

Workshop: Immunity and Inflammation in the Anterior Segment of the Eye

Executive summary

National Eye Institute
Anterior Segment Initiative
May 17, 2021

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Overview

Eye inflammation is the fifth leading cause of blindness in the U.S.¹ with estimated costs similar to that of diabetic retinopathy¹. Host mechanisms that regulate immunity play a critical role in ocular inflammation, even in conditions not typically considered “immune-based”, since immunity regulates not just inflammation but also host tissue response to various stressors. These include diabetic retinopathy, age-related maculopathies, and glaucoma, among others. In 2019, the National Eye Institute (NEI) launched the Anterior Segment Initiative to better understand inflammation and its relationship with ocular pain, dry eye disease, the microbiome, and other eye conditions.

The National Eye Institute hosted the April 23, 2021, workshop, “Immunity and Inflammation in the Anterior Segment of the Eye,” to identify relevant gaps in knowledge, barriers to the development of new therapies, and promising areas for research exploration.

Reza Dana, M.D., M.Sc., M.P.H., Harvard Department of Ophthalmology, and Nida Sen, M.D., M.H.S., Janssen Retina and NEI volunteer faculty, co-chaired this event, which gathered two dozen subject-matter experts to address key topics, broken into four sessions: 1. immune homeostasis and regulation, 2. aging, the microbiome, and environmental factors, 3. neuro-immune interactions, and 4. resolution of inflammation.

Session 1: Immune homeostasis and regulation

Moderators:

Victor L. Perez, M.D., Duke Eye Center

Mary Ann Stepp, Ph.D., George Washington University School of Medicine and Health Sciences

When properly working, the immune system quickly responds to pathogens. Once infection is controlled, inflammation is quelled, and homeostasis returns. Achieving and maintaining homeostasis involves the orchestration of myriad components that can be influenced by an equal number of factors, such as genetics, age, and resident microorganisms. Failure to achieve homeostasis can lead to chronic inflammation and vision loss.

In an introduction, Perez underscored the importance of understanding the relationships between the ocular surface (cornea) and the surrounding tissue (adnexa) and between the innate and acquired immune systems. Studying specific conditions such as graft versus host disease and dry eye offer opportunities to explore the interplay between innate and adaptive immunity, he said.

Sharmila Masli, Ph.D., Boston University Medical Center, presented “Regulation of Ocular Mucosal Immune Homeostasis.” Masli explained that mucin-secreting goblet cells are key to maintaining the physical barrier between the environment and host cells. During chronic inflammation, goblet cells lose function or die off, leading to destabilization of the ocular surface’s tear film accompanied by reduced epithelial cell anti-inflammatory factors, which results in triggering of the adaptive immunity.

Studies of Sjögren’s syndrome patients provide an opportunity to better understand ocular inflammation. In Sjögren’s disease, damage to the conjunctival epithelium is accompanied by a loss of goblet cells and reduced tear mucin levels. Mucin 5AC is a large gel-forming glycoprotein and, according to Masli, the measurement of tear MUC5A levels provides an indicator of goblet cell function. Masli said that in intestinal mucosa, goblet cells are involved in antigen presentation via transepithelial dendrites (TEDs), enabling dendritic cells to sample intestinal microbes; however, not much is known about the function of TEDs in the conjunctiva.

Future directions to help understand ocular mucosal homeostasis, said Masli, include identifying additional factors related to the process; identifying dendritic cell-mediated mechanisms and their immune surveillance; and how toll-like receptor (TLR)-induced responses influence ocular surface immune homeostasis. Other opportunities include using RNA-seq analyses to explore cellular changes in the conjunctiva and lacrimal gland. A barrier to such studies is the sparsity of human tissue.

Holly Rosenweig, Ph.D., Oregon Health and Science University, presented “The Interplay Between Innate Immune Receptors and T Cells in Uveitis.” Uveitis is a heterogeneous group of intraocular inflammatory diseases affecting all ages, genders, and races. Together, the uveitides account for 10 to 15 percent of blindness worldwide. Two-thirds of uveitis cases are autoimmune; the rest have an infectious origin.

The commonly used Experimental Model of Uveitis (mouse) has implicated innate immune receptors as important factors in uveitis. Researchers have explored the role of TLRs. Rosenweig suggested that the exploration of other innate immune receptors, including C-type lectin receptors (CLRs) and nod-like receptors (NLRs), could be insightful. Rosenweig and others have shown that the NLR Nod2 is expressed in the eye. The pediatric disease Blau syndrome, which causes uveitis, is linked to a Nod2 mutation that causes hypersensitivity to pathogens, which may implicate the interplay between infectious organisms and autoimmunity.

Eric Pearlman, Ph.D., University of California Irvine, presented “Future Directions in Microbial Keratitis Research.” Keratitis, or inflammation of the cornea, due to bacterial or fungal infections is a major cause of

visual impairment worldwide. A 2019 study found that microbial keratitis causes more than a million clinic visits in the U.S.

The *Fusarium* fungus was responsible for a 2005/2006 outbreak of keratitis in the U.S. and Western Europe involving contamination of contact lens solution. These fungi can form biofilms on contact lens storage cases and contact lenses, increasing transference to the cornea where the fungi can penetrate the corneal stroma. Fungal keratitis is one of the most common causes of corneal ulcers.

Work by Pearlman and others suggests neutrophils are the predominant immune cell type present in patients with corneal ulcers caused by bacteria and fungi. Neutrophils kill microbes via reactive oxygen species and antimicrobial peptides. Neutrophils release proteases that can break down the intercellular matrix, causing corneal opacification.

Opportunities for future research include the exploitation of new -omics techniques (genomics, transcriptomics, metabolomics) to better understand subpopulations of neutrophils and other immune cells.

Future research needs include a better understanding of the role B cells play in ocular surface homeostasis. Traditionally, neutrophils have been difficult to study due to their short half-life; however, new techniques are enabling the identification of additional cell subtypes. Also needed is a greater understanding of the relationship between tissue environment and B cell activity.

