# Table of Contents

Message From The President...................... 3  
Board of Directors.................................. 4  
Scientific Advisory Board......................... 5  
2014 Research Grants............................... 7  
Financial Summary.................................. 11
Message from the President

Dear Friends:

2014 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 2014, we hosted an award-worthy 21st Annual International Think Tank in New York City. Forty-nine participants from around the world gathered to address: “Exfoliation Syndrome: Tying It All Together”. Enormous positive progress was demonstrated throughout the session, with the hope being that the same exciting report will be forthcoming from the twenty-second Annual Think Tank that will be held in September of 2015 once more in New York City.

Thanks to your generosity and commitment to us, revenue flows remained strong in most categories of gifts. The Glaucoma Foundation Ball honored world-renowned physician, Dr. Bruce E. Spivey, attracted 250 guests and raised nearly $300,000 in revenue. Expenses are analyzed continually for their value to the organization and are deemed by the Board to be well under control.

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

Scott R. Christensen
President
Chief Executive Office

Board of Directors

Gregory K. Harmon, MD
Chairman
New York, NY

Robert Ritch, MD
Medical Director, Vice President,
Secretary & Founder
Shelley and Steven Einhorn Distinguished Chair;
Professor of Ophthalmology; Chief, Glaucoma
Services; and Surgeon Director
The New York Eye & Ear Infirmary, New York, NY

Joseph M. La Motta
Chairman Emeritus
Pound Ridge, NY

William C. Baker
New York, NY

Ilene Giaquinta
New York, NY

Kumar Mahadeva
Greenwich, CT

Salvatore P. Ciampo
Albert Einstein School of Medicine
Bronx, NY

Barbara W. Hearst
Charleston, SC

Kenneth Mortenson
New York, NY

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

Gerald Kaiser, Esq.
Old Westbury, NY

John H. Roberts
Crowley Chemical Company, Inc.
New York, NY

Lori G. Feldman, Esq.
Milberg LLP
New York, NY

Paul Kaufman, MD
University of Wisconsin-Madison
Madison, WI

Sheldon M. Siegel
Boca Raton, FL

David Fellows
Vistakon
Jacksonville, FL

Theodore Krupin, MD
Northwestern Medical School
Chicago, IL

Mary Jane Voelker
Pueblo, CO

Murray Fingeret, OD
St. Albans VA Medical Center
Hewlett, NY

Irving Wolbrom
New York, NY

Barry Friedberg
FriedbergMilstein, LLC
New York, NY

Alcon Laboratories, Inc
Gary Menichini
Fort Worth, TX

Maurice H. Luntz, MD
New York, NY

Allergan, Inc
Julian Gangolli
Irvine, CA
Scientific Advisory Board

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

R. Rand Allingham, MD
Richard and Kit Barkhouser
Professor of Ophthalmology
Director, Glaucoma Service
Duke University

Terete Borrás, PhD
Professor of Ophthalmology
University of North Carolina

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

John Danias
Professor of Ophthalmology and Cell Biology
Co-Director of Glaucoma Service
SUNY Downstate Medical Center

John H. Fingert, MD, PhD
Associate Professor
Department of Ophthalmology & Visual Sciences
Carver College of Medicine, University of Iowa

Jeffrey L. Goldberg, MD, PhD
Director of Research Professor
Shiley Eye Center

Neeru Gupta, MD, PhD
Professor and Dorothy Pitts Chair of Ophthalmology & Vision Science
Laboratory Medicine & Pathobiology
University of Toronto, Canada
Director, Glaucoma & Nerve Protection Unit
Keen Research Centre at the Li Ka Shing Knowledge institute
St. Michael’s Hospital, Canada

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

Paul L. Kaufman, MD
Peter A. Duehr Professor and Chair
Department of Ophthalmology & Visual Sciences
University of Wisconsin-Madison

Uday B. Kompella, PhD
Professor
Department of Pharmaceutical Sciences
University of Colorado Denver

Theodore Krupin, MD
Professor of Ophthalmology
Northwestern University Medical School

James F. Leary, PhD
SVM Professor of Nanomedicine
Professor of Basic Medical Sciences and Biomedical Engineering
Purdue University

Jeffrey M. Liebmann, MD
Clinical Professor of Ophthalmology
New York University Medical Center
Director, Glaucoma Services
Manhattan Eye, Ear & Throat Hospital

