2025 Vision Research Seminar Series

Feb. 13, 2025: Degui Zhi, Ph.D.

Seminar Title: Learning Endophenotypes from Retina Images
Time: 3 p.m. via Zoom and in person

Speaker Bio: Degui Zhi, Ph.D., is the founding chair of the Department of Bioinformatics and Systems Medicine. He is the Glassell Family Professor and founding director of the Center for AI and Genome Informatics (AIGI). Zhi joined the McWilliams School of Biomedical Informatics at UTHealth Houston, formerly UTHealth Houston School of Biomedical Informatics (SBMI), on July 1, 2016. He received his PhD in bioinformatics at UC San Diego. Before joining UTHealth, he was a tenured associate professor of statistical genetics at the University of Alabama at Birmingham.

Dr. Zhi is interested in developing AI deep learning and informatics methods for biomedical big data. His team develops multiple generalist deep learning frameworks for the modeling of biobank-scale biomedical data. Recent works from the lab include Med-BERT, a clinical foundation model for structured clinical data; gene2vec, a distributed representation embedding model for genes based on their co-expression patterns; and several unsupervised deep learning models for deriving endophenotypes from imaging data for genetic discovery. His team also developed advanced PBWT-based data structures and algorithms for population genetics informatics.

His methodological research has been funded by multiple PI/MPI grants totaling over $20 million. His current funded projects cover topics in AI-powered brain imaging and retina imaging genetics, population genetics informatics, and EHR predictive modeling. He is also interested in using LLM and generative AI for scientific discovery. He has been teaching the deep learning for biomedical informatics course since 2018. Dr. Zhi is an elected fellow of the American College of Medical Informatics (ACMI).

Seminar Summary: While genome-wide association studies (GWAS) have fueled the amazing genetic discovery in the past 15 years or so, most existing studies were using traditional phenotypes. With deep learning-based AI, it is possible to generate many new phenotypes. Powered by big data in biobanks, many new loci can be discovered. As a result, the landscape of GWAS might be different. In this talk, I will discuss a possible future with large-scale AI-driven GWAS. Using retina fundus photos, we derive several versions of endophenotypes that empower genetic discovery.

March 13, 2025: Zheng Jiang, Ph.D.

Seminar Title: Functional Variations of RGC Subtypes in Different Retinal Regions Revealed by High-Density Multielectrode Array Recordings
Time: 3 p.m. via Zoom and in person

Speaker Bio: Dr. Zheng Jiang received a B.S. in clinical medicine from China Medical University in 2004 and went on to earn a Ph.D. in integrative biology from Florida Atlantic University in 2009. He completed his postdoctoral training in Neuroscience at Johns Hopkins University in 2015 under the mentorship of Dr. King-Wai Yau, where he investigated the phototransduction mechanisms of intrinsically photosensitive retinal ganglion cells (ipRGCs) with a focus on melanopsin pathways. His research uncovered a novel phototransduction pathway in M2- and M4-ipRGCs, challenging established views on phototransduction motifs across species. In 2019, he joined the Department of Ophthalmology at Baylor College of Medicine as an assistant professor. His lab's research focuses on melanopsin transduction mechanisms and the electrophysiological properties of retinal ganglion cells using high-throughput methods.

Seminar Summary: The retina offers a unique model for studying neural processing, with retinal ganglion cells (RGCs) serving as the output neurons that transmit visual information to the brain. Employing High-Density Multielectrode Array (HD-MEA) technology, we recorded light responses from thousands of RGCs simultaneously, using diverse visual stimuli to investigate retinal circuitry systematically. Through these recordings, we identified over 30 RGC subtypes, each with unique electrophysiological characteristics. Interestingly, we found that RGC subtypes exhibited significant physiological variation across different retinal regions (dorsal, ventral, temporal and nasal), highlighting region-specific functionality within the retina. Additionally, our analysis also revealed subtype-specific synchronized firing patterns, providing new insights into how RGCs encode visual information through network interactions. The extensive, cell-type-specific dataset generated by this work holds significant potential for understanding retinal ganglion cell functions in healthy and diseased retinas.