Session 2: Aging, microbiome, and environment

Moderators:

Cintia S. de Paiva, M.D., Ph.D., Baylor College of Medicine

Louise D. McCullough, M.D., Ph.D., McGovern Medical School, University of Texas Health

The biggest risk factor for many immune-mediated diseases is age. Life's wear and tear on the body accumulates and systems increasingly falter as age advances. Risk of AMD, cataract, glaucoma, and ocular surface disease all increase with age. The microorganisms living in and on the body, collectively known as the microbiome, are increasingly understood to influence health and disease, yet little is known about the microbiome's impact on the ocular surface. Genetics, too, play a role in susceptibility to chronic inflammation.

The eye is an exposed mucosa; however, the existence of an ocular surface microbiome remains a subject of debate. Research has shown that microbial communities in other parts of the body influence ocular immunity. Studies of Sjögren's syndrome found that the absence of gut microbes in mouse models worsens dry eye symptoms. People with Sjögren's syndrome have decreased gut bacteria diversity.

Eric Huseby, Ph.D., University of Massachusetts Medical School, addressed the need for better understanding of the genetic determinants of autoimmune disease. Huseby explained how genetic variants of major histocompatibility complex II—a key protein involved in antigen presentation—affects susceptibility to diabetes. In combination, risk alleles promote tissue infiltration by T cells, thereby driving inflammation.

Age affects the body's immune composition, according to Vishwa Dixit, D.V.M., Ph.D., Yale University. Specific populations of T cells and B cells expand with age; however, other immune cell populations decrease. Dixit is studying inflammatory mediators that drive persistent release of proinflammatory cytokines via inflammasomes. He recently discovered that the sugar fucose specifically blocks the NLRP3 inflammasome assembly in macrophages. In fruit flies, supplemental fucose led to a 15-percent life extension.

Age also affects the microbiome. Louise McCullough presented data showing that older mice are less capable of sequestering gut bacteria. Younger mice have more beneficial bacteria (Bacteroidetes) and fewer harmful

bacteria (Firmicutes). Transferring gut bacteria from a younger mouse into an older mouse restores the gut's mucus barrier. Providing the prebiotic inulin and bacteria that produce short-chain fatty acids (SCFA) improves gut goblet cells, increases regulatory T cells, and decreases proinflammatory IL-17 cells.

Opportunities for future research include identifying specific bacterial strains or bacterial by-products such as SCFA that promote immune homeostasis via improved health in the brain, gut, liver, and other organs.

Session 3: Neuro-immune interactions

Moderators:

Zsuzsanna Fabry, Ph.D., University of Wisconsin

Jerry Y. Niederkorn, Ph.D., University of Texas Southwestern Medical Center

The cornea is one of the most densely innervated parts of the body. Corneal nerves are key to maintaining ocular surface homeostasis; however, relatively little is known about how they regulate it.

Loss of corneal nerves due to infection with herpes simplex virus (HSV) can give rise to conditions like neurotrophic keratitis. Sensory peptides such as Substance P are important drivers of inflammation via signaling with natural killer (NK) cells, explained Susmit Suvas, Ph.D., Wayne State University, who has examined their role in HSV infection. However, clinical trials of agents aimed at blocking Substance P/NK cell receptor binding (to disrupt inflammation) have unfortunately failed so far. Such findings, said Suvas, potentially hint at an alternative unknown signaling pathway for Substance P.

The eye is an immune-privileged organ, which is not the same as immune isolation, according to Monica Carson, Ph.D., University of California Riverside, who addressed what is known about resident macrophages in the central nervous system. Immune cells, she said, must be considered within the context of the tissue they inhabit and factors such as age and sex.

Technological innovations of the past two decades have advanced understanding of neuro-immune interactions. Such technologies include single-cell RNA sequencing, various -omics techniques, and better cell imaging. Functional heterogeneity within immune cell types is increasingly recognized as an important theme. A possible seven major types of microglia carry out a variety of roles. Microglia act as biosensors, defend against pathogens, and nourish other cells.

Lauren Sansing, M.D., Yale School of Medicine, provided perspective from research of inflammation in the brain. Stroke, she said, is a good model for studying the kinetics of neuroinflammation in acute injury. Microglia and macrophages both have distinct transcriptional profiles in the brain and cycle through distinct phases in response to injury. For example, macrophages are initially proinflammatory after brain injury but then later become crucial to neurological recovery. Microglia express homeostatic genes during the naive state, stop during the acute phase, and turn back on during recovery. She said macrophages can be considered the bulldozers that gobble up cellular debris; microglia fine-tune repair.

Session 4. Resolution

Moderators:

Pedram Hamrah, M.D., Tufts Medical Center

Michael E. Zegans, M.D., Dartmouth-Hitchcock Medical Center

Resolution of inflammation was once considered a passive process where the immune system simply fizzled out. Recent evidence redefines resolution as an active process where the immune system works to restore immune homeostasis.

Karsten Gronert, Ph.D., University of California Berkeley, provided an overview of specialized pro-resolving mediators (SPMs) that induce gene expression that stops recruitment of leukocytes and macrophages to sites of inflammation. SPMs are lipid mediators—short-acting cell membrane-derived signaling molecules. Scientists have extensively studied lipid mediators called eicosanoids. Every cell on the ocular surface expresses eicosanoids; however, little is known about eicosanoids' role in ocular immunity. Use of drugs that target eicosanoids, such as prostaglandins, in the eye are based on research in other organ systems. Opportunities for future research include defining “healthy” inflammatory responses—ones that likely resolve without intervention—and pathological inflammation that requires therapy.

Daniel Saban, Ph.D., M.S., Duke University, summarized the state of the science on neuro-immune interactions in the eye. Corneal nerves are central to corneal structure and function, sensing the environment and maintaining the epithelium. Inflammatory processes can perturb normal corneal nerve physiology and contribute significantly to clinical disease. “But we know almost nothing about how the immune system maintains nerve homeostasis in the cornea,” he said. Studies of the peripheral nervous system suggests that macrophages play a key role in homeostasis. As an example, he cited the intestinal muscularis where intestinal neurons bathe macrophages in colony-stimulating factor (CSF1). Saban has characterized the functional diversity of ocular macrophages using a lineage tracing technique. Ratios of short- and long-lived macrophages varied by eye structure. He has also shown that corneal macrophages reside near corneal nerves. These observations suggest yet-to-be-understood relationships.

