



RETINA
RESEARCH
FOUNDATION

ANNUAL
REPORT
2024

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Established in 1969

The mission of the Retina Research Foundation is to reduce retinal blindness worldwide by funding programs in research and education.

Cover image courtesy of UW - Madison, Saha Laboratory
Human model of Leber Congenital Amaurosis
treated with CRISPR nanoparticles

President's Message

Dear Friends,

Retina Research Foundation (RRF) has a singular purpose, simply put, to prevent blindness by bringing curative therapies to those whose sight is threatened by retinal diseases. RRF funds original research that leads to future vision-saving discoveries. Excitingly, we are in a period of accelerated progress as the research tools available to scientists are increasingly sophisticated and AI-assisted, enabling discovery at unprecedented rates and degrees of specificity. On the pages that follow, we share our Foundation's activities throughout 2024. Excellent work is being accomplished, as RRF affiliated researchers make headway in our understanding of complex retinal diseases such as degenerative diseases, inherited genetic diseases, and diabetes and human systemic disease related eye disorders, to name a few.

RRF's research and educational program is multi-faceted, with particular focus on basic research by early-career investigators. One of the objectives of the RRF's basic research grant program is to function as a funding bridge for novel and innovative vision research concepts, too early in their development to attract other major, significantly larger funding sources. Additionally, RRF supports clinical research by established and nationally renowned physician scientists to further pursue their interests, funds academic-based research and clinical care through research chairs and professorships, and provides opportunity for education of international ophthalmology fellows. Through research initiatives, we also provide support for educational materials for ophthalmologists at all stages of the profession, and fund travel scholarships to important vision conferences for young scientists whose research will make a difference for future generations.

Dr. Alice McPherson believed in the power of research, she understood that research advancements are fundamental to providing the most innovative clinical care to patients. Today, vision disorders and blindness remain widespread, particularly among people in their older years, and most of these hard-to-treat conditions are caused by retinal disease. Her firmly held belief led to the founding of our organization, and with the support of donors like you, the RRF research program has grown from the first \$30,000 pilot study grant to fund over \$44.6 million in retina research as of the end of December, 2024. Subsequently, total research funding has increased to over \$47 million.

RRF dedicates over 90% of its resources to research programs aimed to improve retina care. Our ambitious goals rest upon the power of research, essential to the discovery of the therapeutic tools physicians need to treat their patients. This requires sustained and committed effort, and we are indebted to you for being a vital partner in this pursuit. We are appreciative of your continuing interest and support, your belief in our mission propels every new discovery we pursue. RRF believes retina research will provide the answers we seek because just as our founder said, "The only hope of reducing blindness is through research."

Sincerely,



Arthur W. Willis, Jr., MD
President



Research Program Overview - 2024

Retina Research Foundation supports an exemplary variety of programs in retina research all around the world. The following is a brief overview of RRF research supported in 2024, which illustrates the wide scope of the Foundation's activities.

RRF Pilot Study Grants – Investigation of New Research Topics

Baylor College of Medicine, Houston, TX

Samuel Wu, PhD – Kayser Research Project

Rui Chen, PhD – Manning Research Project (relocated to UC-Irvine)

Yingbin Fu, PhD – Dana and Gil Petri Research Project

Richard Hurwitz, MD – Wilson Research Project

Graeme Mardon, PhD – Miller Research Project

Texas A&M Health Science Center, Bryan, TX

Lih Kuo, PhD – Gueymard Research Grant

University of Texas Medical Branch-Galveston, Galveston, TX

Wenbo Zhang, PhD – Bovay Research Project

University of Wisconsin, Madison, WI

Curtis Brandt, PhD – Murfee Macular Degeneration Project

University of Toronto, Toronto, Ontario, Canada

Timothy Corson, PhD – Lawrence Research Project

West Virginia University School of Medicine, Morgantown, WV

Jianhai Du, PhD – Basic Research Project

University of Tennessee, Memphis, TN

Francesco Giorgianni, PhD – Basic Research Project

Vanderbilt University, Nashville, TN

Milam Brantley, MD, PhD – Basic Research Project

Northeastern University, Boston, MA

James Monaghan, PhD – Basic Research Project

Institute for Vision Research Center, University of Iowa, Iowa City, IA

Seongjin Seo, PhD – Basic Research Project

University of California, Irving, Irving, CA

Vladimir Kefalov, PhD – Basic Research Project

University of Illinois at Chicago, Chicago, IL

Adrius Kazlauskas, PhD – Basic Research Project

University of Arizona, Tucson, AZ

Erika D. Eggers, PhD – Basic Research Project

University of Kentucky, Lexington, KY

Ann C. Morris, PhD – Basic Research Project

Augusta University, Medical College of Georgia, Augusta, GA

Ming Zhang, MD, PhD – Basic Research Project

Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, MA

Kinga Bujakowski, PhD – Basic Research Project

University of Texas at Austin, Austin, TX

Jeffrey M. Gross, PhD – Basic Research Project

University of California, San Francisco (UCSF), San Francisco, CA

Alex J. Smith, PhD – Basic Research Project

University of Wisconsin-Madison, Madison, WI

Christine M. Sorenson, PhD – Basic Research Project

University of Michigan, Ann Arbor, MI

Eric Weh, PhD – Basic Research Project

Schepens Eye Institute, Harvard Medical School, Boston, MA

David M. Wu, MD, PhD – Basic Research Project

University of Texas Health Science Center, San Antonio, TX

Ching-Kang Jason Chen, PhD – Basic Research Project

University of Kentucky, Lexington, KY

Jakub K. Famulski, PhD – Basic Research Project

University of Michigan, Ann Arbor, MI

Thanh Hoang, PhD – Basic Research Project

Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, MA

Eleftherios Paschalis Ilios, PhD – Basic Research Project

University of Connecticut

Georgia Zarkada, PhD – Basic Research Project

RRF Cox Macula Society Research Grant – New Clinical Research Project

Administered by The Macula Society

Leo Kim, MD, PhD, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, MA

Research Chairs – Ongoing Proven Research Projects

University of Wisconsin, Madison, WI

Nader Sheibani, PhD – RRF Research Chair

David Gamm, MD, PhD – Humble Distinguished Director, McPherson Eye Research Institute

Kevin W. Eliceiri, PhD – Helmerich Chair, Assoc. Director, McPherson Eye Research Institute

Krishanu Saha, PhD – Murfee Chair, McPherson Eye Research Institute

Bikash Pattnaik, PhD – Albert Chair, McPherson Eye Research Institute

Baylor College of Medicine, Houston, TX

RRF Research Chair – nation-wide search completed at end of 2024

Research Program Overview - 2024

Research Professorships – Ongoing Proven Research Projects

University of Wisconsin, Madison, WI

Sarah Gong, PhD – Gamewell Professor, McPherson Eye Research Institute

Timothy Gomez, PhD – Matthews Professor, McPherson Eye Research Institute

Mrinalini Hoon, PhD – Brown Professor, McPherson Eye Research Institute

Established Awards – Awards Recognizing Lifetime Achievement and Ongoing Research

RRF Award of Merit – presented by The Retina Society

Anat Loewenstein, MD – Tel Aviv Medical Center, Tel Aviv, Israel

RRF Pyron Award – presented by American Society of Retina Specialists (ASRS)

Anat Loewenstein, MD – Tel Aviv Medical Center, Tel Aviv, Israel

CL Schepens MD/AAO Award – presented by American Academy of Ophthalmology (AAO) and in the spirit of Schepens International Society (SIS)

Steven T. Charles, MD – Charles Retina Institute, Germantown, TN

RRF Kayser International Award – presented by International Society for Eye Research (ISER)

Hendrik P.N. Scholl, MD – Medical University of Vienna, Vienna, Austria

RRF Gonin Lecturer – presented by Club Jules Gonin

Anat Loewenstein, MD – Tel Aviv Medical Center, Tel Aviv, Israel

Paul Kayser/RRF Global Award – presented by Pan-American Association of Ophthalmology (PAAO) – will be awarded in 2025

Gonin Medal – presented by International Council of Ophthalmology (ICO) - will be awarded in 2026

International Fellowships – Advanced Subspecialty Training

RRF Helmerich International Fellowships – presented by Ophthalmology Foundation (OF) and administered by International Ophthalmological Fellowship Foundation e. V. (IOFF)

Aim-on Saengsirinavin, MD – from Thailand to Stanford University, Byers Eye Institute, Palo Alto, CA in Uveitis

Lily Myint, MD – from Myanmar (formerly Burma) to LV Prasad Eye Institute in Hyderabad, India in Cataract, Pediatric, Strabismus, and Neuro Ophthalmology

Gillingham Pan-American Fellowships – administered by Pan-American Association of Ophthalmology (PAAO)

Y. Marcela Huertas Bello, MD from Colombia to Toronto Western Hospital, Toronto, Ontario, Canada in Cornea, Anterior Segment, Refractive Surgery

Carolina L. Mercado, MD, from Colombia to Bascom Palmer Eye Institute, Miami, FL in Medical Retina

Nancy Paola Arias González, MD from Dominican Republic to Bascom Palmer Eye Institute, Miami, FL in Pediatric Retina and ROP

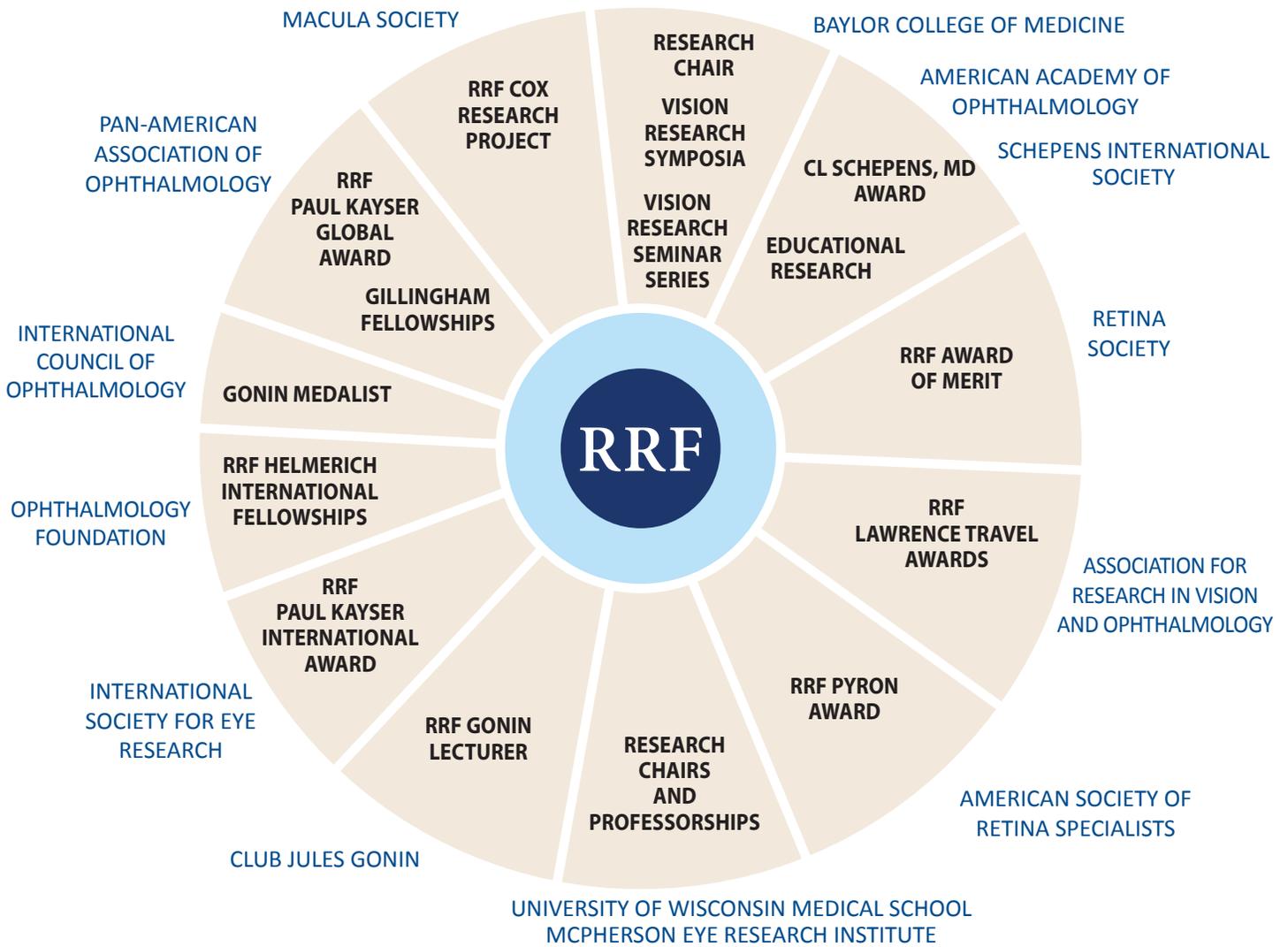
Research Initiatives – Educational and Travel Scholarships

AAO Educational Trust Fund – administered by The Foundation of the American Academy of Ophthalmology (FAAO)

Retina-related educational research programs for clinical and basic science

RRF Lawrence Travel Scholarships – administered by Association for Research in Vision and Ophthalmology (ARVO) – 27 in-person travel scholarships awarded in 2024

Collaborating Organizations



COLLABORATING ORGANIZATION	AWARD	DATE OF FIRST COLLABORATION WITH RRF
RETINA SOCIETY	RRF Award of Merit in Retina Research	1978
ARVO Assoc. for Research in Vision and Ophthalmology	RRF Lawrence Travel Awards	1984
ISER International Society for Eye Research	RRF Paul Kayser International Award	1986
ASRS American Society of Retina Specialists	RRF Pyron Award	1988
PAAO Pan-American Association of Ophthalmology	Gillingham Pan-American Fellowships Paul Kayser/RRF Global Award	1992 2012
AAO American Academy of Ophthalmology	Educational Trust Fund	1993
MACULA SOCIETY	RRF Cox Research Project	1993
CLUB JULES GONIN	RRF Gonin Lecturer	1996
ICO International Council of Ophthalmology with University of Lausanne and Swiss Ophthalmological Society	Gonin Medalist	1998
BAYLOR Baylor College of Medicine	Research Chair	1998
UW University of Wisconsin School of Medicine and Public Health	Research Chairs and Professorships	1998
MERI McPherson Eye Research Institute	Research Chairs and Professorships	2007
AAO American Academy of Ophthalmology with Schepens International Society	Charles L. Schepens, MD/AAO Award	2008
ICO/ICOF International Council of Ophthalmology	RRF Helmerich International Fellowships	2009
OF Ophthalmology Foundation/IOFF	RRF Helmerich International Fellowships	2021
BAYLOR Baylor College of Medicine	BCM Vision Research Symposia	2022
BAYLOR Baylor College of Medicine	BCM Vision Research Seminar Series	2024