April 10, 2025: Theodore Wensel, Ph.D.

Seminar Title: Nanoscale Imaging of Wildtype and Mutant Retinas
Time: 3 p.m. via Zoom and in person

Speaker Bio: Dr. Theodore G. Wensel is the Welch Professor of Biochemistry and Molecular Pharmacology at Baylor College of Medicine. His laboratory investigates the

molecular mechanisms of intracellular signal transduction, primarily within the nervous system, and explores innovative strategies to understand and develop therapies for diseases linked to signaling defects and sensory neurodegeneration. His research employs diverse techniques, including X-ray crystallography, cryo-electron microscopy, mutagenesis, and time-resolved luminescence energy transfer, to elucidate structural bases of signaling at molecular and atomic resolutions. Additionally, the lab extensively utilizes genetically engineered animal models to examine the consequences of molecular defects in vivo.

Seminar Summary: Nanoscale imaging has become one of the most powerful methods for understanding retinal structure, function, and mechanisms underlying retinal degeneration. Our work employs conventional transmission electron microscopy, cryo-electron tomography, and advanced fluorescence microscopy techniques. This seminar will present an overview of our findings, highlight significant conclusions drawn from our studies, and introduce cutting-edge techniques currently under development. These include Correlative Light and Electron Microscopy (CLEM), cryo-Focused Ion Beam/Scanning Electron Microscopy (FIB-SEM) with lift-out preparation, Machine Learning-Powered Sub-tomogram Averaging, Single Molecule Localization Microscopy (SMLM: STORM & PALM), Structured Illumination Microscopy (SIM), and Expansion Microscopy (exM). Illustrative examples will focus on rod sensory cilia—specifically, the connecting cilia and outer segments—and ciliopathy-associated proteins, featuring mouse models with mutations in CEP290, SPATA7, RPGR, Bardet-Biedl Syndrome (BBS) proteins, and the cyclic nucleotide-gated (CNG) channel beta-subunit.

May 15, 2025: Chi Hwan Lee, Ph.D.

Seminar Title: Smart Contact Lenses and Beyond: Translational Wearable Technologies for Chronic Disease Management
Time: 3 p.m. via Zoom and in person

Speaker Bio: Dr. Chi Hwan Lee is a Fellow of the American Institute for Medical and Biological Engineering (AIMBE) and the Lesli A. Geddes Professor of Biomedical Engineering and Mechanical Engineering, and by Courtesy, of Materials Engineering, Electrical and Computer Engineering, and Speech, Language, and Hearing Sciences at Purdue University. He obtained his M.S. and Ph.D. degrees in Mechanical Engineering from Stanford University in 2009 and 2013, respectively. His research focuses on developing wearable devices to address unmet clinical needs and translate them into measurable clinical impacts. For his notable contributions, Dr. Lee has been honored with prestigious

awards such as the 2025 Purdue CoE Faculty Research Award, 2021 Sensors Young Investigator Award, 2020 Purdue CoE Early Career Research Award, 2019 NIH Trailblazer Award, and 2019 Korean-American Scientists and Engineers Association (KSEA) Young Investigator Award. He has published over 90 journal papers and six book chapters, and issued 11 U.S. patents, filed > 15 utility patents, and co-founded four startup companies.

Seminar Summary: My laboratory at Purdue University focuses on bridging the gap between engineering and unmet clinical needs through innovations in wearable technologies. We develop novel yet simple flexible micro-transducers with a clear translational pathway to clinical impact. Our research explores wearable biomedical devices that safely attach to the skin or eye, enabling continuous, remote monitoring of health and chronic diseases with applications in healthcare, rehabilitation, and telemedicine. In this talk, I will present: (1) Sticktronics—sticker-like thin-film electronics attachable to curved surfaces for broader industrial and healthcare use; (2) sensory skin patches designed for urgent clinical needs in telemedicine; (3) smart contact lenses, built on commercial soft lenses, for continuous monitoring of chronic ocular diseases such as glaucoma; and (4) flexible, biodegradable patches embedded with injectable silicon nanoneedles for painless, sustained ocular drug delivery. I will share experimental and theoretical insights across these platforms.