Sunil Chauhan, D.V.M., Ph.D., Schepens Eye Research Institute, advocated for revisiting mast cells, which play a principal role in allergic responses and are present on the ocular surface and in high numbers in the limbus, conjunctiva, and lid margin. In addition to sensing external stimuli and initiating inflammation, mast cells help resolve inflammation. Chauhan highlighted mouse studies that show mast cell activity increases during corneal transplantation but that blocking mast cells delays allograft rejection. Mast cells also play a role in angiogenesis. Exploration of mast cell function outside of non-allergic eye conditions is needed, said Chauhan. Because they interact with corneal nerves, more research should be directed toward their role in itch, pain, and inflammation.

Conclusions

Co-chairs Reza Dana and Nida Sen summarized the sessions.

While the cornea and ocular surface are most often the target of anterior segment inflammation, it is important to recall that the adnexae (lacrimal and meibomian glands, eyelids, etc. that support the functioning of the ocular surface) are also increasingly recognized as playing a crucial role in pathogenesis of anterior segment inflammatory disorders. Certainly, more research is needed to understand the interplay between the cornea and its surrounding tissues. Multi-omics approaches will help identify key cells and biological mechanisms. A greater understanding of the relationship between the innate and adaptive immunity is also needed.

Factors including aging, diet, the microbiome, and the environment influence inflammation. However, more research is needed to understand how these factors exert their influence. For example, why does the aging eye tilt toward a ‘proinflammatory’ profile? Why and how does caloric restriction reduce inflammation? And how do genetics interact with these factors? And, to what extent are these principles applicable to the ocular environment?

Neuro-immune interactions remain an area of great interest. Microglia have disparate roles in the central nervous system that are influenced by age, sex, and anatomic region—both in driving inflammation and regaining homeostasis. Gaps include a lack of understanding of the relevance of immune cell lineage.

Among others, immune-resolution gaps include limited knowledge of lipid mediator networks, regulatory T cells and their potential as therapeutic targets, and the role of macrophages and mast cells in the context of corneal surface inflammation.

Agenda

Welcome by NEI director

10:00 – 10:10 a.m.

Michael F. Chiang, M.D.

Welcome and Logistics by Workshop Co-Chairs

10:00 – 10:15 a.m.

Reza Dana, M.D., M.Sc., M.P.H., Harvard Department of Ophthalmology/Massachusetts Eye and Ear

H. Nida Sen, M.D., M.H.Sc., National Eye Institute, NIH

Session 1: Immune Homeostasis and Regulation

10:15 – 11:35 a.m.

Moderators:

Victor L. Perez, M.D. Duke Eye Center

Mary Ann Stepp, Ph.D., George Washington School of Medicine and Health Sciences

Regulation of Ocular Mucosal Immune Homeostasis

Sharmila Masli, Ph.D., Boston University Medical Center

The Interplay Between Innate Immune Receptors and T Cells in Uveitis

Holly L. Rosenzweig, Ph.D., Oregon Health & Science University/ VA Portland Health Care System

Future Directions in Microbial Keratitis Research

Eric Pearlman, Ph.D., University of California Irvine

Discussion

11:35 – 11:45 a.m.

Session 2: Aging, Microbiome, and Environment

11:45 a.m. – 1:05 p.m.

Moderators:

Cintia S. de Paiva, M.D., Ph.D. Baylor College of Medicine

*Louise D. McCullough, M.D., Ph.D. McGovern Medical School at UTHealth

How Do MHC-II Polymorphisms Influence Autoimmune Susceptibility

Eric Huseby, Ph.D., University of Massachusetts Medical School

Metabolic Checkpoints of Inflammation

Vishwa Deep Dixit, D.V.M, Ph.D. Yale University

Age-Related Changes in Microbiota-Derived Metabolites

Susan McKarns, Ph.D., University of Missouri School of Medicine

*Dr. McCullough gave a presentation as Dr. McKarns could not attend due to an emergency.

Discussion

1:05 – 1:45 p.m.

Session 3: Neuro-Immune Interactions

1:45 – 3:05 p.m.

Moderators

Zsuzsanna Fabry, Ph.D., University of Wisconsin

Jerry Y. Niederkorn, Ph.D., University of Texas Southwestern Medical Center

Back to Basics: What Do Resident Tissue Macrophages Do in Immune Privileged CNS?

Monica J. Carson, Ph.D., University of California, Riverside

Diversity and Dynamics of Myeloid Responses in the Brain

Lauren H. Sansing, M.D., M.S., FAHA, FANA Yale School of Medicine

Neuropeptide-Mediated Regulation of Corneal HSV-1 Infection

Susmit Suvas, Ph.D. Wayne State University

Discussion

3:05 p.m. – 3:15 p.m.

Session 4: Resolution

3:15 – 4:35 p.m.

Moderators:

Pedram Hamrah, M.D. Tufts Medical Center

Michael E. Zegans, M.D., Dartmouth-Hitchcock Medical Center

Lipid Mediator Networks as Therapeutic Targets for Ocular Diseases

Karsten Gronert, Ph.D., University of California Berkeley

Neuro-Immune Interactions in the Eye

Daniel Saban, Ph.D., M.S., Duke University and Health System

Revisiting Mast Cell Function in Ocular Inflammation

Sunil Chauhan, D.V.M, Ph.D. Schepens Eye Research Institute

Discussion

Summary by the Co-Chairs

4:35 – 4:55 p.m.

Reza Dana

H. Nida Sen

Closing remarks

4:55 – 5:00 p.m.

Michael A. Steinmetz, Ph.D. National Eye Institute, director, NEI Division of Extramural Science Programs

Speaker Bios

Michael F. Chiang, M.D.