Carlo D. Montemagno, PhD
Scientific Director
National Institute of Nanotechnology
University of Alberta, Canada

Robert Nickells, MD
Professor and Vice Chair for Research
Department of Ophthalmology & Visual Science
University of Wisconsin-Madison

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

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

Julia E. Richards, PhD
Harold F. Falls Professor of Ophthalmology & Visual Sciences
Professor of Epidemiology
W.K. Kellogg Eye Center
University of Michigan

Mansoor Sarfarazi, PhD
Professor of Human Molecular Genetics
Director of Molecular Ophthalmic Genetics Lab
University of Connecticut Health Center

Ursula Schütz-Schreiber, PhD
Professor
Department of Ophthalmology
University of Erlangen-Nuremberg, Germany

Gülgün Tezel, MD
Professor of Ophthalmology & Visual Sciences & Department of Anatomical Sciences & Neurobiology
University of Louisville School of Medicine

Michael A. Walter, PhD
Professor and Chair
Department of Medical Genetics
University of Alberta, Canada

Robert N. Weinreb, MD
Distinguished Professor of Ophthalmology
Director, Shiley Eye Center
Director, Hamilton Glaucoma Center
University of California-San Diego

M. Roy Wilson, MD, MS
Deputy Director Strategic Scientific Planning and Program Coordination
National Institute on Minority Health and Health Disparities
National Institutes of Health

Barbara Wirostko, MD
Clinical Adjunct Associate Professor
University of Utah

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

Michael J. Brubaker, PhD  
Vice President, Glaucoma Development, R&D (Acting)  
*Alcon Research, Ltd.*

Larry A. Wheeler, PhD  
Senior Vice President  
*Biological Sciences*  
*Allergan, Inc.*
2014 RESEARCH GRANTS

MICHAEL G. ANDERSON, PhD
The University of Iowa

Development of mouse models for dissecting LOXL1-mediated pathology

Exfoliation syndrome is a disease affecting approximately 80 million people worldwide and is a significant cause of the potentially blinding eye disease, glaucoma. Recent genetic research has identified a gene, named LOXL, that is clearly associated with risk for exfoliation syndrome. In order to better understand the role of this gene, we are creating a new mouse model in which the human LOXL1 gene will be added into the genome of mice. With these mice, we will test the role of LOXL1 in the eye and simultaneously create a lasting resource empowering many possible future experiments for better understanding, and ultimately curing, exfoliation syndrome.

TERETE BORRÁS, PhD
University of North Carolina, Chapel Hill

Targeting wild-type and N-terminus-deleted LOXL1 to the iris pigment epithelium (IPE) in living rats. Effect on the formation of the IPE elastin network on IOP.

Glaucoma is a disease of the eye caused by the death of some cells of the retina called Retinal Ganglion Cells (RGCs). These cells are on the back of the eye and have a prolongation called axon that leaves the eye and carries the visual signal to the brain. All the axons together form a bundle which is called the optic nerve. Damage to these cells and their optic nerve can be provoked by many different “insults”, and thus the many types of glaucoma. One of the major insults to the RGC and their axons/optic nerve is elevated intraocular pressure (IOP). In pseudoexfoliation glaucoma (PEX), cells appear to secrete a “dandruff”-like material that is sticky, and get deposited in the front part inside the eye. Needless to say that this material will block the normal drainage of eye fluid and will induce elevated IOP. It is not known how the PEX material is formed, but it is known that involves elastic fibers, a sort of support ligament on the outside of the cells. Recent discoveries have found a gene which appears to be changed in patients with PEX. Curiously, one of the functions of the gene is to help forming what one will call the scaffold on the elastin network. The gene is called Lysyl Oxidase-like 1, or LOXL1 for short. In order to understand if this gene is directly responsible for all the effects that clinicians see in PEX, in this project we propose to deliver different amounts of this normal and mutated protein to cells suspected of producing the material. We will engineer gene transfer viral vectors in such a way that they will deliver their LOXL1 cargo to the cells we want. We will do this in living rats, and then monitor the IOP and look at their tissues for the formation of the “dandruff”. If successful, this project will give us a little more understanding on how PEX occurs, which if the first step towards finding a cure for exfoliation glaucoma.
JOHN FINGERT, MD, PhD
The University of Iowa

**Stem-cell based studies of retinal ganglion cells, the TBK1 gene and normal tension glaucoma**

Retinal ganglion cells are the part of the eye that are primarily affected by glaucoma, however, these cells are very difficult to study in the laboratory. Consequently, we have used a stem cell-based approach to generate retinal ganglion cells in culture. We will use these cells to study the mechanism by which a known glaucoma gene, TANK binding kinase 1 (TBK1) leads to retinal ganglion cell loss and eye disease. These studies will provide new insights in the biology of glaucoma and will facilitate the development of new glaucoma therapies.