June 26, 2025: Wei Li, Ph.D.

Seminar Title: Ligandomics: A New Approach to Drug Discovery for Ocular and Systemic Diseases
Time: 3 p.m. via Zoom and in person

Speaker Bio: Dr. Wei Li is a professor and the Knights Templar Eye Foundation Presidential Chair in Ophthalmology at Baylor College of Medicine, Texas, USA. He earned his B.Sc. in Pharmacy from Zhejiang University, China, in 1983, followed by an M.S. in Pharmacology from Zhejiang University College of Medicine in 1986. He received his Ph.D. in Pharmacology from the University of Nebraska Medical Center in 1991 and completed five years of postdoctoral training in pharmacology at Yale University. Dr. Li previously served for 19 years at the Bascom Palmer Eye Institute, University of Miami, where he held positions as Assistant, Associate, and Full Professor. In 2020, he joined Baylor College of Medicine. His research centers on the molecular mechanisms of retinal diseases, drug target discovery, and the development of novel therapies. Dr. Li has authored over 70 peer-reviewed publications and holds seven pending or issued patents.

Seminar Summary: We developed ligandomics, a groundbreaking technology that enables comprehensive mapping of cell-binding ligands to uncover disease mechanisms and identify therapeutic targets. Unlike existing omics platforms, such as proteomics and transcriptomics, which are either quantitative or functional, ligandomics uniquely offers simultaneous quantitative and functional profiling across biology. In ocular vasculopathies, comparative ligandomics identified disease-selective angiogenic factors, including secretogranin III (Scg3), leading to two targeted anti-angiogenic therapies. We have since expanded its application to glaucoma to discover neuroprotective targets, and are now pursuing therapeutic innovations in fibrotic disease, Alzheimer's disease, chronic kidney disease, and atherosclerosis. This seminar will introduce the ligandomics platform, highlight successful case studies for drug discovery, and explore its broad potential for future applications and collaborations.

July 10, 2025: John O'Brien, Ph.D.

Seminar Title: Dynamic Electrical Synapses: Unravelling Plasticity One Protein at a Time
Time: 3 p.m. via online and in person

Speaker Bio: Dr. John O'Brien is the Benedict/McFadden Professor of Optometry at the University of Houston College of Optometry. He earned his B.A. in Biochemistry at Bowdoin College and his Ph.D. in Biochemistry at the University of California, San Diego. Dr. O'Brien was on the faculty of the McGovern Medical School of the University of Texas Health Science Center at Houston for 23 years, where he held the Luisa Stude Sarofim Distinguished Chair in Ophthalmology prior to moving to UHCO in 2021. Dr. O'Brien has a long-standing interest in electrical synapses, especially molecular mechanisms of their functional plasticity. He has identified intrinsic mechanisms that regulate electrical synapse function and elucidated signaling pathways that control coupling in AII amacrine cells and photoreceptors. His recent studies have shed light on the complex suites of proteins that control the Connexin 36 lifecycle and form electrical synapses. Dr. O'Brien's lab has recently begun to study regeneration of rod photoreceptors in a transgenic zebrafish model of Retinitis Pigmentosa developed in his lab. His group has mapped out transcriptional pathways involved in the proliferation and differentiation of progenitor cells to form new rods and studies the role of microglia in retinal regeneration. His lab applies a variety of biochemical, molecular and genetic techniques in their investigations.

