



## Franklin-Prasher-Kimmel (FPK)

### Webinar Series 2026

Organized by the Young Researcher Committee (YoRC)  
European Zebrafish Society (EzS) & International Zebrafish Society (IZFS)

Generously supported by the ZDMS Early Career Investigator (ECI) Committee

**Dates:**

April 23 • June 25 • September 24 • November 19, 2026

# PROGRAMME

## April 23, 2026

- **Joaquín Navajas Acedo**  
*Postdoctoral Researcher – Biozentrum*  
*“Spatiotemporal emergence of somatosensory neuron diversity”*
- **Ece Atayeter**  
*PhD Student – University of Texas at Austin*  
*“Cilia.io: a machine learning approach reveal spatial patterns of cilia beating dynamics”*

## June 25, 2026

- **Wei Qin**  
*Research Fellow – Oklahoma Medical Research Foundation*  
*“The Umax Platform: A High-Efficiency Base Editing Toolkit for Rapid Functional Assessment of Genetic Variants in Zebrafish”*
- **Stefan Choy**  
*PhD Student – Lehigh University*  
*“Identifying the genetic architecture underlying the evolution of stress response in blind Mexican cavefish”*

## September 24, 2026

- **Shuyu (Iris) Zhu**  
*Postdoctoral Researcher – Washington University in Saint Louis*  
*“The effect of competing prey on neural and behavioral responses in larval zebrafish”*
- **Samudra Gupta**  
*PhD Student – S.N. Pradhan Centre for Neurosciences, University of Calcutta*  
*“Decoding epigenetic dynamics regulating neural progenitor cell proliferation during spinal cord regeneration in zebrafish”*

## November 19, 2026

- **Bruna Figueiredo Costa**  
*Postdoctoral Researcher – Champalimaud Foundation*  
*“The zAvatar model as a surrogate for patient response to chemotherapy and metastatic potential in colorectal cancer”*
- **Cassia Michael**  
*PhD Student – Albert Einstein College of Medicine*  
*“Deciphering the Mechanisms that Regulate Neutrophil Migration and Prioritization in Polytraumatic Injury”*



with the generous support of the ZDMS ECI Committee

# Franklin-Prasher-Kimmel Series 2026

April 23 • June 25 • September 24 • November 19



**Joaquín Navajas Acedo**  
Postdoctoral Researcher  
Biozentrum  
Switzerland



**Ece Atayeter**  
PhD Student  
University of Texas at Austin  
USA



**Stefan Choy**  
PhD Student  
Lehigh University  
USA



**Wei Qin**  
Research Fellow  
Oklahoma Medical Research Foundation  
USA



**Shuyu (Iris) Zhu**  
Postdoctoral Researcher  
Washington University  
USA



**Samudra Gupta**  
PhD Student  
University of Calcutta  
India



**Cassia Michael**  
PhD Student  
Albert Einstein College of Medicine  
USA



**Bruna Figueiredo Costa**  
Postdoctoral Researcher  
Champalimaud Foundation  
Portugal

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# SPEAKERS, BIOGRAPHIES & ABSTRACTS

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## Joaquín Navajas Acedo

**Affiliation:** Biozentrum at the University of Basel, Switzerland

**Title:** *Spatiotemporal emergence of somatosensory neuron diversity*

**Short-bio:**

*Originally from Spain, Joaquín obtained his PhD in Biology at the Stowers Institute for Medical Research in Kansas City MO, USA. In the laboratory of Dr. Piotrowski, he focused on dissecting the role that two signaling pathways -Wnt and Planar Cell Polarity- have during the development of the lateral line in zebrafish, using a combination of mutant analysis, live imaging, immunostaining, and more.*

*Currently, he is a postdoc at the Schier lab at the Biozentrum of the University of Basel, Switzerland, where he wants to answer a fundamental question in biology: what are the mechanisms behind the acquisition of the different layers of neuron diversity? A developmental biologist by training, Joaquín's work combines old-school and cutting-edge techniques to study Rohon-Beard neurons, a population of zebrafish spinal cord neurons that for around 150 years were thought to disappear during early development but Joaquín discovered remain until at least juvenile stages.*

*Joaquín hopes to become an independent group leader to pursue this and other questions regarding sensory neuron diversity, lineage tracing, evolution, and more.*