Retina Research Sites

Past and Present

TEXAS : 13

Baylor Center for Biotechnology	Texas Children's Hospital
Baylor College of Medicine	University of Houston
Houston Advanced Research Center	University of Texas at Austin
Houston Methodist Hospital	University of Texas at Galveston
UT MD Anderson Cancer Center	University of Texas at Houston
Southwest Research Institute	University of Texas at San Antonio
Texas A&M Health Science Center	

PAN AMERICAN : 23

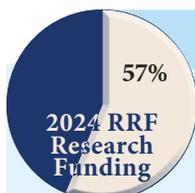
Buenos Aires, Argentina	San Salvador, El Salvador
Curitiba, Argentina	Port-au-Prince, Haiti
La Paz, Bolivia	San Lorenzo, Honduras
Belo Horizonte, Brazil	Aguascalientes, Mexico
Recife, Brazil	Mexico City, Mexico
São Paulo, Brazil	Nuevo León, Mexico
Porto Alegre, Brazil	Asunción, Paraguay
Santiago, Chile	Lima, Peru
Bogotá, Colombia	San Juan, Puerto Rico
Cali, Colombia	Montevideo, Uruguay
San Juan, Costa Rica	Caracas, Venezuela
Santo Domingo, Dominican Republic	

INTERNATIONAL : 52

Al Shifa Trust Eye Hospital	Rawalpindi, Pakistan
Aravind Eye Hospital	Madurai, India
Asahikawa Medical College	Asahikawa, Japan
Beijing Institute of Ophthalmology	Beijing, China
Bern University Hospital	Bern, Switzerland
Centre for Eye Research	Melbourne, Australia
Copenhagen University	Copenhagen, Denmark
Eskisehir Osmangazi University	Eskisehir, Turkey
Eye & Laser World Center	Giza, Egypt
Eye Foundation Hospital	Lagos, Nigeria
Ghent University Hospital	Ghent, Belgium
Hospital Fondation Rothschild	Paris, France
Institut de la Vision	Paris, France
Intercommunal Hospital of Crèteil	Crèteil, France
Jimma University	Jimma, Ethiopia
Jules-Gonin Eye Hospital	Lausanne, Switzerland
Kasindo Eye Clinic	E. Sarajevo, Bosnia & Herzegovina
Keio University	Tokyo, Japan
L V Prasad Eye Institute	Hyderabad, India
Lariboisiere Hospital	Paris, France
Lidcombe Hospital	Sydney, Australia
Lund University	Lund, Sweden
Magrabi ICO Cameroon Eye Institute	Yaounde, Cameroon
Mashhad University Medical Services	Mashhad, Iran
McGill University/Montreal General Hospital	Montreal, Canada
Melles Cornea Clinic	Rotterdam, Netherlands
Moorfields Eye Hospital	London, England
Osaka Medical School/Osaka University	Osaka, Japan
Research Institute of Ophthalmology	Cairo, Egypt
Royal College of Ophthalmologists	Edinburgh, Scotland
Sadguru Netra Chikitsalaya Eye Hospital	Satna, India
Sankara Nethralaya Eye Hospital	Chennai, India
Singapore National Eye Center	Singapore
Siriraj Hospital	Bangkok, Thailand
St. Thomas Hospital	London, UK
Sussex Eye Hospital	Brighton, UK
Tehran University of Medical Sciences	Tehran, Iran
Tel Aviv Medical Center	Tel Aviv, Israel
Toronto Western Hospital	Toronto, Canada
University of Bonn	Bonn, Germany
University of Cambridge	Cambridge, England
University of Erlangen-Nuremberg	Erlangen, Germany
University of Iceland	Reykjavik, Iceland
University of Leipzig	Leipzig, Germany
University of Oxford	Oxford, England
University of Paris	Paris, France
University of Regensburg	Regensburg, Germany
University of Toronto	Toronto, Canada
University of Tübingen	Tübingen, Germany
University of Vienna	Vienna, Austria
University Vita-Salute San Raffaele	Milan, Italy
Western General Hospital	Edinburgh, Scotland

NATIONAL : 69

Augusta University College of Medicine	Augusta, GA
Bascom Palmer Eye Institute	Miami, FL
Beaumont Eye Institute/Hospital	Royal Oak, MI
Byers Eye Institute/Stanford University	Palo Alto, CA
California Institute of Technology	Pasadena, CA
Carver College of Medicine	Iowa City, IA
Case Western Reserve University	Cleveland, OH
Casey Eye Institute	Portland, OR
Charles Retina Institute	Germantown, TN
City College of New York	New York, NY
Cleveland Eye Clinic/Cole Eye Institute	Cleveland, OH
Columbia University	New York, NY
Cornell University Medical College	Ithaca, NY
Dean McGee Eye Institute	Oklahoma City, OK
Duke Eye Center/University Medical School	Durham, NC
Emory University Eye Center	Atlanta, GA
Eye Tech Pharmaceuticals	Worcester, MA
Greater Baltimore Medical Center	Baltimore, MD
Harvard Medical School	Boston, MA
Indiana University	Indianapolis, IN
Johns Hopkins University Medical School	Baltimore, MD
Joslin Diabetes Center	Baltimore, MD
Jules Stein Eye Institute	Los Angeles, CA
Kellogg Eye Center/University of Michigan	Ann Arbor, MI
Kresge Eye Institute	Detroit, MI
Massachusetts Eye & Ear Infirmary	Boston, MA
Massachusetts Institute of Technology	Boston, MA
McPherson Eye Research Institute	Madison, WI
Medical University of South Carolina	Charleston, SC
National Eye Institute	Bethesda, MD
Northeastern University	Boston, MA
Northwestern University	Evanston, IL
Rockefeller University	New York, NY
Schepens Eye Research Institute	Boston, MA
Sheie Eye Institute	Philadelphia, PA
Shiley Eye Center, UC San Diego	La Jolla, CA
St. Joseph's Hospital	Baltimore, MD
Tulane University Medical School	New Orleans, LA
Thomas Jefferson University	Philadelphia, PA
University of Alabama at Birmingham	Birmingham, AL
University of Arizona	Tucson, AZ
University of Buffalo/SUNY	Buffalo NY
University of California	Berkeley, CA
University of California	Irvine, CA
University of California	Los Angeles, CA
University of California	San Francisco, CA
University of Colorado	Aurora, CO
University of Connecticut	Storrs, CT
University of Florida	Gainesville, FL
University of Illinois at Chicago	Chicago, IL
University of Iowa	Iowa City, IA
University of Kansas Medical College	Kansas City, KS
University of Kentucky	Lexington, KY
University of Miami Medical School	Miami, FL
University of Michigan	Ann Arbor, MI
University of Nebraska HSC	Omaha, NE
University of Pennsylvania	Pittsburgh, PA
University of Rochester	Rochester, NY
University of Southern California	Los Angeles, CA
University of Tennessee	Memphis, TN
University of Utah, John A. Moran Eye Center	Salt Lake City, UT
University of Washington	Seattle, WA
University of Wisconsin Medical School	Madison, WI
Vanderbilt University	Nashville, TN
Washington University	St. Louis, MO
Weill Cornell Medicine	New York, NY
West Virginia University School of Medicine	Morgantown, WV
Wills Eye Hospital	Philadelphia, PA
Wilmer Eye Institute	Baltimore, MD



In 2024, the pilot study program represented nearly 60% of RRF's total research funding for the year. RRF funded 30 pilot studies, including five that received funding for the first time. Nine established projects are named in recognition of individuals who have generously supported the RRF mission. Pilot studies are experimental, basic science studies, conducted at leading research institutions that are designed to investigate novel lines of inquiry into the causes of retinal diseases. The hope is that these studies will result in new understanding, advance scientific knowledge, mature into more robust projects, and ultimately, develop into new therapies. RRF commits to funding these studies for a period of up to three years, based upon progress made toward the stated scientific outcome.

The Kathryn and Latimer Murfee Macular Degeneration Project



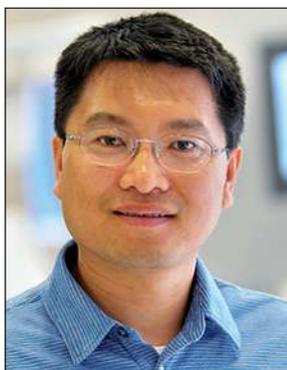
Curtis R. Brandt, PhD
Department of Ophthalmology
and Visual Sciences
McPherson Eye Research
Institute
University of Wisconsin
Madison, WI

Gene Therapy for Retinal Degenerative Diseases

The intent of Dr. Brandt's project is to improve gene therapy for human ocular diseases by reducing the impact of host restriction factors on viral vector transduction efficiency and by gaining greater understanding of the immune response to gene delivery vectors (GDVs).

In 2024, his laboratory examined the effect of clinical-grade AAV vectors on the expression of innate and adaptive immune response genes in human retinal cell lines, and began studies on the human cytomegalovirus vMIA protein, demonstrating that this protein blocked apoptosis and signaling through common innate immune receptors in human Mueller cells.

W.O. Manning Research Project



Rui Chen, PhD
Department of Ophthalmology
University of California
Irvine School of Medicine
Irvine, CA

(Began year affiliated with Baylor College of Medicine, Houston, TX)

Identification and Functional Analysis of Genes Involved in Retina Diseases

The long-term goal of Dr. Chen's research is to improve the ability to prevent, diagnose, and treat human retinal diseases accomplished by the identification and functional characterization of newly discovered disease genes underlying the diseases. Since many human eye disease genes are involved in normal eye development, the Chen laboratory utilizes animal model systems to study retinal

development. Results from these studies deepens our knowledge about human retinal disease and forms the basis of developing optimal treatments.

In 2024, Dr. Chen continued his investigation of the function of a newly identified inherited retinal disease gene, *Tlcd3b* in the retina, and performed gene therapy on the *Tlcd3b* KO model, which demonstrated significant rescue via gene replacement therapy. This preclinical result lays the foundation for future development of human gene therapy, and was published in ARVO's publication, *IOVS*.

Dr. Chen also performs functional studies for candidate disease genes and gene therapy. By analyzing whole genome sequencing data on patients with inherited retinal diseases, his laboratory has identified several novel candidate disease genes, including *DDX41*, whose function in the retina has not been investigated. To further establish the association of *DDX41* with Retinitis Pigmentosa disease, a *Ddx41* knockout mice model was developed, and will be characterized to provide critical insights of *DDX41* pathophysiology in patients.

Joe M. and Eula C. Lawrence Research Project



Timothy W. Corson, PhD
Faculty of Pharmacy
University of Toronto
Toronto, ON, Canada

Effect of Soluble Epoxide Hydrolase Loss on a Dry AMD-like Phenotype

Dr. Corson's long-term goal is to find new therapeutic approaches for age-related macular degeneration (AMD), a disease characterized by abnormal blood vessel growth ("wet" AMD) and retinal pigment epithelium (RPE) dysfunction ("dry" AMD). The specific goal of the current project is to explore how a chemical called SH-11037 that his laboratory developed, influences all the gene readouts in blood vessel and RPE cells. SH-11037 blocks the function of soluble, epoxide hydrolase (sEH), an enzyme identified to be important for abnormal new blood vessel growth and more recently for inflammation.

In previous years of RRF funding, Dr. Corson found sEH as a target of SH-11037, and showed that sEH is present at high levels in human and mouse eyes with AMD-like features. The lab further found that sEH inhibitors can block new blood vessel growth in the eye. The team characterized the molecular mechanism of how SH-11037 inhibits sEH, and identified factors that increase its levels in the eye. Dr. Corson's team assessed their library of novel chemicals to build a "structure activity relationship" for blocking sEH function. They showed differential expression in sEH between the sexes, found that depletion of sEH with a genetic tool they developed reduces inflammatory signals, and revealed RPE as a major source of this protein. They also explored whether sEH regulates the permeability of blood vessels in culture, finding that sEH inhibition can decrease leakiness of blood vessels. In 2024, Dr. Corson began to assess if dry AMD-like features are improved in the mouse model in which sEH can be specifically "turned off" in the RPE cells, and showed that these features can be improved by another novel anti-angiogenic and anti-inflammatory drug-like molecule he worked with. Dr. Corson filed a patent in May, 2024 based upon his work, and shared his research findings in a manuscript published in the *Archives of Pharmacal Research*.

The Paul Kayser Research Project



Samuel Wu, PhD
Cullen Eye Institute
Neurosensory Center
Baylor College of Medicine
Houston, TX

Pharmacological and Genetic Mechanisms Underlying Retinal Cell Death in Age-Related Macular Degeneration (AMD) and Glaucoma

Dr. Wu's lab team continued their research by using the novel 8-patch electrode recording system, developed by his laboratory, and used for the first time in retinal research. His lab has successfully developed novel analytic tools for studying retinal synaptic connectivity and spatiotemporal receptive field properties of ganglion cells in normal, AMD and glaucomatous retinas. They study the morphology and

synaptic inputs of various types of retinal ganglion cells (RGCs) and identify targets for drug and gene therapies for treating RGC dysfunction in glaucoma and AMD.

The team investigates postsynaptic responses elicited by current injections into presynaptic retinal neurons and the receptive fields of retinal ganglion cells (RGCs) in normal, AMD and glaucomatous retinas. They also are identifying targets for drug and gene therapies for treating glaucoma and AMD, and specifically, studied photoreceptor-bipolar cell-retinal ganglion cell (RGC) synapses in healthy and glaucomatous retinas. Additionally, they are determining the linear and non-linear behaviors of the photoreceptor-coupled network that regulate retinal function in age-related macular degeneration (AMD). Moreover, as the Director of the Baylor Vision Research Training Program, Dr. Wu continues to manage his five-year National Eye Institute T32 grant. His group published three papers throughout the year and gave numerous presentations on their research.