Seminar Summary: Neural circuits in the retina and brain are constructed with both chemical and electrical synapses, which have different functional properties and serve distinct roles within circuits. In the retina, electrical synapses form central elements of the

rod visual pathways, and their plasticity is critical for adaptation between dark and light conditions. Our work examines the mechanisms that control this functional plasticity that tunes retinal circuits to function optimally under different conditions. In this seminar, I will present circuit-specific signaling mechanisms that control electrical synapse plasticity in photoreceptors and AII amacrine cells over time scales of a few minutes. These mechanisms control circuit changes during light adaptation by implementing an order of magnitude dynamic plasticity of electrical synapse function. However, they do not represent a complete picture of electrical synapse plasticity. To develop a holistic understanding of electrical synapse regulation, we have recently employed proximity biotinylation-based proteomics in the retina to characterize the suites of proteins associated with Connexin 36, the most abundant protein forming electrical synapses. This has led to new insights into the complex of proteins forming the electrical synapse equivalent of the post-synaptic density, as well as proteins involved in trafficking and turnover of the connexins. This allows us to name players in the connexin lifecycle and identify candidate genes that may help us to understand how electrical synapses become dysregulated in disorders such as retinal degeneration or ischemia.

Sept. 11, 2025: Akrit Sodhi, M.D., Ph.D.

Seminar Title: Transient Hypoglycemia: A New Culprit in Diabetic Retinopathy's Molecular Story
Time: 3 p.m. via online and in person

Speaker Bio: Dr. Sodhi graduated Summa Cum Laude from the University of California at Los Angeles with a B.S. in Microbiology and Molecular Genetics in 1994 and received his M.D. and Ph.D. from the University of California, Davis in June 2004. During this time, he performed research at the National Institutes of Health (NIH) in the laboratory of Dr. J. Silvio Gutkind, a member of the National Academy of Medicine. Dr. Sodhi completed residency training in Ophthalmology at the Wilmer Eye Institute at the Johns Hopkins School of Medicine in June 2008 and is Board Certified in Ophthalmology. He then received subspecialty training in Medical and Surgical Diseases of the Vitreous and Retina from the Wilmer Eye Institute as the Mary Hutchinson Endowed Vitreoretinal Fellow and served as the Wilmer Assistant Chief of Service and the Associate Director of Ocular Trauma at Johns Hopkins from 2009 to 2010. Dr. Sodhi joined the Retina Division at the Wilmer Eye Institute as an Assistant Professor in July 2010 and was promoted to Associate Professor in 2017. From 2010 to 2015, Dr. Sodhi received an NIH K award grant to train as a clinician-scientist with Dr. Gregg L. Semenza, winner of the 2019 Nobel Prize in Physiology or Medicine, as his mentor.

Dr. Sodhi currently holds the inaugural Branna and Irving Sisenwein Professorship in Ophthalmology at the Johns Hopkins School of Medicine and has held that position since 2018. He also serves as the Vice Chair for the Wilmer EyeCare Network, overseeing the satellite offices of the Wilmer Eye Institute. Dr. Sodhi is a practicing clinician and surgeon and maintains a busy practice at the Wilmer Eye Institute. In addition, he runs a basic science and translational research laboratory at Johns Hopkins studying the molecular pathogenesis of diseases of the macula and retina. He has held continued NIH funding since he joined the Retina Division of the Wilmer Eye Institute in 2010. The research focus of his lab is to examine how the neurosensory retina and retinal pigment epithelium respond to injury at the cellular level with the goal of identifying novel biomarkers and effective therapeutic targets for the diagnosis, prevention, and treatment of vision-threatening diseases.