*Abstract not included.*

# Ece Atayeter

**Affiliation:** University of Texas at Austin, USA

**Title:** *Cilia.io: a machine learning approach reveal spatial patterns of cilia beating dynamics*

## **Short-bio:**

*Ece Atayeter is a PhD student in the Cellular and Molecular Biology program at The University of Texas at Austin, under the mentorship of Ryan S. Gray and John B. Wallingford. Ece's research focuses on motile ciliated cells and their underlying fluid dynamics, alongside development of new machine learning-based tools for measuring these structures.*

## **Abstract:**

Motile cilia play a crucial role in tissue physiology and function by generating directional fluid flow. While pronounced ciliary motility defects are well established in ciliopathies, how subtle changes in beating dynamics contribute to disease phenotypes remains unresolved. Precise and quantitative characterization of ciliary motion is therefore essential, yet current approaches are limited by the inherent challenges of measuring cilia morphology and dynamics in vivo. Existing quantification tools rely on coarse-grained models and are largely optimized for in vitro imaging under controlled background and signal levels, restricting their applicability for live samples. To address these limitations, we developed Cilia.io, an automatized machine-learning based tool that accurately detects, segments, and quantifies cilia morphology and dynamics from live confocal images. Using Cilia.io, we uncovered previously unrecognized regional differences in ciliary waveforms within the wild-type zebrafish spinal cord. Furthermore, analysis of the cilia mutant, *bbs2*, revealed spatially restricted defects of motile cilia beating patterns: dorsal spinal cord cilia exhibited altered dynamics, whereas ventral cilia remained largely unaffected. Together, these findings highlight the importance of accurate cilia quantification tools for detecting subtle but important ciliary defects that may underlie cilia-driven disorders.

# Wei Qin

**Affiliation:** Oklahoma Medical Research Foundation, USA

**Title:** *The Umax Platform: A High-Efficiency Base Editing Toolkit for Rapid Functional Assessment of Genetic Variants in Zebrafish*

## **Short-bio:**

*Wei Qin, PhD, is a Research Fellow in the Genes and Human Disease Research Program at the Oklahoma Medical Research Foundation. He is a genome editor and zebrafish geneticist with over 10 years of experience in developing CRISPR-based base editors and in vivo functional genomics platforms for human disease modeling. His research focuses on improving the precision, efficiency, and targeting scope of base editing in zebrafish, including the development of PAM-flexible and TadA-derived base editors, as well as rapid F0 platforms for functional evaluation of disease-associated variants.*

*Dr. Qin has published as first, corresponding, or co-corresponding author in journals including Nature Biomedical Engineering, Nature Communications, Advanced Science, Cell Death and Disease, and BMC Biology, and has led or contributed to multiple funded projects in genome editing and rare disease research.*

## **Abstract:**

Many missense mutations identified in clinical genetic testing are categorized as variants of uncertain significance (VUS), creating a pressing need for systematic functional classification. While CRISPR-mediated base editing offers a precise way to model these variants in vivo, current editors in zebrafish face significant constraints regarding efficiency, PAM compatibility, and unintended bystander mutations. We developed and characterized the "Ultramax" (Umax) suite, a comprehensive family of TadA-derived cytosine (TCBE-Umax) and adenine (ABE-Umax) base editors optimized for high-performance zebrafish research. By engineering the TadA deaminase domain and diversifying the Cas9 architecture, we significantly improved editing efficiency and reduced sequence context bias. The Umax suite offers a versatile toolkit with shifted, narrowed, or broadened editing windows and expanded PAM compatibility, while maintaining low rates of indel formation. Notably, these editors achieve efficient biallelic editing in F0 founders, enabling rapid, high-throughput functional assessment of genetic variants without the need for multi-generational breeding. As a proof of concept, we utilized the Umax platform to evaluate 15 VUS linked to hereditary hearing loss, successfully determining their pathogenicity through in vivo phenotypic analysis. Our results demonstrate that the Umax suite provides a powerful and versatile platform for studying genetic variants and accelerating disease modeling in zebrafish.