Bertha and I.L. Miller Research Project



Graeme Mardon, PhD
Departments of Pathology,
Molecular and Human Genetics
Baylor College of Medicine
Houston, TX

**Genetic and Molecular
Analysis of Retinal
Development**

Dr. Mardon's long-term objective is to improve prevention and treatment for human retinal diseases. He uses the power of the *Drosophila* fly eye system to dramatically increase our understanding of the critical onecut gene family in retinal development. A detailed understanding of the molecular mechanisms of retinal cell fate determination could have broad implications for our ability to diagnose, prevent, and treat human retinal diseases.

There are three mammalian homologs of onecut, and two (Oc1 and Oc2) are required for normal retinal development. Specifically, loss of either gene causes a partial loss of horizontal cells while loss of both genes causes a complete loss of all horizontal cells and partial loss of several other retinal cell types. However, the molecular details of how these genes function is unknown. In 2024, Dr. Mardon completed the collection of single cell RNA sequence data on onecut mutant retinas, and reached a major breakthrough concerning this important retinal gene. In particular, the lab found that onecut is required for expression of many genes involved lipid droplet formation and mitochondrial function, two processes known to play important roles in neural degeneration. Moreover, the lab made complete loss of function mutations in the *Drosophila* onecut gene and found that these mutants undergo progressive photoreceptor degeneration. This work represents a major step forward in the understanding of basic mechanisms of retinal cell fate determination. Dr. Mardon and his collaborators published four manuscripts throughout the year.

Adolphe G. and Josephine Roberts Gueymard Research Project



Lih Kuo, PhD
Department of Medical
Physiology
Texas A&M University Health
Science Center
Bryan, TX

**Activation of Endothelin-
Dependent RhoA/ROCK
Pathway Elicits Retinal
Microvascular Dysfunction in
Diabetic Retinopathy**

This project seeks to explain the mechanisms that are responsible for the microvascular pathogenesis of diabetic retinopathy and to develop strategies and related tools for the prevention and treatment of the sight-threatening disease.

Proper function of the retina depends on an adequate blood supply to the retinal tissue, while dysfunction of the retinal microcirculation could lead to disease development. Dr. Kuo has found that in the diabetic retina, the synthesis of vasoconstrictor/inflammation agent endothelin-1 (ET-1) from vascular endothelin converting enzyme (ECE) is elevated, corresponding to the activation of RhoA kinase (ROCK) and arginase enzymes. He hypothesizes that ECE/ROCK/arginase signaling contributes to microvascular dysfunction and leads to ischemia underlying the development of retinopathy.

Using mouse models and pig models, which resembles the human eye's circulation and cardiovascular disease, Dr. Kuo's laboratory investigates vascular signaling pathways in the initiation and development of diabetic retinopathy with the goal of developing pharmacological strategies for disease prevention and treatment.

Dr. Kuo's results indicate that early diabetes elicits a significant retinal thinning slightly before the development of retinal flow reduction in a mouse model of type 1 diabetes. The flow deficiency is associated with impaired endothelium-dependent nitric oxide-mediated vasodilation in ophthalmic arteries feeding to the retinal microcirculation. The changes in vascular reactivity can promote retinal tissue ischemia due to impaired vasodilation and augmented vasoconstriction under hyperglycemic insults. The lab also found that intravitreal injection of stanniocalcin-1 (STC-1), a secreted peptide displaying multiple regulatory functions in cell survival and death, rescues photoreceptor degeneration with reduced oxidative stress and inflammation in rhodopsin transgenic pigs representing inherited human retinitis pigmentosa (RP). Since the loss of photoreceptor cells is associated with retinal vascular degeneration in RP, retinal blood flow dysregulation might be related to neural degeneration during diabetes initiation/progression. Treatment of neuropathy and blood flow deficiency in early diabetes by STC-1 may be critical before the establishment of overt neurovascular pathology.

Mary Ellen Wilson Research Project



Richard L. Hurwitz, MD
Department of Pediatrics
Baylor College of Medicine
Houston, TX

Immune Consequences of Gene Therapy for Ocular Disorders

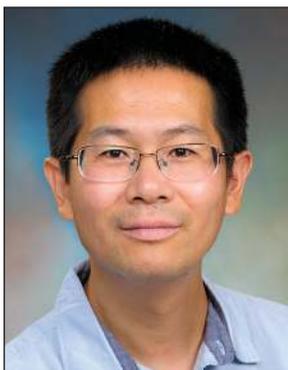
Retinoblastoma, an ocular cancer that develops from the retinas of young children, can be caused either by spontaneous growth of a tumor in one eye or by an inherited mutation that often causes tumors in both eyes. To preserve the vision of these young patients, Dr Hurwitz is intent on developing alternative treatments that will preserve vision, reduce the risk of developing other cancers, and reduce the impact of the emotional and physical scars from enucleation. Dr. Hurwitz completed the first clinical trials that used a suicide gene therapy to treat children with advanced retinoblastoma. The successful reduction of vitreous seeds has encouraged him to continue his research initiatives to improve this innovative therapy. Additionally, Dr. Hurwitz is interested in developing gene therapy options for retinal degenerative disorders such as Stargardt Disease (juvenile macular dystrophy) that currently have no cure and have limited treatment options to slow the degenerative processes.

Some of these disorders are associated with gene mutations that manifest in abnormal structure or function of proteins responsible for normal vision. He approaches this challenge from the perspective of finding therapeutic alternatives to deliver drug and gene therapies that enhance efficacy and reduce toxicity directly in the eye.

The Hurwitz team previously published an association of the vitreous component hyaluronan with the enhanced expression of potentially therapeutic genes transferred by adenoviral vectors. Hyaluronan alone does not account for the entire effect observed. Versican is a component of vitreous that binds hyaluronan and is made up of three domains called G1 and G3, separated by a chondroitin-binding domain. The effects of these domains have been examined using expression constructs that span the known functional elements that may affect transgene expression. G1 and G3 have been shown to have a complex interplay in regulating gene expression. G1 also delivers hyaluronan to the cell surface where it further enhances gene expression. These constructs may be useful in designing more efficient vectors and delivery systems to optimize gene therapy outcomes and limit toxicities, including immune consequences.

Further, Dr. Hurwitz continues to explore an alternative, non-invasive delivery approach that uses microwafers loaded with nanoparticles to deliver therapeutic drugs or genes directly to the eye without the need for surgery or injections. He has shown that chemotherapeutic agents can be delivered locally to the eye and treat retinoblastoma in a murine model of the disease.

Harry E. Bovay, Jr. Research Project



Wenbo Zhang, PhD
Department of Ophthalmology
& Visual Sciences
University of Texas Medical
Branch at Galveston
Galveston, TX

Novel Therapy for Retinal Neovascularization

Dr. Zhang's goal is to develop a novel, effective and inexpensive approach to treat ischemic retinopathy. Ischemic retinopathies, such as diabetic retinopathy, retinopathy of prematurity, and retinal vascular occlusion, affect a variety of patient populations, and often result in irreversible vision loss due to the development and growth of abnormal new vessels subsequent to a period of retinal ischemia. This process is referred as retinal neovascularization. At present, therapies for ischemic retinopathy mainly target retinal

neovascularization. These treatments are limited, not always effective, have considerable side effects, and are expensive.

In 2024, the Zhang lab determined and characterized the role of Epac1 in the injury of retinal inner neurons in ischemic retinopathy using a mouse model of oxygen-induced retinopathy (OIR). The lab found that Epac1 deletion attenuated OIR-induced dysfunction and degeneration of rod bipolar cells, amacrine cells, and the connectivity between rod bipolar cells and amacrine cells. Epac1 deletion also reduced retinal inflammation and gliosis. Using scRNAseq, the team provided the first evidence that Müller glial cells formed two new subclusters during OIR, and that Epac1 may be involved in this process. Dr. Zhang's team shared their results in various ways, including publication of a manuscript based on the data generated in *Molecular Therapy*, presentation of an abstract during the ARVO annual conference on May, 2024, and presentation of two abstracts during ISER conference in October, 2024, one as an oral presentation and the other as a poster presentation.

Dana and Gil Petri Research Project



Yingbin Fu, PhD
Cullen Eye Institute
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Houston, TX

**Targeting Subretinal Fibrosis
in Age-Related Macular
Degeneration**

Age-related macular degeneration (AMD) is a major cause of blindness in the elderly. Neovascular AMD (nAMD), caused by the growth of abnormal, leaky blood vessels beneath the retina, accounts for 80-90% of cases of legal blindness due to AMD. Up to 50% of patients have poor responses to current anti-VEGF treatments, including persistent fluid and unresolved or new hemorrhages. Metabolic pathways have essential roles in regulating blood vessel growth in

endothelial cells. ATP citrate lyase (ACLY) is a key enzyme in cellular metabolism that may play a role in nAMD. The objective of Dr. Fu's research in this area is to develop a novel treatment for nAMD by targeting ACLY.

In the past year, Dr. Fu's laboratory developed a CHP-based imaging tool for the long-term, accurate detection of subretinal fibrosis (scar formation) in an AMD model, eliminating the need for repeated injections. This tool is invaluable for both diagnostic and treatment purposes. Additionally, his team demonstrated that the AIBP/apoA-I/anti-VEGF combination therapy significantly reduced subretinal fibrosis, whereas the current anti-VEGF therapy, aflibercept, was ineffective. This finding is highly significant, as it shows that the combination therapy not only overcomes anti-VEGF resistance but also reduces subretinal fibrosis. Dr. Fu's significant findings were shared through a published article in *Biomolecules*, in August, 2024, with an additional manuscript under revision.

Basic Research Projects



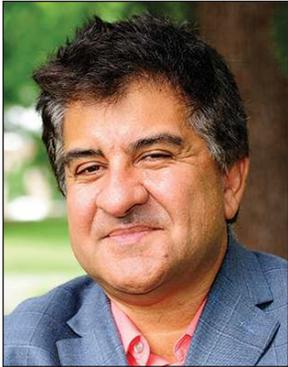
Jianhai Du, PhD
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West Virginia University School
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**Target NAD Degradation
in Age-Related Macular
Degeneration**

Dr. Du's research project will define how the breakdown of NAD⁺, a key molecule controlling metabolism, influences the pathogenesis of age-related macular degeneration (AMD), and determine whether its inhibitors can slow or stop the progression of RPE dysfunction that leads to macular degeneration. Dr. Du wants to understand if stopping the breakdown of NAD⁺ can protect the retina from degeneration. Inhibitors of NAD⁺ degradation are currently available from natural products, so the findings from this research can potentially be readily translated to benefit AMD patients.

In the second year of this pilot project, Dr. Du's lab made significant progress, and found that NAD⁺ primarily breaks down in the RPE, but its product can be exported to regulate metabolism in the neural retina. The team developed new methods to quantify the NAD⁺ breakdown and synthesis, demonstrating that NAD⁺ degradation is enhanced in the aged RPE. Moreover, NNMT, an enzyme by-product of NAD⁺ breakdown, is found in higher concentrations in the RPE of older mice. The laboratory generated and validated a genetically modified animal line to delete this enzyme, and plans to further investigate how NAD⁺ degradation in the RPE is associated with age-related visual decline with age by measuring visual function and resulting enzyme changes. Dr. Du also plans to determine the role of NAD⁺ degradation pathways in an AMD-like animal model in their protection of retinal metabolism, visual function, and retinal degeneration.

Dr. Du published four papers to share his important progress, and additionally, members of the Du Lab travelled to Seattle, WA, to attend ARVO 2024 and to Buenos Aires, Argentina, to attend the International Society of Eye Research (ISER) 2024 conference to present their research findings.



Francesco Giorgianni, PhD
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Memphis, TN

CD5L-Mediated Autophagocytosis in RPE Cells

Currently, there are no effective treatments for “dry” AMD. CD5L is a scavenger receptor that, among other functions, regulates the cellular content of long-chain fatty acids, it is capable of binding OxLDL and, as Dr. Giorgianni’s

preliminary data suggest, mediates its intracellular degradation. OxLDL is a highly cytotoxic and pro-inflammatory product of oxidative stress, and it is known to play a causal role in the onset of AMD. If Dr. Giorgianni’s studies confirm a significant role of CD5L in the clearance mechanisms of oxidized species in the RPE, such as OxLDL, and consequent cytoprotection, these findings will pave the way to design treatments that could eliminate or at least delay the onset of the pathophysiological changes that occur in the RPE during AMD onset and progression. CD5L is a small, secreted protein that cellular uptake is known to be mediated by the CD36 receptor. It is easy to envision treatment of AMD patients by localized injections of human recombinant CD5L analogously to the current intervention methods used to treat wet AMD via injection of anti-VEGF monoclonal antibodies.



Milam A. Brantley, Jr., MD, PhD
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The Cellular Mechanisms by Which Arginine and Citrulline Promote Vision-Threatening Diabetic Retinopathy

Diabetic retinopathy (DR) is a potentially devastating complication of diabetes and a leading cause of blindness worldwide. Given the significant challenges of treating the advanced vision threatening stages of DR, it is critical that new treatments are developed that can slow DR progression and reduce the chance of severe vision loss in these patients. The most important risk factors contributing to vision-threatening DR are poor blood sugar control and increasing duration of diabetes, but these primary risk factors do not explain the high variability among patients in the clinical progression of DR. Specifically, it is unknown why some patients with excellent blood glucose control go on to develop severe DR and other patients with poor glucose control never experience retinopathy. It is clear that additional factors, possibly genetic, metabolic, or environmental, can also significantly influence DR risk. To investigate other potential DR risk factors, Dr. Brantley’s lab performed a metabolomics study in which we measured thousands of molecules called metabolites in the blood of patients with diabetes.

Dr. Brantley found elevated blood levels of two specific molecules, arginine and citrulline, in patients who had DR compared to those without DR. Arginine and citrulline are involved in two critical overlapping metabolic pathways in the body, so we next evaluated blood levels for six key metabolites in these pathways. Arginine and citrulline were found to be the only two abnormally elevated metabolites from these metabolic pathways in patients with DR. These results made scientific sense because previous research studies had suggested that arginine related metabolites could be important in DR, but it is not yet known exactly how these molecules influence the likelihood of a patient developing diabetic retinopathy. To determine if elevated arginine and citrulline could contribute to the development of the abnormal retinal blood vessels seen in sight threatening DR, arginine and citrulline were added in combination to retinal endothelial cells, the cells that make up the blood vessels in the retina. Arginine and citrulline caused the endothelial cells to become leaky and to form new blood vessel tubes, two classic features of diabetic retinopathy. Given this exciting preliminary data, the purpose of the current study is to understand precisely how arginine and citrulline alter the cells in the retina that are specifically involved in DR. In the first three years of experiments, Dr. Brantley has shown that arginine and citrulline promote blood vessel formation by activating the nitric oxide pathway and that this requires AMPK and Akt protein signaling. His lab also demonstrated that arginine and citrulline cause vessel permeability by disruption of claudin-5 localization at the cell membrane. In 2024, Dr. Brantley conducted studies to determine the specific molecular mechanisms by which citrulline and arginine activate the nitric oxide pathway to cause an angiogenic response and increase permeability. Dr. Brantley believes these studies will help develop new ways of treating, or even preventing, diabetic retinopathy.