HECTÓR GONZÁLEZ-IGLESIAS
Instituto Oftalmológico Fernández-Vega

**Validation of pseudoexfoliation glaucoma biomarkers by MRM**

Glaucoma 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.

MARGARET E. HUFLEJT, PhD
New York University School of Medicine

**Anti-glycan antibody immunoprofiles as biomarkers in early detection of exfoliation syndrome**

Glaucoma is the second largest cause of permanent blindness worldwide, affecting over 60 million people. Exfoliation syndrome (XFS) is the most common recognizable cause of open-angle glaucoma and it is estimated that there are about 60 million people with this condition worldwide, about 20% of whom develop glaucoma. This glaucoma usually affects one eye,
progresses more rapidly than primary open-angle glaucoma, responds less well to medical treatment, and requires surgery more often.

The field of chemistry now offers a novel, high-throughput nano-technology called printed glycanarray (PGA) that can identify patterns of glycan-binding proteins, including anti-glycan autoantibodies. Glycan arrays contain large variety of synthetic carbohydrate compounds that are present in nature on surfaces of various human cells and on human pathogens. Various physiological fluids such as e.g. serum can be screened using PGA for the presence of anti-glycan antibodies to define certain types of carbohydrates that are targeted by the human immune system. These anti-glycan antibodies form “immunoprofiles” specific for individual people and these individual immunoprofiles show specific variations during the development of pathological conditions.

We will study the presence of these antibodies in sera and in ocular fluids of “control” patients without XFS, and in patients with XFS. A goal of this project is to identify a specific pattern of anti-glycan autoantibodies characteristic for XFS, which will define an immuno-signature of XFS. The identification of specific antibody patterns in XFS would help in early diagnosis and better understanding of the genetic, molecular and cellular mechanisms underlying the nature of exfoliation material. This information will help to improve the outcomes for exfoliation-syndrome glaucoma though early diagnosis and better management.

YUTAO LIU, MD, PhD
Duke University

Roles of regulatory variants in LOXL1 in pseudoexfoliation glaucoma, Year 2

Sequence variants in the LOXL1 gene have been associated with the increased risk of exfoliation glaucoma (XFS) across many different populations including Caucasian, Chinese, Japanese, Indians and Africans. However, the functional causal variants still remain to be identified. The purpose of this proposal, a renewal of our original grant, is to study how genetic variants of the LOXL1 gene cause XFG. These highly associated genetic variants are located within the promoter, a critical region of another gene, LOXL1-AS1, which could potentially regulate the production of LOXL1. We have determined that this associated region does regulate the expression of LOXL1-AS1 as a promoter. We propose to determine how these highly associated variants cause changes in the function of both the regulatory gene LOXL1-AS1 and LOXL1 itself. This study is designed to identify the key steps that lead to XFG. This information will provide extremely useful information that can be utilized to create therapeutics to control or cure XFG, one of the most common known causes of global blindness.

MANSOOR SARFARAZI, PhD
University of Connecticut Health Center

Exome analyses of families with pseudoexfoliation glaucoma
Pseudoexfoliation syndrome (PEX) is a systemic condition with or without manifestation of glaucoma in the affected subjects. This is a very late-onset ocular condition and, therefore, large families with multiply affected subjects are extremely rare. Over the last 15 years, we used multiply affected PEX families and classical genetic linkage analyses to map few provisional loci for this condition. However, as of this writing, the identity of the defective gene still remains elusive. Last year, we used the same multiply affected PEX families and scanned the entire genome by whole-genome exome sequencing. This is a very rapid and powerful method that is especially fruitful once it is applied to families with multiply affected subjects. Although the massive sequencing data generated by this method are still being fully scrutinized, our preliminary analyses indicate that several interesting genes are most likely to be involved in the etiology of this phenotype. Additional studies are in progress to validate this initial observation. Once proven, this will provide a rapid and accurate method for genetic ascertainment in siblings of known affected or other suspected sporadic cases in a given population. This, together with prior association studies of LOXL1 (and other gene polymorphisms) will allow a rapid molecular diagnosis for a large proportion of PEX subjects.