Dr. Chiang joined the National Eye Institute as director in November 2020. He brought to the institute a background shaped by the intersection of patient care and information technology. His clinical practice focuses on pediatric ophthalmology and strabismus, and he is board-certified in clinical informatics. His research develops and applies biomedical informatics methods to clinical ophthalmology in areas such as retinopathy of prematurity (ROP), telehealth, artificial intelligence, clinical information systems, genotype-phenotype correlation, and data analytics. Dr. Chiang received a BS degree in electrical engineering and biology from Stanford University in 1991, an M.D. degree from Harvard Medical School and the Harvard-MIT Division of Health Sciences and Technology in 1996, and an MA degree in biomedical informatics from Columbia University. He completed residency and pediatric ophthalmology fellowship training at the Johns Hopkins Wilmer Eye Institute. Between 2001 and 2010, he worked at Columbia University, where he was Anne S. Cohen associate professor of Ophthalmology and Biomedical Informatics, director of Medical Student Education in Ophthalmology, and director of the introductory graduate student course in biomedical informatics. From 2010 to 2020, he was Knowles Professor of Ophthalmology and Medical Informatics and Clinical Epidemiology, and associate director of the Casey Eye Institute, at the Oregon Health & Science University (OHSU). He co-directed an NIH-funded T32 training program in visual science for graduate students and research fellows, as well as an NIH-funded K12 clinician-scientist program at OHSU.

Santa J. Tumminia, Ph.D.

Dr. Tumminia was selected as NEI Deputy director in November 2018 and served as Acting director from 2019-2020. In these roles, she provided executive leadership and scientific expertise on NEI policies and initiatives, strategic and organizational leadership, research oversight and priority setting, and financial management. She has expertise in a range of vision research issues and has provided leadership on NIH-wide programs in genetics and genomic medicine, behavioral science, angiogenesis, nanomedicine, translational science, and rare diseases. In addition, she temporarily assumed the role of Acting NEI Scientific director in 2019, managing the NEI Intramural Research Program.

Dr. Tumminia earned a Ph.D. degree in biology from Rensselaer Polytechnic Institute in 1987. In her postdoctoral training, she examined the protein-nucleic acid interactions involved in ribosome assembly at the Roche Institute of Molecular Biology, Department of Biochemistry, Hoffman La Roche, Inc. Dr. Tumminia joined the NEI Laboratory of Mechanisms of Ocular Diseases in 1991. Her research focus was on the mechanisms of ocular diseases, specifically, glaucoma and cataract formation. She studied cataract formation and tested the efficacy of anti-cataract agents. She also developed a model system to mimic the effects of glaucoma. She then transitioned to the Foundation Fighting Blindness (FFB), the largest non-government funder of retinal disease research. At FFB, she held several positions, eventually becoming director of Grants and Awards overseeing the entire grant portfolio of the foundation.

Dr. Tumminia returned to NEI in the Office of the Director in 2003, where she developed policies to foster strategic partnerships with stakeholders including industry, patient advocacy groups, and individuals impacted by vision loss. She has been key in formulating policies on new basic and clinical research concepts and initiatives, such as the NEI Audacious Goals Initiative. She provided NEI leadership on stem cell policies and initiatives, standing up an NEI Office of Regenerative Medicine to catalyze collaboration. Other key initiatives include the Age-Related Macular Degeneration Integrative Biology Initiative designed to correlate AMD disease phenotype and cellular endophenotypes with patient genetics. She also stood up the NEI Office of Data Science

and Health Informatics as well as the Office of Vision Health and Population Science, whose focus is on public health.

Dr. Tumminia has received numerous awards including multiple NIH Director's Awards for the NIH- wide Strategic Plan Working Group, and for leading the eyeGENE® Initiative. In 2018, she received the NIH Director's Award in Mentoring. Her efforts led to increasing the number of female tenure-track investigators in the NEI intramural program. She has also supported workplace diversity through the NEI's Diversity in Vision Research and Ophthalmology (DIVRO) training program and is developing an NEI Strategic Plan on Diversity.

[Sunil Chauhan, D.V.M, Ph.D.](#)

Dr. Chauhan's research interests focus on immunomodulation and regenerative medicine for ocular inflammatory disorders. For the past 15 years, he has been interested in determining the functions of different immune and non-immune cells in the induction of immune tolerance and tissue repair. Dr. Chauhan has published nearly 100 research articles in the field of immunology in highly peer- reviewed journals such as Journal of Immunology, Blood, Mucosal Immunology, Arthritis & Rheumatology, American Journal of Transplantation, The American Journal of Pathology, and The Ocular Surface. His research has led to many important novel findings, including the involvement of highly pathogenic Th17

cells in the pathogenesis of dry eye disease, the loss of function of regulatory T cells in transplant rejection, and mesenchymal stem cell restoration of corneal transparency by secreting high levels of hepatocyte growth factor. More recently, he has focused his research on unraveling the novel mechanisms and functions of mast cells in the setting of non-allergic (IgE-independent) ocular

inflammation and injury. Recent work from his laboratory implicates mast cells as playing a crucial role in the orchestration and amplification of neutrophil-mediated tissue damage and corneal neovascularization. Dr. Chauhan continues investigations on defining the immunomodulatory function of stem cells and the contribution of mast cells in non-allergic inflammatory disorders of the eye.

[Reza Dana, M.D., M.Sc., M.P.H.](#)

Dr. Dana holds the Claes Dohlman Chair in Ophthalmology at Harvard Medical School. He is director of the Cornea Service at Massachusetts Eye and Ear, Senior Scientist and W. Clement Stone Scholar at the Schepens Eye Research Institute's Laboratory of Corneal Immunology, Transplantation and Regeneration. He is also a faculty member of the Immunology Graduate Program at Harvard Medical School.

A graduate of Johns Hopkins and Harvard, his work focuses on the disease mechanisms that underlie autoimmunity, scarring, graft failure, chronic

inflammation, and angiogenesis. A Gold Fellow of ARVO, he has authored over 450 articles and 160 reviews and book chapters. His published work has been cited more than 3,000 times (h-index=93). He has been the recipient of multiple awards, including the ARVO Cogan and Friedenwald Awards, the Physician-Scientist Award, Senior Investigator Award and the Stein Innovation Award from Research to Prevent Blindness, the Alcon Research Institute Award, the Thygeson Lectureship, the Endre A. Balazs Prize from ISER, and the Ellis Island Medal of Honor, among others.

He is Editor-in-Chief of Cornea, Senior associate Editor of The Ocular Surface, and Senior Editor of Encyclopedia of the Eye, and sits on the editorial boards of multiple other journals. In addition to his basic investigations, he leads a translational research program that has received 14 IND permits from the U.S. Food and Drug Administration. He has trained over 130 fellows and graduate students from 36 countries in his laboratory, and

over 90 fellows in his clinics to date; in 2014 he was recipient of the A. Clifford Barger Excellence in Mentoring Award, the highest mentoring award bestowed at Harvard Medical School.