James Monaghan, PhD
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**Stem Cell Fate Determination
During Axolotl Retina
Regeneration**

Dr. Monaghan researches the Mexican axolotl salamander to understand the molecular mechanisms that drive axolotl regeneration of retinal cells. Mammalian retinas do not regenerate. Greater knowledge of the unique mechanisms the axolotl uses that enable this regeneration may ultimately

lead to strategies for the restoration and replacement of damaged or missing human retinal cells and tissues.

This project aims to develop a retinal atlas and stem cell fate maps of axolotl retina regeneration and study the molecular mechanisms driving the process. The Monaghan lab continues to work on developing the axolotl retina regeneration model to gain insights into the molecular and cellular mechanisms behind retinal regeneration. Over the past year, the lab created the first cell-type atlas of the axolotl retina and examined its response to NMDA injury, which was presented at the annual ARVO meeting. This research has become the primary focus for Nicole Calder, a second year PhD student, and Emil Kriukov, a visiting MD bioinformatics researcher. Dr. Monaghan has also conducted a transcriptomic comparison of extracellular matrix genes expressed in the axolotl versus pig retina.



Seongjin Seo, PhD
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**Gene Therapy Vectors To
Treat Retinal Degenerations
Associated With ABCA4,
USH1D and USH1F**

The long-term goal of Dr. Seo's research program is to develop adeno-associated virus (AAV) based gene therapy

vectors to prevent vision loss in individuals with Usher syndrome, a genetic disorder impacting both vision and hearing in children and young adults. While AAV is a safe and efficient gene delivery vehicle, its main drawback is the limited packaging capacity. This study targets the four most commonly mutated Usher syndrome genes: USH2A, MYO7A, PCDH15, and CDH23. These genes collectively represent over 90% of Usher syndrome cases and require at least two AAV vectors for delivery. The objective of this study is to produce effective gene therapy vectors for each specific gene. To that end, Dr. Seo developed three strategies for the delivery of large genes using AAV, and identified the most suitable and effective strategy for the aforementioned Usher syndrome genes.



Andrius Kazlauskas, PhD
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**Hyperglycemia-Induced
Mitochondrial Adaptation**

Diabetic retinopathy (DR) is an eye disease that can happen to people with diabetes and can eventually lead to vision loss. Keeping blood sugar under control helps reduce the risk, but even with good control, about one in four people with diabetes still develop DR. This means more ways to protect vision in diabetic patients are needed.

diabetes. This delay suggests that the body has natural defenses that protect the eyes—a phenomenon called resilience to DR (RDR). Surprisingly, while most research focuses on how diabetes damages the eye, very little has been done to understand why some eyes stay healthy for so long.

Dr. Kazlauskas' long-term goal is to develop treatments that boost these natural defenses so that DR never develops. The first step is to find the specific genes and biological pathways that make this resilience possible.

In 2024, the team studied mice that show RDR. They used advanced DNA sequencing techniques to examine the retinas (the light-sensing tissue in the back of the eye) and identify which genes and cell types are linked to this natural protection. With this information, they aim to design treatments that could one day prevent DR in people with diabetes. Dr. Kazlauskas' team published two papers in 2024 in the peer-reviewed journals: *Journal of Visualized Experiments* and *International Journal of Molecular Sciences*, and a review article in *Progress in Retinal and Eye Research*.

In most people, serious vision problems from DR do not appear until more than 20 years after being diagnosed with



Vladimir Kefalov, PhD
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Understanding How the G90D and G90V Rhodopsin Mutations Cause Blindness

The exquisite sensitivity of the human rod photoreceptors that enables sight in dim to moderately bright light requires that rhodopsin, a pigment-containing sensory protein that converts light into an electrical signal, is extremely stable and does not activate the rods in the absence of light. Recently, two mutations in rhodopsin have been identified that appear to compromise its stability: Glycine 90 to Aspartate (G90D) reported to cause congenital stationary night blindness (CSNB), and Glycine 90 to Valine (G90V) found in patients with retinitis pigmentosa (RP). Dr. Kefalov's project goal is to identify the molecular mechanism by which these mutations cause abnormal photoreceptor function and degeneration

as a first step in developing effective treatments for people carrying these mutations.

Initial studies found that the G90D and G90V mutations altered distinctly the stability of metarhodopsin, resulting in very different Meta II decay rates for the two mutants. To investigate the functional significance of this altered rhodopsin stability and decay, the Kefalov team investigated the kinetics of rod dark adaptation of WT, G90D and G90V homozygous mice using *in vivo* ERG recordings. Following a >90% bleach, the amplitude of rod a-wave recovered to 50% of its pre-bleached level in 63 min for WT rods; 22 min for G90V rods; and 10 min for G90D rods. Thus, both mutations greatly accelerate the regeneration of rhodopsin *in vivo*, while also producing phenotypes distinct from each other. This accelerated dark adaptation indicates a higher-than-normal phototoxicity in the mutant rods, and might help explain their progressive degeneration, which perhaps surprisingly appears to be more severe in the G90D compared to the G90V rods. Excitingly, the results from this project are such that in the fall of 2024, Dr. Kefalov was awarded funding through an R01 grant from the National Eye Institute to continue his research into the next phase.



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Investigation and Modulation of Inner Retinal Dysfunction in Diabetes

Visual deficits in the function of dim light-activated rod pathways are some of the earliest identifiable retinal problems experienced by diabetic patients. Electrical recordings from retinas show dysfunction in the inner retina and these deficits are tied to the development of serious diabetic retinal problems. Dr. Eggers has shown that deficits in the light response of neurons in the inner retina, which are part of the dim light rod pathway, are not due to cell death, but to

some unknown mechanism. Dopamine, a neurotransmitter and hormone, is released by dopaminergic amacrine cells to allow the retina to adapt to increasing levels of background light. Diabetic retinas have been found to have low dopamine levels, and perhaps, supplementation of dopamine can reduce inner retinal deficits in diabetes. Drawing from her extensive expertise in rod pathway signaling for this project, Dr. Eggers will determine if this pathway is specifically vulnerable to diabetic damage and identify the mechanism of dysfunction in order to develop targeted therapeutics for prevention of the neuronal progression of vision loss.

In 2024, Dr. Eggers published studies using the *in vitro* ERG showing that both dopamine D1 and D4 receptors are less sensitive in the diabetic retina with no change in receptor mRNA expression in *Experimental Eye Research*. Her laboratory also obtained preliminary data that the *in vitro* ERG response to light adaptation is reduced in the diabetic retina, which was presented at ARVO.



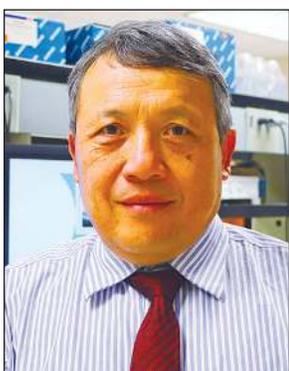
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**Retinal Damage and
Regeneration in the African
Spiny Mouse (*Acomys
Cahirinus*): A Novel
Mammalian Model for
Translational Research**

It is commonly believed that the ability to regenerate neurons in vertebrates is an exclusive property of non-mammalian species such as fish and amphibians. However, in recent years, spiny mice (*Acomys*) have become the focus of intense research for their enhanced wound repair and regenerative ability in many tissues, raising the question of whether these mammals might regenerate retinal neurons in response to damage. The purpose of Dr. Morris' project is to test the hypothesis that the spiny mouse possesses the capacity to

regenerate retinal neurons in response to damage, and to take the first steps in determining the underlying biological mechanism -- this would be the first demonstration of natural regenerative ability in the retina of any mammal.

In 2024, Dr. Morris and her team continued to investigate how the spiny mouse (*Acomys*) retina responds differently to acute injury compared to the common laboratory mouse (*Mus*). Following acute retinal damage that causes extensive loss of retinal ganglion cells (RGCs), Dr. Morris observed a significant increase in RGC number in *Acomys* beginning at 21 days post injury. However, her team also observed extensive variability among animals, with some spiny mice showing a strong RGC recovery, and others displaying weak recovery (and these differences were not due to age, size, or sex). Given the well documented propensity of spiny mice to develop type II diabetes, Dr. Morris hypothesizes that the differential RGC recovery response is due to differences in blood sugar levels, and she will continue to pursue this line of inquiry. Dr. Morris and her research team's results were published in an article in *Experimental Eye Research*, October, 2024.



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**The Roles of RIP Kinase 3
in the Development of
AMD-Like Pathologies
During Cytomegalovirus
Ocular Latency**

Dr. Zhang's research seeks to understand the underlying causes that contribute to the development of AMD, which remain uncertain but are highly correlated with cell immunological/inflammatory mechanisms in the various tissue layers of the retina. Receptor-interacting protein (RIP) kinases have been identified as modulators of inflammatory responses and play an important role in these tissues' specific innate immunity, autophagy and death-inducing processes. Dr. Zhang's studies are the first to

explore a possible viral cause of AMD occurring as a result of an inactive, past ocular viral infection.

In 2024, Dr. Zhang tested the hypotheses that RIP3 contributes to the death and/or degeneration of ocular cells/tissues and development of AMD-like pathologies via the production of inflammatory factors, activation of cell death pathways, and via cross-talk among cell death, autophagy and LC3-associated phagocytosis (LAP) using mouse models of murine cytomegalovirus (MCMV) ocular latency.

His study results suggest that RIP3 contributes to retinal degeneration during MCMV ocular latency via increased inflammation and subsequent cell death by apoptosis and necroptosis. A manuscript based on these results is in preparation. Additionally, the mean retinal thickness was significantly lower in eyes of latently infected macrophage/microglia-specific, RIP3 depleted mice, compared to eyes of latently infected control mice, although no significant difference was noted between RPE-specific, RIP3 depleted mice and control mice. And lastly, a lack of RIP3 increased LC3-associated phagocytosis of photoreceptor outer segment (POS).



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**Modeling EYS Associated
Retinitis Pigmentosa in
Human iPSC Derived Retinal
Organoids**

Mutations in the Eyes Shut *Drosophila* homolog (EYS) gene are the leading cause of retinitis pigmentosa (RP) in Asia and one of the five most mutated RP genes in the US and Europe. Despite the high prevalence of this disease, little is known about the role of EYS in the retina and the molecular mechanism of the EYS-associated disease.

Dr. Bujakowska is using confocal microscopy and high-resolution expansion microscopy (Ex-M) to gain nanoscale

insights into protein localization, particularly in the ciliary/periciliary structures of the photoreceptors in EYS RD retinal organoids and *eyes* knock-out zebrafish. In light of a recent study suggesting that EYS is involved in the trafficking of the outer segment protein G protein-coupled receptor kinase 7 (GRK7)16, which plays a key role in photo-response recovery, her team is further investigating alterations in the trafficking of other phototransduction proteins, particularly those critical to rod function, using our EYS-RD retinal organoids and *eyes* knock-out zebrafish models.

Dr. Bujakowska collaborated with Aves Lab to develop a new anti-EYS antibody and successfully immunized hens, yielding antibodies with strong enzymatic activity as confirmed by ELISA. Additionally, her laboratory used CRISPR-based genome editing to create EYS knock-out iPSC lines, with retinal organoids being generated to study the effects of EYS depletion on retinal development. EYS expression was detected in retinal organoids as early as day 70, and further characterization of EYS-deficient organoids will continue into 2025.



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**A Proteomic Analysis of
Mitochondria-ER Contact Sites
in Muller glia and the Potential
Role of Metabolomic Changes
in Regulating Intrinsic Retinal
Regeneration**

The overarching goal of Dr. Gross' research is to identify the molecular underpinnings of Muller glia-dependent retinal regeneration. In their second year of funding, the Gross laboratory focused on a previously unexplored aspect of the regenerative process – how metabolic changes facilitate Muller glia activation, reprogramming and regenerative

responses. The team performed targeted metabolomic analyses of quiescent and injured retina, with damage achieved using a photoreceptor degeneration paradigm. They profiled several time points post-injury as well as after treatment with DCA (dichloroacetic acid), which blocks pyruvate dehydrogenase kinase and thereby increases the rate of glucose oxidation, shifting the metabolic profile of cells towards oxidative phosphorylation at the expense of glycolysis. Plans for the coming year include experiments that will build off of metabolomic profiling data generated during the 2024 funding period to continue to focus on metabolic changes in Muller glia, and how they regulate Muller glia reprogramming and regenerative responses. These results will be significant because proteins and metabolites that we identify can then potentially serve as foundations for the development of new therapeutic approaches aimed at stimulating intrinsic regeneration in the human retina.



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University of California
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San Francisco, CA

Measuring Fluid Clearance Pathways in Retinal Edema

Retinal edema is a serious condition that can distort vision and if left untreated, can cause blindness. To better understand the causes underlying this condition, Dr. Smith proposes to apply novel imaging techniques, developed

in his laboratory for measuring fluid transport in the perivascular spaces of the brain. He is further developing these tools for application in the eye, and proposes to measure the routes of fluid clearance from the retina and to determine if there are specific structural barriers to clearance. The water channel protein, aquaporin 4, is thought to be important for fluid clearance from the brain, and Dr. Smith will perform additional experiments to test if aquaporin-4 regulates fluid clearance in a mouse model of diabetic retinal edema. Results of these experiments will identify new pathways and molecular targets for therapeutics designed to accelerate retinal fluid clearance. Further in 2024, Dr. Smith characterized changes in extracellular structure that occur in response to retinal inflammation to determine how inflammation alters fluid movement through the extracellular space of the retina.



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Treatment and Prevention of PVR and Retinal Detachment

The knowledge obtained from Dr. Sorenson's research will facilitate the design of more personalized treatments for proliferative vitreoretinopathy (PVR) as well as other eye diseases in which scar formation on the retina is a major problem. Her project's goal is to understand how to prevent or decrease scar formation that can cause blurred vision, distortions, or even permanent vision loss.