MANSOOR SARFARAZI, PhD  
University of Connecticut Health Center

Next generation exome sequencing in families with normal tension glaucoma

Normal tension glaucoma (NTG) is one of the most perplexing forms of primary open-angle glaucoma (POAG) to diagnose, since it is consistently presented with intraocular pressure (IOP) within normal range. As the two previous NTG-causing genes of OPTN and TBK1 account for only a limited number of cases, identification of additional defective genes is still urgently needed. In this application, we are proposing to accomplish this by utilizing a group of affected subjects from our NTG families and scan them through the entire genome using the next-generation exome sequencing technology. This approach will lead to the advancement of accurate molecular genetics diagnosis for NTG cases in the U.S. and other populations worldwide.
## COMPARATIVE FINANCIAL SUMMARY

### ASSETS

#### CURRENT ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>2014</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash and cash equivalents</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available for operations</td>
<td>$972,514</td>
<td>$665,011</td>
</tr>
<tr>
<td>Research reserve - board designated</td>
<td>329,710</td>
<td></td>
</tr>
<tr>
<td>Total cash and cash equivalents</td>
<td>1,302,224</td>
<td>$665,011</td>
</tr>
<tr>
<td>Contributions receivable</td>
<td>67,021</td>
<td>119,894</td>
</tr>
<tr>
<td>Prepaid expense</td>
<td>9,015</td>
<td>10,992</td>
</tr>
<tr>
<td>Total current assets</td>
<td>1,378,260</td>
<td>795,897</td>
</tr>
</tbody>
</table>

#### EQUIPMENT, NET

<table>
<thead>
<tr>
<th>Description</th>
<th>2014</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total current assets</td>
<td>1,378,260</td>
<td>795,897</td>
</tr>
<tr>
<td>EQUIPMENT, NET</td>
<td>3,341</td>
<td>2,186</td>
</tr>
</tbody>
</table>

#### OTHER ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>2014</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assets permanently restricted for endowments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Investments - equity securities</td>
<td>4,677,348</td>
<td>4,777,827</td>
</tr>
<tr>
<td>Investments - money market</td>
<td>368,128</td>
<td>77,249</td>
</tr>
<tr>
<td>Total other assets</td>
<td>5,045,476</td>
<td>4,855,076</td>
</tr>
<tr>
<td>Security deposit</td>
<td>27,796</td>
<td>27,796</td>
</tr>
<tr>
<td>Total other assets</td>
<td>5,073,272</td>
<td>4,882,872</td>
</tr>
</tbody>
</table>

#### TOTAL ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>2014</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOTAL ASSETS</td>
<td>$6,454,873</td>
<td>$5,680,955</td>
</tr>
<tr>
<td></td>
<td>2014</td>
<td>2013</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td><strong>CURRENT LIABILITIES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accounts payable and accrued expenses</td>
<td>$189,963</td>
<td>$143,300</td>
</tr>
<tr>
<td>Grants payable</td>
<td>110,000</td>
<td>3,333</td>
</tr>
<tr>
<td>Charitable gift annuity-current portion</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total current liabilities</td>
<td>299,963</td>
<td>146,633</td>
</tr>
<tr>
<td><strong>LONG-TERM LIABILITIES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Charitable gift annuity-long term portion</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><strong>TOTAL LIABILITIES</strong></td>
<td>299,963</td>
<td>146,633</td>
</tr>
<tr>
<td><strong>NET ASSETS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unrestricted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undesignated</td>
<td>739,266</td>
<td>679,246</td>
</tr>
<tr>
<td>Board designated</td>
<td>329,710</td>
<td>—</td>
</tr>
<tr>
<td>Total unrestricted</td>
<td>1,068,976</td>
<td>679,246</td>
</tr>
<tr>
<td>Temporarily restricted</td>
<td>40,458</td>
<td>—</td>
</tr>
<tr>
<td>Permanently restricted</td>
<td>5,045,476</td>
<td>4,855,076</td>
</tr>
<tr>
<td>Total net assets</td>
<td>6,154,910</td>
<td>5,534,322</td>
</tr>
<tr>
<td><strong>TOTAL LIABILITIES AND NET ASSETS</strong></td>
<td>$6,454,873</td>
<td>$5,680,955</td>
</tr>
</tbody>
</table>