Dr. Dana is co-chair of this workshop.

[Cintia S. de Paiva, M.D., Ph.D.](#)

Dr. de Paiva is an associate professor of Ophthalmology, Baylor College of Medicine in Houston, Texas. She received her M.D. degree from the State University of Campinas-UNICAMP in Sao Paulo, Brazil, and her Ph.D. degree from the University of Sao Paulo, Ribeirao Preto. Her research interests include Dry Eye, Ocular Surface Diseases, Animal Models of Sjogren syndrome, Microbiome, and Aging. The primary objectives of her research are to investigate the pathogenesis of dry eye-related diseases with the ultimate goal to improve the diagnosis, prognosis, and therapy of dry eye. Her laboratory focuses on epithelial immune interactions, with emphasis on the microbiome and aging.

[Vishwa Deep Dixit, D.V.M, Ph.D.](#)

Dr. Dixit is the Waldemar Von Zedtwitz Endowed Professor in the Departments of Comparative Medicine and Immunobiology at the Yale School of Medicine. He completed Bachelor and Master of Veterinary Science degrees in HAU, India. He did Ph.D. research at University of Hannover, Germany, with a fellowship from Deutscher Akademischer Austauschdienst (DAAD) and did his postdoctoral work at the NIH. Prior to Yale University, Dr. Dixit held a faculty position at the Pennington Biomedical Research Center in Baton Rouge. The Dixit laboratory studies immunometabolism and has identified that pro-longevity hormone FGF21 protects against thymic degeneration and T cell senescence during aging. He helped define the role of innate immune sensor NLRP3 inflammasome in causing age-related chronic diseases, insulin-resistance, type 2 diabetes, immunosenescence, and development of “inflammaging.” His laboratory has also identified that ketone metabolite β -hydroxybutyrate (BHB) is a therapeutic target to lower the NLRP3 inflammasome-dependent chronic inflammatory diseases and that ketones protect from infections by expanding gamma-delta T cells. Recently, his laboratory has discovered a new cell type called the nerve-associated macrophages (NAMs), which associate with the sympathetic neurons in adipose tissue, and defined their role in control of catecholamine degradation in aging. The ongoing work in the Dixit laboratory is investigating the mechanism of age-related inflammation as a trigger for chronic disease and to identify immunometabolic targets that enhance healthspan. The research in the Dixit laboratory is funded by the National Institutes of Health, Glenn Foundation for Aging Research, and Cure for Alzheimer Foundation.

[Zsuzsanna Fabry, Ph.D.](#)

Dr. Fabry is the vice chair of research in the Department of Pathology, director of the Cellular and Molecular Pathology Training Program, and a professor of pathology at the University of Wisconsin School of Medicine and Public Health. As a neuroimmunologist, for the last several years, Dr. Fabry has focused her research program on understanding immune privilege and immune surveillance in the central nervous system (CNS) and their contribution to CNS diseases including multiple sclerosis, stroke, and TB meningitis. The goal of her research program is to improve treatments of CNS inflammatory and neurodegenerative diseases.

Her research on the mechanisms of innate and adaptive immune responses in the CNS provided critical findings that led to a better understanding of the pathogenesis of neurological diseases.

Dr. Fabry is a pioneer in investigating the role of dendritic cells, important immune sentinel cells, in the CNS. Her group showed that the functional state, frequency, and distribution of dendritic cells in the central nervous tissue are limiting factors in the induction as well as the effector phases of neuroimmune diseases. This is an important new concept that shows that dendritic cells serve as gatekeepers for invading autoreactive T cells in

neuroinflammation. Dr. Fabry was the first to show that dendritic cells migrate into the CNS via interaction with the endothelial cells of the blood-brain barrier and this migration is regulated through adhesion molecules and chemokines. These studies are paving the way for novel immune-modulating therapies for MS.

In the clinical arena, Dr. Fabry is aiming to define the efficacy of helminth-induced immunomodulatory therapy for relapsing-remitting MS patients. These studies will lead to a better understanding of the underlying immunological mechanisms of helminth-mediated treatments and improve the design of future immunomodulatory clinical trials against a variety of autoimmune diseases.

Dr. Fabry has been engaged in graduate and postgraduate trainings for over 20 years. Her trainees are fully committed to a career in biological science. She is an active member of several training programs in Wisconsin, including the cellular and molecular biology, the neuroscience, the environmental toxicology, and the cellular and molecular pathology training programs.

Dr. Fabry has lent her expertise to serving as a member of numerous committees nationally, including the Veterans Administration Medical Research Committee for Neurobiology, the NIH Novel NeuroAIDS Therapies Integrated Preclinical and Clinical Program, the National Institutes of Health Clinical Neuroimmunology and Brain Tumors Committee, the Brain Injury and Neurovascular Pathologies Study Section, and the National Institute on Alcohol Abuse and Alcoholism Study Section. She is a member of the American Association of Immunologists, the International Association of Neuroimmunologists, the New York Academy of Sciences, the American Association for the Advancement of Science, and the Society for Neuroscience.

[Karsten Gronert, Ph.D.](#)

Born in Germany, Dr. Gronert received his BS and MS degrees in biology from the University of Texas at El Paso. He obtained his Ph.D. degree in cell physiology in 1995 from New Mexico State University and moved to Boston for postdoctoral training in inflammation and molecular pharmacology in the laboratory of Charles Serhan at Harvard Medical School and Brigham and Women's Hospital. He was promoted to Instructor (1999) and assistant professor (2002) at Harvard Medical School. He moved to New York Medical College in 2003, and in 2005 was promoted to associate professor in Pharmacology and Ophthalmology. Dr.