Retinal detachment (RD) is a vision-threatening condition that requires surgical treatment. In up to 20% of patients, RD is accompanied by inflammation that plays a pivotal role in the pathogenesis of PVR thus driving retinal scar formation. The innovation of Dr. Sorenson's current line of research into the role of the gene Bcl2, which makes a protein that helps control whether a cell lives or dies, is that it ties Bcl-2's role in the prevention of cell death to

sustained inflammation that leads to retinal fibrosis and scar establishment and thus prevents optimal vision outcomes for patients who specifically suffer from rhegmatogenous retinal detachment (RRD). By identifying the critical role Bcl-2 plays in this process, and inhibiting it, Dr Sorenson hopes to decrease the size of retinal scars in formation, and to shrink those that have already formed. The knowledge gained from these studies also should translate to other ophthalmic diseases with a fibrotic component. For many of these diseases, limited treatment options exist and therefore, the research Dr. Sorenson is undertaking will provide a novel perspective on how effectively to limit inflammation and prevent retinal fibrosis and scar establishment to improve vision outcomes.

Dr. Sorenson's lab made excellent progress in 2024. The team established the optimal process for assessing scar formation while also determining whether Bcl-2 expression in mononuclear phagocytes (MP) influences the amount of fibrosis development in mice models of PVR. They discovered that responses vary based upon animal gender, which will allow the team to assess different aspects of inflammation and scar development in their models. The research was repeated and quantified, enabling forward movement to the next stage where Dr. Sorenson's studies will pursue better understanding of the importance of modulating immune cell turnover to effectively treat PVR.



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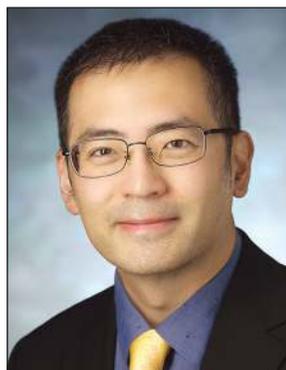
Developing a Novel Treatment to Prevent Vision Loss due to Recurrent Retinal Toxoplasmosis

Regrettably, a fraction of patients experiencing an infection from the common *Toxoplasma gondii* (Tg) parasite will develop ocular toxoplasmosis that can cause vision loss. Even worse, the disease tends to progress with time as the infection will spontaneously resolve and then recur, further eroding vision. There are currently no treatments available to prevent the recurrence of ocular toxoplasmosis, which means that patients with this disease are likely to continue to experience worsening vision over time.

To help identify promising new therapies to prevent recurrence of ocular toxoplasmosis, Dr. Weh is collaborating with his University of Michigan colleague, Dr. Vernon B. Carruthers to develop and test new drugs that can selectively kill Tg parasites and prevent disease. Together they are designing

and testing drugs that can be injected directly into the eye to kill Tg parasites at the site of infection. This method, called intravitreal injection, is used daily in ophthalmology clinics to treat diseases such as Age-related Macular Degeneration (AMD). By killing the Tg parasites in the eye when ocular toxoplasmosis occurs, Dr. Weh and Dr. Carruthers believe they will be able to prevent patients from experiencing recurring ocular toxoplasmosis and preserve their vision.

During the year, Dr. Weh tested two additional candidate compounds for retinal toxicity, and did not find any evidence of toxicity to retinal structure or function, confirming previous data that the target protein inhibitors are non-toxic to the retina. Pharmacokinetic experiments found the inhibitors present in the retina 7 days following a single IVT injection at a therapeutic concentration. Dr. Weh also tested the efficacy of a different target protein following two intravitreal doses, however no significant decrease in parasite burden was found. The concentration was increased 100x the previous dose, and still no evidence of retinal toxicity was found, but the pharmacokinetic analysis showed extremely rapid clearance, suggesting this second protein may be crossing the blood-retina barrier. Dr. Weh is encouraged that systemic administration confirmed the ability for this compound to cross the blood-retina and blood-brain barriers, a positive result in his search for a novel treatment for this recurrent infection.



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Metabolic Modulation of Complement in the Retinal Pigment Epithelium

Dr. Wu's project seeks to understand how the retinal pigment epithelium (RPE), the cell type in the eye central to the pathology of age-related macular degeneration (AMD), becomes more vulnerable to damage as the metabolism of the eye changes with age.

Throughout 2024, Dr. Wu continued his studies in the iPSC-RPE model (induced pluripotent stem cell derived RPE cell) that allows his team to grow RPE cells from patients without damaging their eyes. This allows study of how a patient's own genetic background may affect different cellular processes. His research showed that a normal metabolite that declines in aging – lactate – can cause specific changes to the structure and function of mitochondria of the RPE. The mitochondria are the energy factories of the cell, and deterioration of these factories is known to occur as AMD worsens. This past year Dr. Wu learned that lactate regulation of mitochondria is more complex than previously imagined, as what lactate does can change depending on what other metabolites are present. In trying to understand what metabolites are present and absent, the team also learned that the retinas of mice that are missing lactate also end up having fewer lipids than normal, an important building block for retinal cells.

Basic Research Projects Receiving Initial Funding in 2024



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 University of Texas Health
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**Novel Human Oguichi Disease
 Mechanisms**

Oguchi disease as first reported by Dr. Chuta Oguchi in 1979, results in affected individuals who are night blind with a diagnostic shiny metallic fundus appearance that turns normal following prolonged dark adaptation, aka the Mizuo-Nakamura phenomenon. Named after Dr. Oguchi, this disease is an autosomal recessive disorder disabling the arrestin (*Arr1*) and the rhodopsin kinase (*Grk1*) genes, which are required for timely deactivation of visual pigments in rod and cone photoreceptors. Dr. Chen modeled this disease in the 1990s in the *Grk1*^{-/-} mice and reported a previously unknown light-dependent rod degeneration phenotype⁵, which was later confirmed and found in human patients. This project is built upon Dr. Chen's experience with the *Grk1* gene⁵ and the not fully understood rhodopsin kinase (GRK1)

it encodes to systematically investigate thirteen disease-causing missense mutations found in this gene with the hope to gain insights on its activation mechanism, as well as settling a long-standing mystery concerning the mechanism of reproducibility of rod's single photon response (SPR). Dr. Chen hypothesizes that some *Grk1* missense mutations hamper the activation of GRK1 and reduce its ability to compete with transducin resulting in enlarged and varied rod SPR. The conclusion of this proof-of-principle study will provide compelling preliminary data to enable an NIH RO1 grant application for systemic screening of the rest twelve *Grk1* missense mutations for additional disease mechanisms and/or for identifying mutations affecting GRK1 activation. The information to be obtained will significantly impact the genetically heterogeneous retinitis pigmentosa (RP) field where the rhodopsin gene, GRK1's sole substrate, harbors the most disease-causing mutations⁶⁹. There is no cure yet for rhodopsin-associated RP due to insufficient basic information concerning how it is deactivated and when not, how prolonged rhodopsin signaling kills rod cells. Dr. Chen's study will thus provide the missing piece of information toward a full understanding of GRK1's activation process, an indispensable step toward developing new therapeutic modalities to treat rhodopsin-related and other hereditary human blinding diseases.



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**Analyzing the Functional
 Role of Calyceal Processes
 in CDHR1-Dependent
 Cone-Rod Dystrophy**

This project seeks to enhance the understanding of the molecular mechanisms underlying cone-rod dystrophy (CRD), particularly in relation to the loss of CDHR1 function. CDHR1 is a specialized protein found in the retina and has been linked to CRD in many human patients. It is believed that CDHR1 plays a role in connecting newly forming rod

outer segment discs to the inner segment, influencing their maturation and release. However, researchers still don't fully understand how the loss of CDHR1 leads to CRD, especially in relation to cone photoreceptors and how they are affected.

Dr. Famulski made a novel discovery in his efforts to decipher CDHR1's role in zebrafish cones. His lab found that CDHR1 may form connections between photoreceptor outer segments and inner membrane extensions called calyceal processes by physically interacting with a specific cadherin called *Pcdh15b*. Based on this finding, he is testing the hypothesis that these connections are essential for maintaining and assembling rod and cone outer segments, and that their disruption leads to CRD. To test their hypothesis, the team created a zebrafish model with a non-functional *cdhr1a* gene, which exhibits key features of CRD, including early and severe degeneration of cone photoreceptors and delayed degeneration of rods.



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Ann Arbor, MI

Control of Neurogenesis During Retinal Development

The irreversible loss of retinal neurons underlies the pathology of many blinding diseases, such as macular degeneration, retinitis pigmentosa and glaucoma. Effective cell-based therapies that can replenish these cells via cell transplantation or endogenous reprogramming are needed. It is crucial to gain a deep understanding of the gene regulatory networks and mechanisms that control the generation of different retinal

cell types. Over the course of retinal development, retinal progenitors progressively generate diverse types of retinal neurons and Müller glia; however, the molecular mechanisms that control this process remain largely unclear. Dr. Hoang is examining the effect of both loss-and gain-of-function of *Insm1* on retinal development as well as determining the mechanism of how *Insm1* regulates the process. *Insm1* is believed to act as a key transcription factor that controls the generation of diverse neurons from retinal progenitors during retinal development. His study will enhance the current understanding of how different retinal cell types are produced by identifying the factors and mechanisms controlling neurogenesis during development, as well as help guide cell-based therapies aimed at replacing retinal neurons lost in retinal degenerative diseases via exogenous cell transplantation or endogenous reprogramming of resident Müller glia cells.



Eleftherios Paschalis Ilios, PhD
Department of Ophthalmology
Massachusetts Eye and
Ear Infirmary
Harvard Medical School
Boston, MA

Evaluation of a Novel Bi- Specific Antibody for AMD in Non-Human Primate: A Step Before Transitioning to Clinical Trials

Dr. Paschalis' objective is to develop a new therapy for Age-related macular degeneration (AMD), the leading cause of central vision loss in people over 50. The standard of care for the treatment of wet AMD is frequent intravitreal injections of antibodies against the vascular endothelial growth factor (VEGF), and while these injections check the

development of new retinal vessels, because they require life-long administration, they also gradually cause progressive neuroretinal atrophy that can lead to blindness. Fortunately, more recently bi-specific antibodies that can target two pathways involved in the progression of the disease, such as VEGF and Ang2, were approved by FDA as an alternative therapy for AMD, with clinical data suggesting that the new therapy provides marginal improvement in patients.

Dr. Paschalis has discovered two pathways that cause neovascularization, which if blocked simultaneously, can result in complete inhibition of development of new vessels (neovascularization), with results that are significantly better compared to preclinical data produced by existing FDA approved therapies for AMD. According to Dr. Paschalis' pre-clinical data, the therapy has the potential to become a valuable alternative therapy for humans, and Dr. Paschalis' project objective is to obtain the data necessary to proceed to human phase I clinical trials for this new drug therapy.



Georgia Zarkada, MD, PhD
Department of Physiology and
Neurobiology
University of Connecticut
Storrs, CT

Modulation of Retinal Vascularization by Endothelial Cell Genetic Reprogramming

Abnormal formation of new blood vessels is the fundamental cause of catastrophic vision loss during ocular neovascularization. Current therapies target destroying aberrant vascularization by neutralizing Vascular Endothelial Growth Factor-A (VEGF). Yet, these approaches do not address the causes that underlie vascular pathology, such as hypoxia and inflammation.

Dr. Zarkada's project aims to identify VEGF-independent signaling pathways that could promote vascular repair and regeneration of the diseased eye. One pathway that holds promise is Transforming Growth Factor (TGF) β and its receptor (R) 1 (TGFBR1), which are specifically required during developmental vascular growth in the neuroretinal, responsible for processing light into neural signals.

Dr. Zarkada hypothesizes that activation of TGF β /TGFBR1 signaling could force sprouting endothelial cells to acquire the abilities to form a leak-proof and properly patterned vasculature in the neuroretina. Her lab will generate a new genetic mouse model to test this hypothesis during developmental retinal vascularization and project will provide proof or principle that reprogramming endothelial cell identity could be a successful alternative approach to treat neovascular eye disease. The project will yield key insights into TGF β biology during retinal vascularization.

The RRF Margaret and Mills Cox Macula Society Research Project



Leo Kim, MD, PhD
Schepens Eye Research Institute
Massachusetts Eye and Ear
Infirmery
Harvard Medical School
Boston, MA

Inhibition of Topoisomerase 1 for the Treatment of Proliferative Vitreoretinopathy

Dr. Kim is an Associate Professor at Harvard Medical School, serving on the Retina Service at Mass Eye and Ear, and holds the Monte J. Wallace Ophthalmology Chair in Retina.

Dr. Kim has published over 95 peer-reviewed publications. His work is primarily focused on pathologic retinal angiogenesis and fibrosis, with the use of patient-derived surgically dissected samples to elucidate new molecular mechanisms of disease. His research has been funded by numerous foundation grants, the Department of Defense, as well as grants from the NIH / NEI grants through R21 and R01 mechanisms. As a scientist, Dr. Kim has trained over 20 undergraduate students, graduate students, medical students, postdoctoral fellows, and research associates in his laboratory. As a clinician, he has trained over 30 vitreoretinal fellows in the medical and surgical management of vitreoretinal disease.

Notably, Dr. Kim discovered the role of RUNX1 in pathologic retinal angiogenesis using patient-derived tissues, furthering the understanding of ocular angiogenesis beyond vascular endothelial growth factor (VEGF), by using fibrovascular membranes from patients with proliferative diabetic retinopathy. He has illustrated the role of RUNX1 in proliferative diabetic retinopathy, and expanded it to

include choroidal neovascularization as found in exudative age-related macular degeneration. This work was published in *Diabetes*, *the American Journal of Pathology*, and *FASEB Journal*. These findings have implications beyond diseases of the retina, and are of interest to those investigating angiogenesis and RUNX1 gene mutations which cause bleeding disorders and an increased risk of hematologic malignancies like leukemia.