Seminar Summary: Tight glycemic control (TGC) is the most effective approach to reduce the risk for diabetic retinopathy (DR), the most common microvascular complication in patients with diabetes and a leading cause of vision loss in working-age Americans. Multiple clinical trials have demonstrated the therapeutic benefit of the diligent management of blood sugars in diabetic patients with diet, exercise, oral or injectable (non-insulin) medications, and insulin therapy. However, several studies have shown that these approaches can also cause early worsening of DR. We have recently reported that transient episodes of low glucose, an undesirable but common consequence of TGC, result in hypoxia-inducible factor (HIF)-dependent expression of the glucose transporter, Glut1, thereby promoting glucose uptake and retinal cell glycolysis. Enhanced nuclear accumulation of HIF-1α is independent of its canonical post-translational stabilization, but instead dependent on stimulation of its translation and (independently) its nuclear localization. In the diabetic retina, this physiologic response to low glucose also resulted in a marked increase in the secretion of the HIF-dependent vasoactive mediators that collectively promote breakdown of the inner blood-retinal barrier (iBRB) and retinal vascular leakage. Inhibition of HIF-1α using the pharmacologic HIF inhibitor, 32-134D, prevented the increase in expression of HIF-regulated vasoactive genes following transient episodes of hypoglycemia, blocking breakdown of the iBRB and the promotion of retinal vascular hyperpermeability in diabetic mice. Collectively, these observations help explain why patients with diabetes initiating TGC have worsening of their DR and provide the foundation for clinical studies assessing HIF-inhibition with 32-134D for its prevention.

Oct. 9, 2025: Felice Dunn, Ph.D.

Seminar Title: Detection of Retinal Degeneration by Measuring Reflexive Eye Movements
Time: 3 p.m. via Zoom and in person

Speaker Bio: Felice Dunn began her studies in neuroscience as an undergraduate at Brown University, where she worked with David Berson in the discovery of intrinsically photosensitive ganglion cells. She then continued her studies in vision at the University of Washington with Fred Rieke as a graduate student and with Rachel Wong as a postdoctoral fellow. With Fred Rieke, she examined retinal gain controls that enable vision across a broad range of mean light levels. With Rachel Wong, she uncovered developmental patterns of synapse formation. In between, she studied the fidelity of olfactory processing at the NIH with Mark Stopfer. Currently at UCSF, her lab examines how plasticity can occur in the adult retina. Her lab has developed tools for structural and functional assessment and a novel method of studying loss of primary sensory receptors in the adult retina. Her lab has pioneered unique tools for photoreceptor perturbation to uncover the possibilities for synaptogenesis and functional compensation that could occur or be induced in adult retina. In recent work, her lab is using physiological, anatomical, and computational tools to understand a well-defined visual circuit from the ganglion cells to their behavioral output.

Seminar Summary: Of the dozens of channels coursing from retinal ganglion cells to the rest of the visual system, several are dedicated to non-perceptual functions such as image stabilization. Image stabilization is controlled by both the vestibulo-ocular reflex as well as the optokinetic reflex. I will present work from our lab about the retinal pathways underlying the optokinetic reflex, which is controlled by the accessory optic system. The accessory optic system receives input from specific populations of motion sensitive ON direction selective ganglion cells. In my talk, first I will show how synaptic differences in the ganglion cells tuned for superior vs. inferior motion result in asymmetries at the level of the reflexive eye movement. Next, I will demonstrate that a simple computation across these ganglion cell populations accurately predicts features of eye movements. Finally, I will present how perturbations to retinal circuits influence the behavior. Overall, I would like to convey the advantages of using the accessory optic system, which is relatively simpler than other pathways for conscious visual perception, to understand the relationships between stimulus input, cellular physiology, and a behavioral output.

Nov. 13, 2025: Justin Kumar, Ph.D.

Seminar Title: Interconversion of Compound Eyes and Ocelli Through the Dialing of Transcription Factor Levels
Time: 3 p.m. via Zoom and in person

Speaker Bio: Dr. Justin P. Kumar is a professor in the Department of Biology at Indiana University. His laboratory employs the fruit fly Drosophila melanogaster to investigate how transcription factors, particularly the Pax6 proteins Eyeless and Twin of Eyeless, govern the formation, identity, and interconversion of visual organs such as compound eyes and ocelli. His research has revealed how fine-tuned modulation of gene expression within the retinal determination network can reprogram tissue fates, uncovering fundamental principles of developmental biology and the evolutionary diversification of visual systems. Dr. Kumar has been recognized with numerous honors, including election as a Fellow of the American Association for the Advancement of Science (AAAS) and the Indiana University Trustees Teaching Award. He has published more than 70 scientific papers and serves on the editorial boards of Developmental Biology and Developmental Dynamics.