Gronert joined the University of California, Berkeley, faculty in 2007, was tenured in 2011, and was promoted to professor in the School of Optometry in 2014. He was the Solon M. and Pearl A. Braff Chair in Clinical Optometric Science (2008-2012) and chair of the Vision Science Graduate Program (2014-2018). He is also a member of the Infectious Diseases and Immunity Program at UC Berkeley. His research is focused on elucidating the role and regulation of intrinsic lipid circuits that control healthy and routine execution of ocular surface immune responses and maintain homeostasis and neuroprotection in the retina. His laboratory also has long-standing collaborations that investigate lipid circuits in the GI tract and airway innate immunity and diseases. He has served as a regular grant reviewer for the NIH (NEI, NIDDK, NIGMS), U.S. Department of Defense, and foundations. He is on the board of associate editors for Prostaglandins & Other Lipid Mediators, Eye & Contact Lens, and The Ocular Surface and a regular reviewer for immunology, ocular, and biomedical journals. Dr. Gronert is a consultant for Johnson & Johnson Vision Care. He was a plenary speaker at the New York Academy of Sciences and the keynote speaker at the 10th Biennial Banff Inflammation Workshop and received the Dean's Award from Louisiana State University, Neuroscience Center of Excellence.

[Pedram Hamrah, M.D.](#)

Dr. Hamrah is a clinician-scientist and professor of Ophthalmology, Immunology, and Neuroscience at Tufts University School of Medicine, where he is director of Clinical Research and director of the Center for Translational Ocular Immunology. Dr. Hamrah's research, focusing on neuro-immune crosstalk, has been

supported by numerous grants from the National Eye Institute, Research to Prevent Blindness, industry and foundation grants. He has authored over 150 peer-reviewed articles and over 40 reviews and book chapters, and has given more than 100 lectures and presentations worldwide. Dr. Hamrah currently serves as an associate editor for the journals *The Ocular Surface*; *Translational Vision*,

Science & Technology, *Frontiers in Ophthalmology*, and *BMC Ophthalmology* and is Cornea Section editor of the journal *Eye* and assistant editor of the journal *Ocular Immunology and Inflammation*.

[Eric Huseby, Ph.D.](#)

Dr. Huseby's work focuses on the development and function of autoreactive T cells. Findings have demonstrated that myelin-specific CD8 T cells can induce CNS autoimmunity with many characteristics of patients with multiple sclerosis (Huseby et al., *J Exp Med*, 2001; Sasaki et al., *J Immunol*, 2014), and that age-dependent immune tolerance mechanisms regulate pathogenic CNS-specific T cells (Huseby et al., *Immunity*, 2001). Dr. Huseby's group has pioneered the use of fluorescently labeled recombinant MHC and TCR multimers to probe $\alpha\beta$ TCR- pMHC ligand interactions (Stadinski et al., *Immunity*, 2011; *J Immunol*, 2014), and is pairing these techniques with T1D patients and animal models of autoimmunity to delineate how self-reactive TCRs escape tolerance induction and induce disease (Stadinski et al., *Nat Immunol*, 2016, 2019; Wyss et al., *Nat Immunol*, 2016). They have a strong commitment to studying autoimmune disease models through TCR repertoire analysis, structural biology, and advanced imaging techniques.

[Sharmila Masli, Ph.D.](#)

Dr. Masli's research interests are ocular immune regulation in the context of immune privilege and ocular mucosal immunity, specifically antigen-presenting cells and their ability to induce immunologic tolerance.

Dr. Masli's current projects include determining immunopathogenic mechanisms underlying ocular manifestations of Sjögren's syndrome, which include conjunctival goblet cell-mediated regulation of tolerogenic phenotype of antigen-presenting cells and homeostatic mechanisms that regulate immune responses in the lacrimal gland.

[Louise D. McCullough, M.D. , Ph.D.](#)

Dr. McCullough is the Roy M. and Phyllis Gough Huffington Distinguished Chair and professor of Neurology at McGovern Medical School at UTHealth and Chief of Neurology at Memorial Herman Hospital – Texas Medical Center. She is a physician-scientist and a practicing vascular neurologist with clinical expertise in sex/gender disparities, stroke prevention, stroke and aging, and acute stroke treatments and outcome assessment. She is well recognized for her work in cerebral vascular disease and is known for her research identifying sex differences in cell death pathways during stroke, which have now been shown to be a major factor in the response to ischemic insult. Working closely with the Society for

Women's Health Research (SWHR) and the Office of Research on Women's Health (ORWH), she was instrumental in the National Institutes of Health's requirement to include female animals in basic and translational studies.

[Susan McKarns, Ph.D.](#)

The overarching goal of Dr. McKarns' immunology research laboratory is to develop a better mechanistic understanding of the interactions between genetics and the environment on immune responses to improve precision medicine approaches to treat autoimmune disorders. They have a particular interest in understanding how gut microbiome dysbiosis and lipid dysmetabolism trigger inflammation and axonal demyelination in the central nervous system, including the optic nerve.

[Jerry Y. Niederkorn, Ph.D.](#)

Dr. Niederkorn has been engaged in NIH-sponsored research on the immunology of the eye for over 43 years. His research interests include (a) immune privilege;

(b) immunobiology of corneal allografts; (c) immunobiology of uveal melanoma and its metastases; and (d) immunobiology and pathobiology of *Acanthamoeba* keratitis.

[Eric Pearlman, Ph.D.](#)

Dr. Pearlman has been working on corneal inflammation, immunity, and infection since 1993. His earlier studies as a postdoctoral fellow and assistant professor were on the pathogenesis of ocular onchocerciasis (river blindness) using NEI-funded research on murine models of infection. In 2006, following an outbreak of contact lens-related fungal corneal infections in the United States, he initiated studies on *Fusarium* and *Aspergillus* keratitis, which are major causes of blindness in developing countries. His current research in bacterial and fungal keratitis is to understand the pathogenesis of these infections from the perspective of both the host and the pathogens, with the long-term goal of identifying novel targets to

inhibit growth of these pathogens and to block the tissue damage caused by the host inflammatory response.

Specifically, his current research program examines innate immune responses to fungal and bacterial corneal infections using murine models and human neutrophils. His focus is on the role of neutrophils and monocytes in the context of host defense to bacterial and fungal infections, and he examines the role of neutrophils as a source of the highly pro-inflammatory cytokine IL-1 beta.