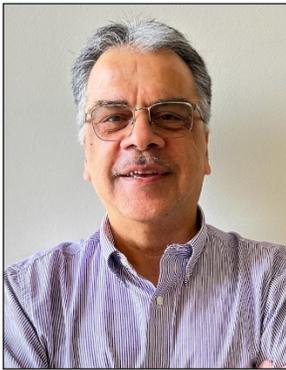
His other significant area of impact is in proliferative vitreoretinopathy (PVR), a leading cause of recurrent retinal detachments, characterized by aberrant fibrosis within the eye. PVR is one of the most vexing problems that vitreoretinal surgeons must face. Dr. Kim has developed three new models based on patient-derived PVR membranes. First, he created a primary PVR cell culture line, which he has called the C-PVR cell line to investigate the mechanism of PVR, as well as a drug screening tool. Next, he has developed an in vivo rabbit model of PVR via injection of C-PVR cells within the vitreous of rabbits to recapitulate the clinical findings of PVR as found in patients. Finally, he developed an explant model of PVR, directly taking PVR membranes samples from the operating room into an extracellular matrix in vitro to directly evaluate the efficacy of therapies for PVR. Using these patient-derived models he showed the effect of methotrexate on PVR, which was published in *Investigative Ophthalmology and Visual Science*. This work was the scientific basis for the recently completed phase 3 GUARD trial testing the efficacy of methotrexate for PVR. He then identified RUNX1 as an important mediator of epithelial-mesenchymal transition in PVR, which was published in *Nature Scientific Reports*. Beyond methotrexate and RUNX1 inhibition, he has found several new promising therapeutic modalities for PVR including rho kinase and topoisomerase inhibition. The results of Dr. Kim's funded project will be presented at the 2025 or 2026 Macula Society meeting.

Research Chairs and Professorships

The academic chairs and professorships that RRF supports are located at nationally recognized vision research institutions in Houston, Texas, and Madison, Wisconsin. Often in collaboration with fellow researchers in related academic disciplines, these scientists conduct their own original retina research, with the potential to dramatically increase the understanding of the retina and/or retinal diseases, while also supervising departmental programs that provide research opportunities for young vision scientists and students to pursue their interests and to receive academic training within established investigative laboratories. Support for these prestigious academic positions represents 16% of RRF's annual research budget.



RRF Research Chair



Nader Sheibani, PhD
Department of Ophthalmology
and Visual Sciences
University of Wisconsin
Madison, WI

Pathophysiology of Eye Diseases with Neovascularization

A significant portion of patients with exudative AMD do not respond to anti-VEGF treatment. Dr. Sheibani's research team showed that polymorphisms in two genes, extensively

studied in his laboratory, namely Thrombospondin-1 and Bim, along with complement factor H, could contribute to this lack of response in patients with exudative AMD. He also led a special topic section in *Frontiers in Ophthalmology* on recent developments and treatment of ocular fibrosis. Dr. Sheibani also showed a significant role for Bax expression in postnatal retinal vascular development and hyperoxia sensitivity. They also showed that Bim expression in choroidal endothelial cells has significant impact on their angioinflammatory properties and response to therapeutic interventions. They also demonstrated a significant role for RIPK3, a key regulator of necroptosis, in ocular vascular development and pathological neovascularization. Reported in: *PLoS One* (Feb 24), *Front Ophthalmol* (Jan 24), *Exp Eye Res* (Nov 24), *Int J Mol Sci* (Sep 24), *Cells* (Dec 24).

Walter H. Helmerich Chair



Kevin W. Eliceiri, PhD
Associate Director, McPherson
Eye Research Institute
Departments of Medical Physics
and Biomedical Engineering
University of Wisconsin
Madison, WI

Open Source Computational Imaging of Cellular Microenvironments

Dr. Eliceiri's research interests are in the areas of developing open source optical and computational approaches to noninvasively study dynamic cellular processes like those in

the eye. His current research focuses on the development of novel optical imaging methods and instrumentation for investigating the cellular microenvironment, and the development of open-source software for multidimensional imaging informatics. Specific interests include developing label free optical approaches for deeper imaging and sensing of the cellular microenvironment, new technologies for metabolic imaging, as well as technologies for multi-scale and multimodal imaging. Recently, his group has been collaborating with computer scientists on deep learning approaches for smart imaging of cellular metabolism. As well his group is very engaged in community building efforts such as BioImaging North America (BINA), which tries to build and bridge imaging expertise across Canada, Mexico and the U.S.

Emmett A. Humble Distinguished Directorship



David M. Gamm, MD, PhD
 Director, McPherson Eye
 Research Institute
 Professor, Department of
 Ophthalmology and
 Visual Sciences
 University of Wisconsin
 Madison, WI

Modeling and Treating Retinal Disease with Human Induced Pluripotent Stem Cells (hiPSCs)

Dr. Gamm employs hiPSC technology to generate retinal organoids, which he uses to model human retinal diseases

and develop gene- and cell-based therapies. In 2024, his lab worked to complete preclinical studies in support of an Investigational New Drug (IND) application for a first-in-human clinical trial to test iPS cell-derived photoreceptor precursors as a treatment for late-stage photoreceptor degenerative diseases. The IND was approved by the FDA, which paved the way for the initiation of a phase 1/2a clinical trial in 2025. The trial (CLARICO) is being conducted by BlueRock Therapeutics, a subsidiary of Bayer AG, who licensed the cell therapy from Ophis Therapeutics, a subsidiary of Fujifilm Cellular Dynamics that Dr. Gamm co-founded. In addition, Dr. Gamm and his UW-Madison colleagues have employed patient-specific and gene-edited iPSC-RPE cultures to advance base and genome editing strategies to treat Leber congenital amaurosis and macular degenerative diseases.

Kathryn and Latimer Murfee Chair

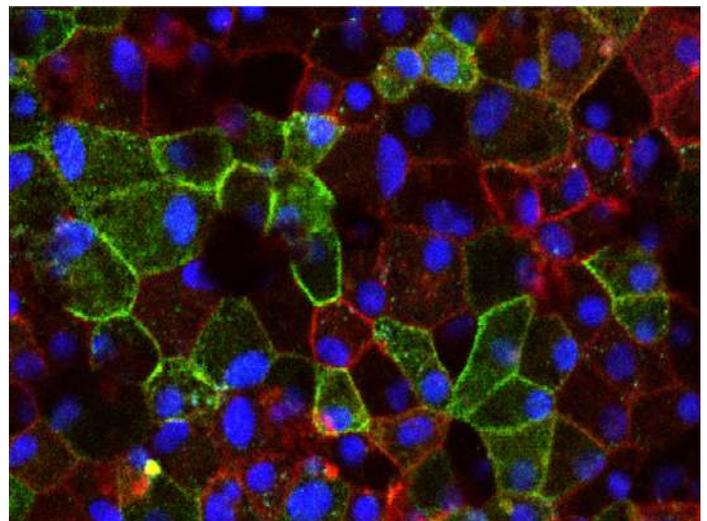


Krishanu Saha, PhD
 McPherson Eye Research Institute
 Departments of Biomedical
 Engineering and Pediatrics
 Wisconsin Institute
 for Discovery
 University of Wisconsin
 Madison, WI

Bioengineering of Novel Cell and Gene Therapies for the Retinal Disorders

Over the past year, Dr. Saha’s lab has achieved notable progress in creating innovative CRISPR-based treatments for inherited retinal disorders. His team successfully met with the FDA to discuss an investigational new drug application that uses a non-viral nanoparticle approach to fix a mutation responsible for Leber Congenital Amaurosis. The team has also developed advanced genome engineering and analysis methods, enabling a platform approach, whereby CRISPR therapies are tailored to target different mutations causing inherited retinal diseases. This ongoing work involves partnerships with the FDA, industry, and the NIH Somatic Cell Genome Editing Consortium. Their discoveries, published in prestigious journals and showcased

at conferences, bring us closer to effective therapies for genetic disease.



Human model of Leber Congenital Amaurosis treated with CRISPR nanoparticles. Green marks the rescue of protein lost by the mutation (Kir7.1 channel arising from the KCNJ13 gene). Blue marks the cell nuclei, while green labels a different channel on the surface of these cells (Na K ATPase).

Research Chairs and Professorships

Daniel M. Albert Chair



Bikash R. Pattnaik, PhD
McPherson Eye Research
Institute
Department of Pediatrics,
Ophthalmology and Visual
Sciences
University of Wisconsin
Madison, WI

Treatment for Rare Diseases That Challenge Healthcare

The mission of Dr. Pattnaik's research laboratory is to bring precision genomic medicine closer to clinical application for rare and currently untreatable causes of childhood blindness.

Dr. Pattnaik's research group continues to advance translational research in retinal and inherited eye diseases, with a strong focus on channelopathies and gene-based therapies. The group's overall approach is to develop gene-specific, mutation-specific, and generalized treatments, and they have made progress in gene editing, gene therapy, engineered tRNA, and drug-mediated read-through.

Keeping the testing scheme common to use both a patient-derived iPSC-RPE lines and a mouse model, Dr. Pattnaik used base editing to specifically edit the mutant nucleotide (a single base change that causes blindness). Over the past year, his team made significant strides in the development of a gene-agnostic engineered tRNA therapy to correct nonsense mutations in *KCNJ13*, a gene implicated in Leber Congenital Amaurosis (LCA16). These studies demonstrate both in vitro rescue of ion channel function and in vivo preclinical efficacy.

Dr. Pattnaik's research group also developed new disease-relevant iPSC-RPE and mouse models, enabling the validation of novel therapeutic strategies, including gene-agnostic approaches that hold promise for treating a broader range of inherited retinal disorders. They are furthering their therapeutic efforts for Best disease and Congenital Stationary Night Blindness to correct the function of chloride and calcium channels, respectively. The work has resulted in multiple peer-reviewed publications, conference presentations, and the training of students at all academic levels, furthering the mission of the Daniel Albert Chair in nurturing the next generation of clinician-scientists.

Edwin and Dorothy Gamewell Professor

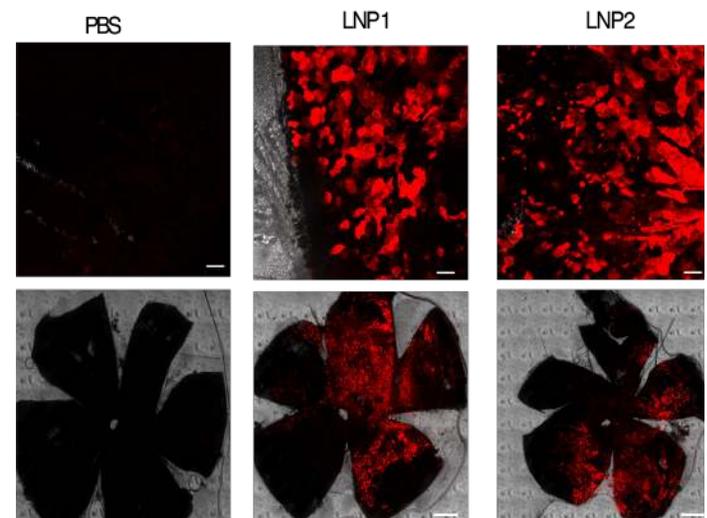


Shaoqin "Sarah" Gong, PhD
McPherson Eye Research
Institute
Department of Ophthalmology
and Visual Sciences
Wisconsin Institute for
Discovery
University of Wisconsin
Madison, WI

Ocular Gene and Cell Therapy

In 2024, Dr. Gong's laboratory continued its focus on innovative biomaterials for biomedical applications, emphasizing ocular gene and cell therapy. Collaborating with several McPherson Eye Institute labs, significant progress was made on two key projects: the photoreceptor/retinal epithelium cell scaffold and ocular CRISPR genome editing. A major milestone was achieved with the first INTERACT meeting with the FDA for the ocular CRISPR genome editing project, marking a crucial step towards developing an IND-enabling product for genetic ocular diseases. These advancements underscore Dr. Gong's lab's commitment to translating biomaterial research into potential ocular therapies. The lab's broader impact in nanomedicine was evidenced by a *Nature Nanotechnology* publication titled "Multimodal nanoimmunotherapy engages neutrophils to

eliminate *Staphylococcus aureus* infections," highlighting their continued contributions to cutting-edge research.



Retinal epithelium cell floret in Ai14 transgenic mice: CRISPR genome editors delivered via subretinal injection of lipid nanoparticles (LNPs) resulted in robust tdTomato expression, indicating efficient delivery and genome editing. Scale bars: 50 μm (top panel); 500 μm (bottom panel).

Data collection in collaboration with Dr. Bikash Pattnaik's laboratory.

M.D. Matthews Research Professor



Timothy M. Gomez, PhD
 McPherson Eye Research
 Institute
 Department of Neuroscience
 University of Wisconsin
 Madison, WI

**Validating Expression
 Changes of Motility-associated
 Proteins in hiPSC-derived
 Photoreceptors”**

Cell replacement therapies are one of the best options for multifactorial diseases of the retina like age-related macular degeneration (AMD), which normally cannot be treated by correcting mutations in individual genes. The retina is also highly surgically accessible, allowing stem cell derived neurons to be precisely transplanted and tracked post-operatively. While retinal pigment epithelia (RPE) and photoreceptor (PR) transplant therapies are ongoing, including Phase I human trials, success has been limited due to the inability of PRs to integrate properly into complex existing circuits. One potential means to improve PR integration is to transplant genetically modified PRs that have enhanced ability to extend processes and form

synapses. We recently found that human stem cell derived PRs have a limited time they are capable of autonomous axon extension, which correlated with a loss of an organized actin cytoskeleton within their terminals and an upregulation of synaptic proteins (Rempel et al, 2022, Cell Reports). Specifically, we found that PRs became immobile between 40 days of differentiation (d40) and 80 days. This rapid loss of PR motility may be due to their decreased ability to polymerize and organize actin, which is essential for cell motility and axon extension. Therefore, identifying age-related molecular changes that account for the loss of actin filaments and decreased PR terminal motility would provide potential targets to potentiate intrinsic PR terminal motility to support regeneration.

During the initial year of his Matthews professorship, Madison Dillerud, a 2nd year MCP graduate student in the Gomez lab, has been mining transcriptomic data generated by the Gamm lab to verify gene expression changes of motility-associated proteins in human stem cell derived PRs. Validated genes that are reproducibly down-regulated in aged PRs will be further tested using several functional assays in future gain of function experiments. The Gomez laboratory is also ramping up retinal organoid production for future proteomic analysis of expression changes between d40 and d80.