Seminar Summary: The adult visual system of Drosophila melanogaster is composed of two compound eyes, a pair of extra retinal eyelets, and a trio of simple eyes called ocelli. These distinct eye types have evolved independently from each other for over 400-500 million years and are anatomically distinct, connect to different regions of the brain, and control unique arrays of visual and circadian behaviors. The fate of compound eyes and ocelli is controlled by the activity of the Paired Box-6 (Pax6) transcription factors Eyeless (Ey) and Twin of Eyeless (Toy). Pax6 plays prominent roles in the developing eye of all seeing animals, with strong loss-of-function mutants being associated with microphthalmia or anophthalmia-like phenotypes in humans, mice, zebrafish, and the fruit fly. Pax6 is also characterized by its ability to reprogram the fate of entire cell populations – the forced expression of Ey and Toy can induce the formation of ectopic eyes within non-ocular tissues. Ey and Toy differentially control the fate of the compound eyes and ocelli, with the former specifying the identity of the compound eye and the latter directing the fate of the ocelli. These proteins appear to regulate a common set of downstream targets but activate them at vastly different levels within the two visual organs. This is due to Ey having a much stronger transcriptional activation domain than Toy. Quite surprisingly, simply modulating the levels or activity of Ey/Toy downstream targets induces a dramatic reprogramming of the ocelli into compound eyes. One target gene, sine oculis (so), encodes a DNA binding protein that can function as a transcriptional activator when bound to Eyes Absent (Eya) or a repressor when bound to Groucho (Gro). Our findings suggest that the stoichiometric balance between So-Eya and So-Gro complexes is critical for selecting between the two

different types of visual organ fates. Finally, ratcheting down levels of Ey and Toy (as well as their downstream targets) within the developing compound eye results in the reverse fate change and induces its conversion into ocelli, antennae, maxillary palps, and head epidermis. Our results suggest that adjusting the expression levels of field-level selector genes across a wide threshold range is a viable mechanism for specifying a diversity of cell and organ fates.

Dec. 11, 2025: Shiva Swamynathan, Ph.D.

Seminar Title: Corneal Functions of Apicobasal Polarity-Determinant PARD3
Time: 3 p.m. via Zoom and in person

Speaker Bio: Shiva Swamynathan graduated with a Ph.D. in Life Sciences from the Center for Cellular and Molecular Biology in Hyderabad, India, in 1996. Shiva trained as a post-doctoral fellow with Dr. Joram Piatigorsky at the National Eye Institute, NIH, and worked as an Assistant Professor of Ophthalmology (2007-2015), and Associate Professor of Ophthalmology with Tenure (2015-2023) at the University of Pittsburgh School of Medicine. Shiva joined the Department of Ophthalmology, Morsani College of Medicine, University of South Florida, Tampa in Jan 2023 as a Tenured Professor, where he directs the Ocular Surface Biology Laboratory. Shiva is an Editorial Board Member for 'The Ocular Surface' (2023-Present), a member of the ARVO Professional Development Committee (2024-2027) and a standing member of the Biology and Development of the Eye study section (2024-2028). The research in the Swamynathan laboratory, supported by NIH grants RO1 EY026533 and RO1 EY031684, is focused on elucidating the ocular surface functions of: (i) Krüppel-like transcription factors KLF4 and KLF5; (ii) immunomodulatory molecule SLURP1; and (iii) apicobasal polarity-determinant PARD3.

Seminar Summary: Shiva Swamynathan will discuss the corneal epithelial functions of PARD3, a key determinant of epithelial apicobasal polarity. The Swamynathan lab used a mouse model with cornea-specific ablation of Pard3 and a complementary human corneal epithelial cell culture model with CRISPR-mediated mutation in PARD3 to discover that PARD3 ablation disrupts corneal epithelial apicobasal polarity, barrier function, and actin cytoskeleton, while elevating cell proliferation and expression of EMT transcription factors. These findings will be presented along with more recent results that suggest involvement of PARD3 in regulating corneal sensory innervation and genome stability.