[Victor L. Perez, M.D.](#)

Dr. Perez was recruited to Duke University School of Medicine in September 2017 as professor of Ophthalmology to lead the development of the unique Foster Center for Ocular Immunology at Duke Eye Center. He is an established clinician-scientist investigator in the fields of ocular immunology, transplantation, and ocular surface diseases. He has been supported by the National Eye Institute of the National Institutes of Health for the last 16 years to develop a translational program in ocular immunity and transplantation. Presently, Dr. Perez is the Stephen and Frances Foster Distinguished Endowed Chair in Ocular Immunology and director of the established Foster Center at Duke Eye Center in a translational

clinical practice. He complements this with his work in the clinic, evaluating and treating patients with ocular inflammatory diseases and conditions of the anterior segment. In addition to ocular surface, he also manages patients with uveitis. Dr. Perez's laboratory focuses primarily on researching the immunology of corneal transplantation and inflammatory dry eye in ocular graft versus host disease (GVHD). Dr. Perez and his colleagues use a mouse model of corneal transplantation and ocular GVHD that allows for translational research relevant to patients with penetrating keratoplasty and inflammatory dry eye. Members of Dr. Perez's laboratory have also used the mouse eye as an in vivo imaging platform to study T cell recruitment and in situ activation. The aim of this work is to develop a translational research clinic to study the natural history of disease progression in inflammatory ocular surface diseases and dry eye, by testing the role of mediators of inflammation in this process. One of the most innovative aspects of this program is the multidisciplinary team—consisting of scientists from the ophthalmology, basic immunology, and oncology, rheumatology, and solid organ transplantation units—whose members work together to comprehensively tackle the research and care of patients with ocular inflammatory diseases. The knowledge and development of potential new preventive therapies that are being generated in the laboratory will have a direct translational impact on the care of patients with ocular inflammatory disease at the Duke Eye Center.

Holly L. Rosenzweig, Ph.D.

Dr. Rosenzweig, has a long-standing interest in how innate immune receptors shape autoinflammatory/autoimmune disease. This began with her Ph.D. training in immunology (where she investigated Toll-like receptors in ischemic brain injury). She then completed a fellowship at Casey Eye Institute (OHSU), where she initiated her research pertaining to the then newly discovered Nod-like receptors in uveitis. Subsequently, she expanded her research experience in the field of rheumatology in order to further appreciate uveitis susceptibility in the context of multisystemic diseases, work that involved a variety of experimental arthritis models.

Dr. Rosenzweig is currently an associate professor of Molecular Microbiology and Immunology at Oregon Health & Science University and the Portland VA Health Care system (where her laboratory is located). Current research efforts in the Rosenzweig laboratory support a new paradigm that complex environmental and genetic interactions result in break in immune tolerance and trigger autoreactive T cells that cause disease. Innate immune receptors exist at the interface of host-microbe interactions and play a pivotal role in host defense, yet can participate in autoimmune/autoinflammatory diseases. The laboratory's work has brought new insight into the cross-talk among innate immune receptors (such as Nod2) and T cell responses in uveitis, which are relevant to clinical diseases such as Blau syndrome.

Daniel Saban, Ph.D., M.S.

Dr. Saban is an associate professor at Duke University School of Medicine, Departments of Ophthalmology and Immunology. He also serves there as the Scientific director of the Foster Center for Ocular Immunology, as well as the co-director of the Neuroimmunology and Glia Group at the Duke Institute for Brain Sciences. Dr. Saban also chairs the Immunology and Microbiology Section program committee for the 2021 ARVO Annual Symposium. He is also a co-chair of the Immunology Subcommittee for the Stephen J. Ryan Initiative for Macular Research at Doheny Eye Institute this year, and the Immunology Incubator at Roche pRED.

Dr. Saban's laboratory studies the interactions between immune and nonimmune cells in the mammalian visual system, with a particular focus on tissue-resident immune cells and their accessory contributions to the function of primary tissue cells such as neural cells, fibroblasts, and epithelial cells in the eye.

Lauren H. Sansing, M.D. , M.S., FAHA, FANA

Dr. Sansing is Academic Chief, Division of Stroke and Vascular Neurology, and associate professor of Neurology and Immunobiology. She is a physician-scientist studying inflammatory mechanisms of secondary brain injury after stroke, intracerebral hemorrhage, and other neurological diseases, applying advanced immunological methodologies to discover potential therapeutic targets. Her work integrates studies in human biological samples and murine models in order to identify inflammatory pathways that can be targeted to improve outcomes after brain injury. Her research has been recognized with the Michael S. Pessin Award from the American Academy of Neurology, the American Neurological

Association's Derek Denny-Brown Neurological Scholar Award, and election into the American Society of Clinical Investigation. She co-directs the Yale Neurosciences R25 training program, is the PI of Yale's program in the first-ever NINDS multisite preclinical trial platform testing candidate neuroprotectants for the treatment of stroke (SPAN), collaborates broadly across basic and clinical disciplines, and serves as a dedicated faculty mentor for trainees and junior faculty.

H. Nida Sen, M.D. , MHSc

Dr. Sen obtained her M.D. degree from Hacettepe University of Turkey and a Master of Health Sciences degree from Duke University. She completed her ophthalmology residency at The George Washington University and

her uveitis and ocular immunology fellowship at the National Eye Institute (NEI). She is a Lasker Clinical Research Scholar and clinical investigator at the NEI. Dr. Sen's research primarily focuses on understanding the mechanisms involved in different forms of human uveitis on the premise that better understanding of the disease will lead to developing innovative methods of diagnosis and novel treatment approaches. She has led many clinical trials and natural history studies

in ocular inflammatory diseases. She has been and remains a collaborator on several NIH-funded grants. She has received several awards for her research including Senior Achievement Award of the American Academy of Ophthalmology, Prevention of Blindness (POB) Society of Metropolitan Washington Research Award, and NIH Lasker Clinical Research Scholar Award. She is also an NIH Distinguished Scholar. In addition to her research activities, Dr. Sen is an active clinician and an educator. She is the director of the Uveitis Clinic and Uveitis and Ocular Immunology Fellowship Program at NEI and has mentored many clinical fellows, residents, and MRSP students. She is a board-

certified ophthalmologist, a participating member of the American Academy of Ophthalmology and international research networks, and is the President of The American Uveitis Society.