Rebecca Meyer Brown Professor



Mrinalini Hoon, PhD
 McPherson Eye Research
 Institute
 Department of Ophthalmology
 and Visual Sciences
 University of Wisconsin
 Madison, WI

**How loss of input impacts
 neuronal pathways regulating
 retinal output**

Two parallel and inter-connected neural pathways in the mammalian retina, ON and OFF that encode light increments and decrements respectively, regulate visual information processing. Diseases such as congenital stationary night blindness (CSNB) involve suppression

of ON pathway retinal input. Using genetic murine models of ON pathway suppression that mimic CSNB, and by combining 3D light microscopy with single cell electrophysiology, the Hoon lab is determining how the structure and function of retinal output neurons (ganglion cells) is impacted in CSNB unveiling the cellular mechanisms of dysfunction. The lab's studies are identifying a permissive level of ON pathway input needed to maintain the functional profiles of retinal output neurons with adaptive measures being recruited by output neurons to compensate for the imbalanced input. Complete input suppression, however, severely disrupts the neuronal stability of ganglion cells with different impairments manifested across ganglion cell types. The studies are thus revealing the neuronal mechanisms underlying disease conditions such as CSNB uncovering new targets for therapeutic interventions.

RRF Research Chair at Baylor College of Medicine, Houston, TX

Following the departure of vision researcher, Ching-Kang Jason Chen, PhD, at the end of 2022, a nationwide search for a candidate to hold the RRF Research Chair at Baylor College of Medicine is being conducted and was still ongoing near the end of 2024. Dr. Chen was the first recipient of the chair and held the position from 2013 to 2022.

Established Research Awards

Representing 11% of RRF's research budget, established research awards are presented to renowned physician scientists in recognition of their lifetime achievement. RRF's mission is global in scope, and in 2024, five international awards were given. 2024 was a unique year in that for the first time, a single physician/scientist received three RRF awards in recognition of research contributions in the same year.



Anat Loewenstein, MD, MHA
Tel Aviv Medical Center, Tel Aviv, Israel

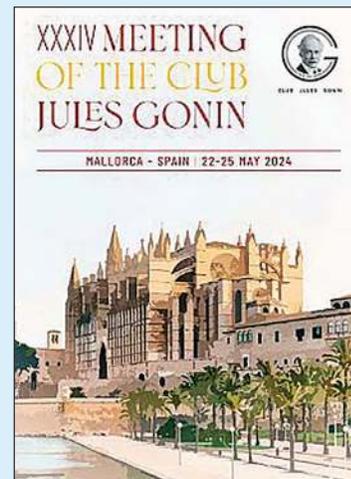
Anat Loewenstein, MD was awarded three RRF established research awards by her global peers from within the prestigious ophthalmological societies: The Gonin Lecturer, The Pyron Award, and The Award of Merit. She spoke at all three society meetings on the new opportunities arising from imaging technology advancements enabling home monitoring and longer acting treatments, technologies that will usher in a new era in retinal disease management.



Club Jules Gonin

The Jules Gonin Lecturer of the Retina Research Foundation

Every two years, the members of Club Jules Gonin select a Gonin Lecturer to present at their biennial meeting. The award recipient is chosen for making a significant contribution to the understanding and treatment of eye diseases. Dr. Loewenstein presented the Jules Gonin Lecture at XXXIVth Meeting of the Club Jules Gonin, Mallorca, Spain, on May 24, 2024, sharing her research related to home-OTC monitoring for AMD.



ASRS American Society of Retina Specialists

RRF Pyron Award for Outstanding Achievement in Retina Research

The RRF Pyron Award, given by the American Society of Retina Specialists (ASRS) was created by RRF to recognize outstanding vision scientists whose work contributes to knowledge about vitreoretinal disease. Dr. Anat Loewenstein was presented with the Pyron Award at the ASRS annual meeting held in Stockholm Sweden on July 17, 2024.





The Award of Merit in Retina Research

The Award of Merit in Retina Research was established in 1978 by RRF to recognize outstanding vision scientists whose research contributes significantly to new knowledge about the retina and retinal diseases or disorders. As the 2024 recipient of the award, Dr. Anat Loewenstein gave the Charles L. Schepens Lecture at the Retina Society's 57th Annual Scientific Meeting held in New York City on October 12, 2024.



Anat Loewenstein, MD, MHA, serves as Professor and Director of the Division of Ophthalmology at Tel Aviv Medical Center and is also the Vice President of Ambulatory Services at Tel Aviv University. She holds the prestigious Sidney Fox Chair of Ophthalmology at the Sackler Faculty of Medicine at Tel Aviv University.

Dr. Loewenstein's primary research interests focus on drug administration and toxicity related to the retina, the early detection of macular degeneration, and the home monitoring of retinal diseases. She leads several groundbreaking technologies, including the development and implementation of large-scale clinical trials and innovations in clinical settings. Notably, she pioneered the Home OCT, the first-of-its-kind artificial intelligence-enabled algorithm designed for monitoring retinal diseases using an OCT device at the patient's home. Additionally, she has been instrumental in developing technology that integrates virtual reality into vitreoretinal surgery, aiming to replace the standard operating microscope.

Dr. Loewenstein has published more than 500 papers in peer reviewed journals, and contributed multiple chapters to

ophthalmology textbooks. She currently serves as the Editor in Chief of the Journal Case Reports in Ophthalmology, is an associate editor of Investigative Ophthalmology & Visual Science Journal, European Journal of Ophthalmology, and of Ophthalmologica.

As a leader in the field of retinal disease, Dr. Loewenstein serves in numerous national and international roles, including as a member of the National Council of Surgery, the Israeli Academy of Medicine, and the Academia Ophthalmologica Internationalis. She leads mentorship groups for ARVO, and Euretina. She is currently the President of the Israeli Ophthalmological Society and President Elect of Euretina. She currently serves on the Board of NotalVision and Pulsenmore companies as well as on the board of ESASO (European School for Advanced Studies in Ophthalmology) and previously was a board member at Given Imaging. In addition to her service on advisory boards of a majority of the world's largest pharmaceutical companies, she leads industry sponsored programs.

Established Research Awards

Paul Kayser International Award



Hendrik P. N. Scholl, MD, MA, PhD
Medical University of Vienna,
Austria

Lecture: Therapy development for
inherited macular degeneration

Made possible by a gift to RRF in honor of Paul Kayser, the RRF Paul Kayser International Award in Retina Research was first presented in 1986. This international award recognizes lifetime achievement by a vision scientist who has made a significant contribution to the understanding of vitreoretinal diseases or disorders. This \$50,000 award is given every two years.

Prof. Scholl received his award and gave his lecture at the XXVI Biennial Meeting of the International Society for Eye Research (ISER), in Buenos Aires, Argentina in October 2024.

Prof. Scholl is Adjunct Professor in the Department of Clinical Pharmacology, Medical University of Vienna, Austria, and Chief Medical Officer, Belite Bio in Zug, Switzerland. Hendrik Scholl is one of the two founding directors of the Institute of Molecular and Clinical Ophthalmology Basel (IOB). He was

previously a Professor and past Chairman of the Department of Ophthalmology, University of Basel, Switzerland. He specializes in the treatment of retinal diseases, including age-related macular degeneration and inherited retinal and macular dystrophies such as Stargardt disease.

Prof. Scholl is a graduate of the Medical Faculty of the University of Tübingen/Germany. After having held several academic positions at the Medical Faculty of the University of Bonn, Germany, he was appointed as Professor of Ophthalmology in 2010 at the Wilmer Eye Institute, Johns Hopkins University, in Baltimore, United States. At the Johns Hopkins Hospital, he headed the Retinal Degeneration Clinic and the Visual Neurophysiology Service.

Prof. Scholl has authored over 280 articles and reviews in peer-reviewed journals and received numerous prestigious awards, including the European Vision Award, the President's Award of the American Society of Retinal Specialists, the W. Richard Green Award and the Paul Henkind Memorial Award of the Macula Society, the Swiss Alfred-Vogt Award, and the Kupfer award of the Association for Research in Vision and Ophthalmology (ARVO). He holds an honorary doctorate from Semmelweis University in Budapest, Hungary. Prof. Scholl participates and/or chairs several Data Monitoring Committees of large clinical trials and has further leadership roles in ophthalmology such as President of the European Vision Institute (EVI) and Chairman of the European Vision Clinical Research Network (EVICR.net).



Buenos Aires, Argentina



International Society
for Eye Research

Charles L. Schepens, MD/AAO Award



Steven T. Charles, MD
Charles Retina Institute
Germantown, TN

Schepens Lecture: Systems Engineering at the Intersection of Technology and Technique

The 2024 RRF Charles L. Schepens, MD/AAO Award was given to Dr. Steven T. Charles. He delivered the Charles L. Schepens lecture during the morning session of the Retina Sub-specialty Day at AAO's annual meeting on October 18, 2024 in Chicago, IL.

Dr. Charles has developed many of the techniques and devices used by vitreoretinal surgeons worldwide. He has performed over 46,000 vitreoretinal surgeries, lectured in 51 countries and operated in 25, delivered over 25 named lectures, and over 2000 speaking trips. He authored a leading textbook in the field which is now in the 6th edition and authored over 200 articles in the medical literature and over 50 book chapters. Dr. Charles is also a mechanical and electrical engineer and has over 200 issued or pending patents. He is the Founder of MicroDexterity Systems, which developed robots for dexterity enhancement for minimally invasive knee and hip replacement, spine surgery, and skull base neurosurgery;



Dr. Steve Charles and Dr. Donald D'Amico

cofounder, systems engineer, and Chairman of CamPlex Inc, developing advanced visualization technology for MIS spine, neurosurgery and trans-oral approaches to head and neck cancer; and a consultant for Alcon Laboratories and the principal architect of the Alcon Accurus and Alcon Constellation Vision System, advanced vitreoretinal surgical instrumentation.

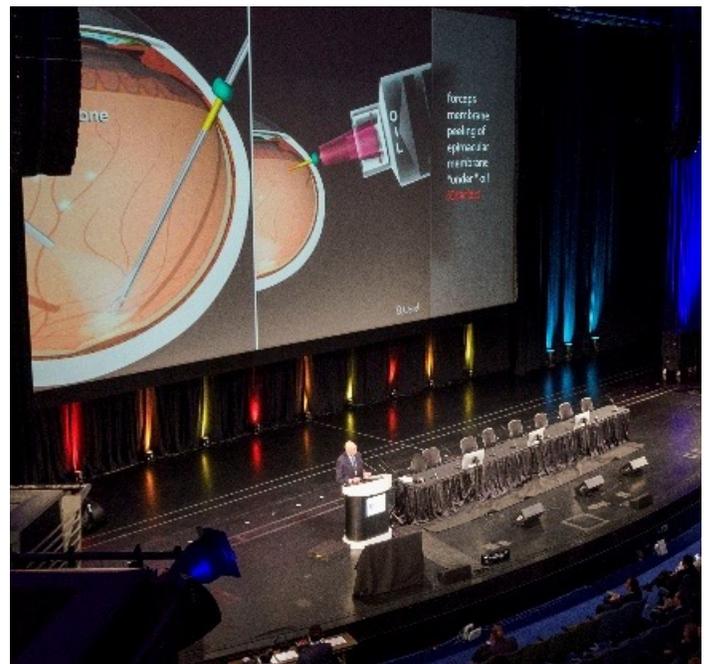
Dr. Charles is the founder and CEO of the Charles Retina Institute located in Germantown, a suburb of Memphis, TN. The Institute has 11 clinic locations throughout Tennessee, Mississippi and Arkansas, and employs 11 retina specialists and fellows. Dr. Charles is affiliated with the University of Tennessee where he is a Clinical Professor of Ophthalmology. Active in his professional societies, Dr. Charles is a Fellow in the American Society of Retinal Specialists, American College of Surgeons and International College of Surgeons

and a member of the Retina Society, Macular Society, American Society of Retinal Specialists, Club Jules Gonin, American Society of Cataract and Refractive Surgery, American Academy of Ophthalmology, and the Dowling Society. He served on the Board of Governors of the ARVO Foundation for Eye Research. He has received the Laurate honor from the American Academy of Ophthalmology, gave the Schepens lecture at the Retina Society and the Kelman



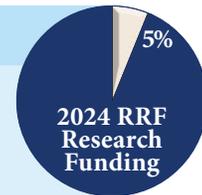
Dr. Steve McLeod, AAO CEO, Dr Steve Charles

lecture at the ASCRS, received the Wacker Medal from the Club Jules Gonin, the first Founders Medal from the Vitreous Society, was inducted into the University of Miami School of Medicine Medical Alumni Association Hall of Fame, and was named by Ocular Surgery News as one of the top ten innovators in the past 25 years. He won the Lifetime Achievement Award from Clinical Trials at the Summit. He is consistently listed in Best Doctors in America, Becker's Top 34 Ophthalmologists in America, and Newsweek's America's Best Ophthalmologists lists.



International Fellowships

The International Fellowship Program, representing five percent of the RRF annual research budget, funds two programs: a 12-month fellowship and a six-month fellowship.



RRF Helmerich International Fellowships

Since 2009, RRF has offered two international fellowships with income from an endowment created by Walter H. Helmerich, III and his family. The 12-month fellowships provide advanced subspecialty training for young ophthalmologists from developing countries who are recommended by the head of a teaching or public service institution and are committed to returning to a position at a teaching institution or public service hospital in their home country following the fellowship.

During 2024, two physicians received training as Helmerich International Fellows. The application process for these fellowships is highly competitive, and the selected candidates have ambitious goals regarding what they hope to gain in terms of specific enhanced skills and expertise they hope to offer to patients and their fellow ophthalmologists upon return to their home countries.



Dr. Aim-on Saengsiravin from Bangkok, Thailand was awarded an IOFF-RRF Helmerich Fellowship in Uveitis at the Byers Eye Institute at Stanford University under the guidance of Prof. Quan Dong Nguyen.

Dr. Saengsiravin participated in clinical activities during her fellowship, typically consulting on 15-20 individual patients visits each day. These visits gave her experience in the complex

field of uveitis management in taking patient histories, performing eye examinations, reviewing laboratory results and imaging, followed by a detailed discussion with her mentor, Prof. Nguyen. The diversity of patients in the Bay Area, combined with Stanford's role as a tertiary center, exposed Dr. Saengsiravin to a wide range of infectious and non-infectious uveitis cases, including conditions rarely seen in Thailand, such as White Dot syndromes, Blau's syndrome with ocular involvement, S-cone syndrome with retinal vasculitis, autoimmune retinopathy, and cancer associated retinopathy. The management of uveitis at Stanford follows a step-ladder approach, starting with systemic steroids and progressing through steroid-sparing agents, biologics, and other therapies. For complex cases where conventional treatments are insufficient, she learned about various

clinical trials, including JAK inhibitors, IL-17 inhibitors, IL-6 inhibitors, and ACTH.

