></td>
<td>4,685,436</td>
<td>5,045,476</td>
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<tr>
<td>Security deposit</td>
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<td>27,796</td>
</tr>
<tr>
<td>Total other assets</td>
<td>4,713,232</td>
<td>5,073,272</td>
</tr>
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</table>

**TOTAL ASSETS**

<table>
<thead>
<tr>
<th></th>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOTAL ASSETS</td>
<td>$5,930,169</td>
<td>$6,454,873</td>
</tr>
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</table>
## LIABILITIES AND NET ASSETS

### CURRENT LIABILITIES

<table>
<thead>
<tr>
<th>Description</th>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accounts payable and accrued expenses</td>
<td>$51,024</td>
<td>$189,963</td>
</tr>
<tr>
<td>Grants payable</td>
<td>50,574</td>
<td>110,000</td>
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<tr>
<td><strong>Total current liabilities</strong></td>
<td><strong>101,598</strong></td>
<td><strong>299,963</strong></td>
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</tbody>
</table>

### TOTAL LIABILITIES

<table>
<thead>
<tr>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>101,598</td>
<td>299,963</td>
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</tbody>
</table>

### NET ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unrestricted</td>
<td></td>
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</tr>
<tr>
<td>Undesignated</td>
<td>813,246</td>
<td>739,266</td>
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<tr>
<td>Board designated</td>
<td>329,889</td>
<td>329,710</td>
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<tr>
<td><strong>Total unrestricted</strong></td>
<td>1,143,135</td>
<td>1,068,976</td>
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<tr>
<td>Temporarily restricted</td>
<td>—</td>
<td>40,458</td>
</tr>
<tr>
<td>Permanently restricted</td>
<td>4,685,436</td>
<td>5,045,476</td>
</tr>
<tr>
<td><strong>Total net assets</strong></td>
<td>5,828,571</td>
<td>6,154,910</td>
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### TOTAL LIABILITIES AND NET ASSETS

<table>
<thead>
<tr>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>$5,930,169</td>
<td>$6,454,873</td>
</tr>
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## COMPARATIVE STATEMENT OF ACTIVITIES

<table>
<thead>
<tr>
<th></th>
<th>2015</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unrestricted</td>
<td>Temporarily Restricted</td>
</tr>
<tr>
<td>Revenue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Support, contributions, and other revenue</td>
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<td></td>
</tr>
<tr>
<td>Individual and corporate donations</td>
<td>$1,327,956</td>
<td>$ —</td>
</tr>
<tr>
<td>Fundraising benefit</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Board designated restrictions</td>
<td>420,040</td>
<td>—</td>
</tr>
<tr>
<td>Net assets released from restrictions</td>
<td>40,458</td>
<td>(40,458)</td>
</tr>
<tr>
<td>Total support, contributions, and other revenue</td>
<td>1,788,454</td>
<td>(40,458)</td>
</tr>
<tr>
<td>Investment income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interest and dividend income</td>
<td>94,456</td>
<td>—</td>
</tr>
<tr>
<td>Investment management fees</td>
<td>(84,152)</td>
<td>—</td>
</tr>
<tr>
<td>Net unrealized and realized gains (losses) on investments</td>
<td>(429,956)</td>
<td>—</td>
</tr>
<tr>
<td>Total investment income</td>
<td>(419,652)</td>
<td>—</td>
</tr>
<tr>
<td>Total revenue</td>
<td>1,368,802</td>
<td>(40,458)</td>
</tr>
<tr>
<td>Expenses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operating expenses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Program services</td>
<td>1,118,278</td>
<td>—</td>
</tr>
<tr>
<td>Management and general</td>
<td>48,514</td>
<td>—</td>
</tr>
<tr>
<td>Fundraising</td>
<td>127,851</td>
<td>—</td>
</tr>
<tr>
<td>Total operating expenses</td>
<td>1,294,643</td>
<td>—</td>
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<tr>
<td>Fundraising benefit expenses</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total expenses</td>
<td>1,294,643</td>
<td>—</td>
</tr>
<tr>
<td>Change in net assets</td>
<td>74,159</td>
<td>(40,458)</td>
</tr>
<tr>
<td>Net assets, beginning of year</td>
<td>1,068,976</td>
<td>40,458</td>
</tr>
<tr>
<td>Net assets, end of year</td>
<td>$1,143,135</td>
<td>$ —</td>
</tr>
</tbody>
</table>