[Mary Ann Stepp, Ph.D.](#)

Dr. Stepp is professor of anatomy and cell biology and professor of ophthalmology at George Washington School of Medicine & Health Sciences in Washington, DC, where she studies the cornea with a focus on the corneal epithelial cells and the intraepithelial corneal nerves. Her work using mouse models has contributed to our understanding of the roles played by time of day, age, sex, and dry eye disease in corneal wound healing and reinnervation. She is currently a member of the National Advisory Eye Council (NAEC) for the NEI.

[Susmit Suvas, Ph.D.](#)

The focus of Dr. Suvas' laboratory is to understand the pathogenesis of herpes stromal keratitis (HSK) and find novel targets whose manipulation can reduce the severity of herpes simplex virus-induced corneal inflammation. They are looking at neuropeptides' role in HSV-1-induced corneal inflammation and the latency of HSV-1 in the trigeminal ganglia.

[Michael E. Zegans, M.D.](#)

Dr. Zegans has been on the faculty at Geisel School of Medicine at Dartmouth in Hanover, New Hampshire, since 1998. He is a professor of Surgery and of Microbiology and Immunology and the Section Chief of Ophthalmology at Dartmouth-Hitchcock Medical Center as well as the Francis A. L'Esperance, Jr., M.D., Visual Sciences Scholar. He completed his residency at the University of Virginia and a fellowship in cornea and uveitis at the F.I. Proctor Foundation at the University of California, San Francisco. His subspecialty clinical focus is on corneal diseases and uveitis. He was the primary investigator for 5K08EY13977-4 - Biofilm Formation and P. aeruginosa infection of the eye and R21 EY02877-01 -

Fungal virulence factors during corneal infections. He has served as a co-investigator for the Steroids for Corneal Ulcers Trial (SCUT) and Mycotic Ulcer Treatment Trial (MUTT). Additionally, he is the Dartmouth Site director for the National Eye Institute-funded Standardization of Uveitis Nomenclature (SUN) study and on the steering committee for the Zoster Eye Disease Study (ZEDS). Dr. Zegans' current laboratory research focuses on fungal corneal infection.

[Houmam Araj, Ph.D.](#)

Dr. Araj is director of the Lens and Cataract Program; the Oculomotor Systems and Neuro-Ophthalmology Program; the Ocular Pain Program; and the Conference Grants. Dr. Araj is also the NEI representative on the

trans-NIH CounterACT (Countermeasures Against Chemical Threats) Program. Prior to becoming Program director, Dr. Araj served as Scientific Review Officer for 10 years: 5 years at NEI and 5 years before that at NIMH. Among Dr. Araj's professional activities, he organized the Ocular Health Subgroup of the Indoor Air Pollution workshop and he co-organized the 2020 Trans-Agency Scientific Meeting on Developing Medical Countermeasures to Treat the Acute and Chronic Effects of Ocular Chemical

Toxicity. Prior to joining NIH, Dr. Araj did postdoctoral work in the Department of Neuroscience at the Johns Hopkins University School of Medicine.

[Nataliya Gordiyenko, Ph.D.](#)

Dr. Gordiyenko is director of the Retinal Angiogenesis and Immunology Program at the National Eye Institute, NIH. Earlier in her career, she was Scientific Review Officer for the Diseases and Pathophysiology of the Visual System (DPVS) Study

Section at the NIH's Center for Scientific Review. Previously Dr. Gordiyenko was an investigator in the Ophthalmic Genetics and Visual Function Branch and a postdoctoral fellow in the Laboratory of Retinal Cell and Molecular Biology at National Eye Institute. Her research focused on the mechanisms of cholesterol uptake and transport within the retina and the role of oxidized lipids and anti-oxidative enzymes in the pathogenesis of age-related macular degeneration (AMD)

and on the mechanisms of retinal pigment epithelium degeneration in the pathogenesis of Choroideremia. Dr. Gordiyenko earned her Ph.D. degree in biology with specialization in biophysics from the Institute of Physiology and Biophysics in Tashkent, Uzbekistan (former USSR).

[George A. McKie, D.V.M, Ph.D.](#)

Dr. McKie is Program Officer at the National Eye Institute for Structure, Functions and Diseases of the Cornea. She joined the NEI in 2011.

Dr. McKie did her undergraduate work in biology at the University of Missouri- Columbia, where she obtained her D.V.M degree in 1994. She obtained a Ph.D. degree in 2004 in comparative biosciences from the University of Wisconsin- Madison, where her research focused on corneal wound healing. She completed a residency in veterinary ophthalmology at the University of Wisconsin-Madison in 2000.

[Michael A. Steinmetz, Ph.D.](#)

Dr. Steinmetz joined the National Eye Institute after serving 5 years as the Scientific Review Administrator for the Central Visual Processing and Cognitive Neuroscience study sections and as a Referral Officer for the Health of the Population and Risk Prevention and Health Behavior review groups at NIH's Center for Scientific Review. He served as the director of the Strabismus, Amblyopia, and Visual Processing Program at NEI from 2007 to 2014 and as the director of the Division of Extramural Science Programs since October of 2014. He serves on the coordinating committees of numerous trans-NIH and trans-agency initiatives including the NIH BRAIN and Neuroscience Blueprint programs, and the programmatic panel of the DOD's Vision Research Program.

[Sangeeta Bhargava, Ph.D.](#)

Dr. Bhargava is a Program director in the Collaborative Clinical Research Program (CCR) at the National Eye Institute (NEI). She provides scientific leadership and guidance to the planning, development, implementation, and evaluation of assigned biomedical research in CCR. She has overall programmatic responsibility for the assigned program area and is the primary liaison and contact point for that program for all interactions within and outside the National Institutes of Health.

Prior to joining NEI, she was an assistant director at the CSR in the Division of Receipt and Referral and a Program director for Immunology and Immunotherapy

at the NIDCR. Previously Dr. Bhargava was a senior scientist at Wyeth Pharmaceuticals, now called Pfizer, where she was responsible for research on mucosal immunology with an emphasis on the discovery and preclinical development of viral vaccines. Dr. Bhargava earned a Ph.D. degree in medical sciences from the All-India Institute of Medical Sciences and subsequently was a postdoctoral fellow in mucosal immunology at the University of Pennsylvania.

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