Dr. Saengsiravin was actively involved in several research projects and clinical trials. Stanford's role as a referral center means patients are seeking not just second opinions but even sixth or seventh opinions. Patients are highly informed about their conditions and ask in-depth questions about prognosis and treatment modalities. The clinical trials (being conducted at Stanford), offer the unique option of investigational drugs for patients, making Stanford an exceptional institute for treating uveitis patients.

Throughout her fellowship, Dr. Saengsiravin participated in organizing and presenting at various conferences, including presenting a research poster at ARVO, 2024; and attending BAOC (Bay Area Ophthalmology Course) 2024, where she delivered a talk on ocular tuberculosis and management in Uveitis session. BAOC is renowned for providing comprehensive lectures and wet labs to ophthalmology residents worldwide. She writes, "My lecture on unique cases of ocular tuberculosis and their management was well-received, and it was an honor to contribute to this esteemed course. It was a dream come true to join BAOC not as a participant but as a lecturer, sharing my knowledge and experiences with the next generation of ophthalmologists."

Upon completion of her fellowship, Dr. Saengsiravin shared, "Being at Stanford has been a transformative experience, both professionally and personally. The opportunity to work with world-class professionals and learn from a diverse group of colleagues has been invaluable. The collaborative and inclusive environment fostered by Professor Nguyen has allowed me to grow as a clinician and researcher. My time at Stanford has been enriching and fulfilling, allowing me to develop new skills, contribute to impactful research, and form lasting professional relationships. I look forward to applying this knowledge and experience to advance ophthalmic care in Thailand and beyond. This fellowship has not only furthered my professional development but has also allowed me to make meaningful contributions to the field of ophthalmology. I look forward to continuing my work and applying the knowledge and experience gained here to advance ophthalmic care in Thailand and beyond."



Dr. Saengsiravin (front, far right), her mentor, Prof. Nguyen (center, with bowtie) and Stanford colleagues.



Dr. Lily Myint, from Burma (Myanmar) was awarded a one-year IOFF-RRF Helmerich fellowship in Strabismus and Pediatric Ophthalmology at LV Prasad Eye Institute in Hyderabad, India. Dr. Myint completed an intensive two-week training, including virtual training, to provide her foundational skills and knowledge crucial for her specialty. Her rotations within the

Pediatric, Strabismus, and Neuro Ophthalmology departments provided her with hands-on patient experience, further enhancing her understanding of these specialized fields.

Dr. Myint shared, “I extend my heartfelt thanks to you for believing in my potential and contributing to my professional development. Your sponsorship is not only assisting me in achieving my career goals but also making a positive impact on patient care in the field of ophthalmology in my home country.”



International Fellowships



Gillingham Pan-American Fellowships

A collaboration with the Pan-American Association of Ophthalmology (PAAO), the RRF Gillingham Fellowships program offers two, six-month fellowships to Latin American ophthalmologists for training at leading institutions in the United States or Canada.

Initiated in 1993 by RRF's founder, Dr. Alice R. McPherson, and made possible through the legacy of W.J. Gillingham, the fellowship program aims to nurture the next generation of ophthalmic leaders. Annually, deserving Latin American

scholars who have secured a position in an accredited training program in the U.S. or Canada, are awarded these fellowships. This collaboration has provided 60 Gillingham fellowships over the last 31 years, to aid recipient ophthalmologists in their quest to receive additional medical specialty training and improve patient care in their home countries.

Following the increase in the amount of annual scholarship monies to \$20,000 for each recipient in 2024, PAAO awarded three fellowships.



Dr. Carolina L. Mercado
(Colombia)

Medical Retina at Bascom
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Miami, Florida



Dr. Nancy Paola Arias González
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Pediatric Retina and ROP at
Bascom Palmer Eye Institute,
Miami, Florida



Dr. Y. Marcela Huertas Bello
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Cornea, Anterior Segment, and Refractive
Surgery at Toronto Western Hospital,
Toronto, Canada



Research Initiatives

Stewardship of endowed gifts enables RRF to generously fund educational programs, which disseminate research laboratory knowledge to practicing ophthalmologists and vision scientists worldwide. These efforts comprise three percent of RRF's annual research funding.

American Academy of Ophthalmology Educational Trust Fund

RRF collaborates with the American Academy of Ophthalmology (AAO) to provide ophthalmologists with high-quality educational resources needed to enhance their clinical research skills in the field of retina, and empowers them with knowledge of the latest advancements necessary to treat patients more effectively. RRF provides \$50,000 annually to this educational effort in support of the development of physician CME credit learning materials designed to meet the learning needs and interests for the retina subspecialist audience, comprehensive ophthalmologists, and trainees. These materials are utilized by members of the worldwide ophthalmology community, throughout all stages of their careers.

RRF Lawrence Travel Scholarships

The Lawrence Travel Scholarships program, funded through a bequest from Joe M. and Eula C. Lawrence, provides an opportunity for young researchers to attend the annual meeting of the Association for Research in Vision and Ophthalmology (ARVO). Receiving a travel scholarship opens the door to the world's premier gathering for eye and vision scientists at all career stages. It is an opportunity to share the latest research findings and collaborate on innovative solutions. The 2024 meeting theme highlighted how vision research is continually being transformed by new information and technologies, driven by expanding computing power enabled AI-applications and big data computation. Sophisticated molecular techniques and imaging modalities coupled with gene therapy breakthroughs are transforming the research pursuing new therapeutic means to prevent and cure blinding eye diseases.

Approximately 11,600 scientists attended the 2024 ARVO meeting in Seattle WA, in May, including RRF's sponsored 27 travel grant recipients. These young scientists, from research institutions across the country, participated in poster and abstract presentations, with their research eligible for publication in ARVO's journal *Investigative Ophthalmology & Visual Science (IVOS)*.



**ARVO Foundation/Retina Research Foundation/
Joseph M. and Eula C. Lawrence
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Retina Research Foundation 2024 Luncheon

The RRF community of leaders, scientists, supporters and friends gathered on May 11th for the 2024 RRF Luncheon and Honorary Lecture, providing an opportunity to celebrate RRF's achievements in vision preservation since its founding in 1969, and to educate attendees about new advances in vision science and academics.



Dr. David Gamm
Honorary Lecturer

Extending the long-held tradition of inviting speakers who are outstanding leaders of the academic and medical community, RRF's honorary speaker was Dr. David Gamm, Emmett A. Humble Distinguished Director of the McPherson Eye Research Institute (McPherson ERI) at the University of Wisconsin, Madison. RRF was privileged to have Dr. Gamm as its speaker at the luncheon, which was the first following the death of RRF's founder, Dr. Alice McPherson. The University

of Wisconsin was Dr. McPherson's alma mater for both her baccalaureate and medical school degrees, and she completed her ophthalmology residency at Madison as well. With Dr. Dan Albert, Director Emeritus, Dr. McPherson founded the school's Eye Research Institute, and the Institute was renamed in her honor in 2012. She was actively involved with the Institute's development and served on its Advisory Board until her death. Dr. McPherson became a mentor and friend to Dr. Gamm, encouraging his research interests from his earliest days at UW, and before he became the director of the Institute.

Dr. David Gamm's lab is at the forefront in developing cell-based therapies to combat retinal degenerative diseases, and Dr. Gamm specifically shared updates on his work using human induced pluripotent stem cells to advance gene therapies for inherited retinal diseases, such as Leber Congenital Amaurosis (LCA) and Best Vitelliform Macular Dystrophy disease. His research has resulted in patented methodology and technology to grow functional, three-dimensional retinal tissues and organoids containing rods and cones from human stem cells. Significantly, this technology has developed to the point that its therapy is currently in the first stage of human clinical trials, Phase 1/2a, designed to evaluate the safety and tolerability of subretinal administration in people with primary photoreceptor disease such as retinitis pigmentosa and cone-rod dystrophy. This is a most encouraging development for patients who suffer

from these inherited retinal diseases, since these diseases affect the structure and function of the photoreceptor cells in the retina and lead to irreversible vision loss.



John C. Dawson, Jr., RRF Chairman



Dr. Willis, Dr. Gamm, Mr. Dawson





From left back row: Bruce Lurie, Martha Lurie, Travis Lauritsen, Haley Boehm, Shawn Kavoussi
Front row: Susan Jensen, Mac Jensen, Mike Heim, Pat Heim, Jennifer Kavoussi



From Left, back row: Yingbin Fu, Wei Li, Peter Chang, Sam Wu, Theresa Chang, Zheng Jiang
Front row: Nicholas Tran, Elizabeth Zuniga Sanchez, Rinki Ratnapriya, May Chu

BCM Vision Research Symposium 2024 and Vision Seminar Series

In support of RRF’s educational goals, RRF is the sole sponsor of Baylor College of Medicine’s (BCM) **Vision Research Symposium**, held for the second time in December, 2024. Offered virtually and free to all participating vision scientists, the symposia was organized by RRF pilot grant scientist, Dr. Yingbin Fu, and Dr. Wei Li, both members of the Cullen Eye Institute at BCM. Sessions highlighted the latest, cutting-edge retina research, age-related macular degeneration, glaucoma, immune cells and inflammation, and corneal diseases along with a session of multiomics and artificial intelligence. Presenters included Connie Cepko, PhD, Harvard Medical School, who spoke on *Gene-agnostic gene therapy to prolong vision* as the keynote address, along with speakers from Baylor and other nationally recognized vision institutes. Over 355 scientists registered with nearly two-thirds attending the Saturday event, and attendees indicated they appreciated the excellent, high-quality science shared and vigorous discussion that followed each presentation.

RRF also collaborates with BCM to hold the **Vision Research Seminar Series**. The Vision Seminar Series provides a platform to connect vision research investigators and clinicians for scientific discussion and research collaboration through six to eight seminars held over the course of the year. The seminar series attracts vision research scientists from the Texas Medical Center in Houston, as well as from across the U.S. and around the world. The goal of this program is to develop a rich academic environment, cultivate new scientific ideas and dialog, and advance the frontier of vision research through presentation of the latest scientific breakthroughs in vision research.

Baylor Medicine



We are thrilled to invite you to the 2024 Baylor College of Medicine Vision Research Symposium. This exciting symposium will be held on ZOOM. The symposium is open to all vision scientists. The registration is FREE. We look forward to “meeting” you in the symposium!

BCM VISION RESEARCH SYMPOSIUM 2024

SATURDAY DECEMBER 7th

Register early to secure your spot



SYMPOSIUM ORGANIZERS

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The Sarah Campbell Blaffer
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RETINA RESEARCH FOUNDATION

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December 31, 2024

(with summarized financial information as of December 31, 2023)

	General Funds			Endowment Funds			2024 Total All Funds	2023 Total All Funds (Memorandum Only)
	Without Donor Restrictions	With Donor Restrictions	Total	Without Donor Restrictions	With Donor Restrictions	Total		
Assets								
Cash and cash equivalents	\$ 795,378	\$ 502,000	\$ 1,297,378	\$ -	\$ 500,739	\$ 500,739	\$ 1,798,117	\$ 1,416,568
Promises to give	196	-	196	-	1,000	1,000	1,196	116,446
Investments	3,477,817	-	3,477,817	6,010,725	67,837,613	73,848,338	77,326,155	70,781,579
Furniture and equipment, net of accumulated depreciation of \$12,588	13,300	-	13,300	-	-	-	13,300	13,414
Intangible assets	12	-	12	-	-	-	12	12
Other assets	6,772	-	6,772	-	-	-	6,772	8,686
Total assets	\$ 4,293,475	\$ 502,000	\$ 4,795,475	\$ 6,010,725	\$ 68,339,352	\$ 74,350,077	\$ 79,145,552	\$ 72,336,705
Liabilities and net assets								
Accounts payable	\$ -	\$ -	\$ -	\$ -	\$ 82,048	\$ 82,048	\$ 82,048	\$ 76,607
Grants payable	-	-	-	-	-	-	-	50,000
Total liabilities	-	-	-	-	82,048	82,048	82,048	126,607
Net assets	4,293,475	502,000	4,795,475	6,010,725	68,257,304	74,268,029	79,063,504	72,210,098
Total liabilities and net assets	\$ 4,293,475	\$ 502,000	\$ 4,795,475	\$ 6,010,725	\$ 68,339,352	\$ 74,350,077	\$ 79,145,552	\$ 72,336,705

RETINA RESEARCH FOUNDATION COMBINED STATEMENT OF ACTIVITIES AND CHANGES IN NET ASSETS

For year ended December 31, 2024

(with summarized financial information for the year ended December 31, 2023)

	General Funds			Endowment Funds			2024 Total All Funds	2023 Total All Funds (Memorandum Only)
	Without Donor Restrictions	With Donor Restrictions	Total	Without Donor Restrictions	With Donor Restrictions	Total		
Revenues								
Contributions	\$ 168,027	\$ 559,500	\$ 727,527	\$ -	\$ 130,847	\$ 130,847	\$ 858,374	\$ 469,200
Investment income, net	111,132	-	111,132	152,675	1,726,679	1,879,354	1,990,486	1,723,931
Realized and unrealized gains on investments, net	309,118	-	309,118	521,701	5,918,692	6,440,393	6,749,511	8,007,430
Mineral interest income and other income	30,141	-	30,141	-	-	-	30,141	18,822
Income transferred from Endowment Fund investments	2,249,244	50,000	2,299,244	(186,248)	(2,112,996)	(2,299,244)	-	-
Net assets released from restrictions - satisfaction of program and timing restrictions	238,356	(238,356)	-	-	-	-	-	-
Total revenues	3,106,018	371,144	3,477,162	488,128	5,663,222	6,151,350	9,628,512	10,219,383
Expenses								
Program services								
Research projects and grants	2,540,495	-	2,540,495	-	-	-	2,540,495	2,309,379
Supporting services								
Management and general	234,611	-	234,611	-	-	-	234,611	221,662
Total expenses	2,775,106	-	2,775,106	-	-	-	2,775,106	2,531,041
Changes in net assets	330,912	371,144	702,056	488,128	5,663,222	6,151,350	6,853,406	7,688,342
Net assets, beginning of year	3,962,563	130,856	4,093,419	5,522,597	62,594,082	68,116,679	72,210,098	64,521,756
Net assets, end of year	\$ 4,293,475	\$ 502,000	\$ 4,795,475	\$ 6,010,725	\$ 68,257,304	\$ 74,268,029	\$ 79,063,504	\$ 72,210,098

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