# Stefan Choy

**Affiliation:** Lehigh University, USA

**Title:** *Identifying the genetic architecture underlying the evolution of stress response in blind Mexican cavefish*

**Short-bio:**

*I am a 4th-year PhD candidate in the Kowalko lab at Lehigh University. I am broadly interested in the molecular underpinnings that drive differences in behaviors. Currently, I study the genetic of behaviors in *Astyanax mexicanus*, a fish species that is comprised of surface fish and blind cavefish. After graduate school, I aim to continue in academia as a postdoctoral fellow and ideally continue investigating the mechanisms underlying behavior!*

**Abstract:**

Animals perform a variety of behaviors in response to encountering stressful stimuli. Despite the wide range of behaviors, many of the molecules, hormones, and brain regions underlying stress responsiveness are largely conserved across species; however, the genetic factors contributing to the evolution of stress responsiveness and whether certain genetic mechanisms are repeatedly targeted via evolution of similar shifts in response to stress are not well understood. To investigate the genetic basis of the evolution of stress response, we utilize *Astyanax mexicanus*, the Mexican tetra. *A. mexicanus* is a small, freshwater fish with multiple, interfertile populations of riverine surface fish and over 35 populations of cavefish. In addition to many common cave-adapted traits like eye degeneration and albinism, multiple *A. mexicanus* populations have evolved a reduced response to stressors. Here, we quantified stress-associated behaviors in cave-surface F2 hybrids derived from multiple populations of cavefish and performed quantitative trait loci mapping. We identified the genetic architecture underlying the evolved reductions in stress response in *A. mexicanus* cave populations, as well as candidate genes for evolution of stress responsiveness. Further, we have begun to quantify stress response in surface fish with CRISPR-Cas9 induced mutations in candidate stress responsive genes. Together, these experiments increase our understanding of how complex traits repeatedly evolve, and provide future genetic targets for gene editing to further understand how changes to stress have evolved.

# Samudra Gupta

**Affiliation:** S.N. Pradhan Centre for Neurosciences, University of Calcutta, India

**Title:** *Decoding epigenetic dynamics regulating neural progenitor cell proliferation during spinal cord regeneration in zebrafish*

## **Short-bio:**

*Research Scholar investigating the epigenetic regulation of spinal cord regeneration after injury in zebrafish, with a focus on neural progenitor cell proliferation and axonal regrowth and subsequent reformation of neural circuit through post-regenerative synapse formation, developing a predictive animal model to assess the spinal cord repair in human and predicting probable translational strategies towards the regenerative niche formation after spinal cord injury in human.*

## **Abstract:**

Spinal cord injury (SCI) in humans leads to permanent sensory and motor impairments, but adult zebrafish exhibit remarkable regenerative abilities through the injury-induced proliferation of CNS-resident neural progenitor cells (NPCs) that generate functional neurons at the injury site. In zebrafish, SCI is characterized by epigenetic changes, offering critical insights for deciphering potential therapies for enhancing SCI recovery. In this scenario, we investigated the role of Sirtuin1 (Sirt1), a non-classical histone deacetylase, in promoting NPC proliferation and axonal regrowth following SCI in zebrafish by using whole genome MeDIP sequencing and observed that Sirt1 plays a crucial role in NPC proliferation and induces glial bridging during spinal cord regeneration. We demonstrate that Sirt1 regulates the HIPPO pathway by deacetylating and inactivating Dnmt1, leading to the hypomethylation of the yap1 promoter which subsequently induces Ctgfa expression, driving NPC proliferation and axonal regrowth following SCI. Subsequently, we generated a comprehensive miRNA library by Next generation Sequencing and selected the most significant stable novel miRNA for further study to validate the role of novel miRNA behind NPC proliferation. In-silico target prediction and functional validation revealed a unique interaction of the novel miRNA to a member of the zebrafish odorant receptor family. To our knowledge, this is the first study to demonstrate a close association of miRNA to the regulation of the 'ectopic' expression of odorant receptors in the spinal cord which may represent a critical determinant of the fate of the NPCs, priming these cells to undergo proliferation in response to SCI. In conclusion, our findings uncover two major epigenetic dynamics of during SC regeneration in zebrafish where miRNA modulates the early proliferative fate of NPCs and a novel interaction among Sirt1-Dnmt1-Yap1 modulating the DNA methylation and regulates HIPPO pathway-mediated Ctgfa expression to induce NPC proliferation for subsequent glial bridge formation and axonal regeneration.

# Shuyu (Iris) Zhu

**Affiliation:** Washington University in St. Louis, USA

**Title:** *The effect of competing prey on neural and behavioral responses in larval zebrafish*

## **Short-bio:**

*I am a senior scientist in the Goodhill Lab with a research focus on the neural basis of behavior in larval zebrafish. Combining behavioral imaging, whole-brain imaging, and quantitative computational analysis, I investigate how the brain processes sensory information to guide behavior under external challenges, such as competing sensory inputs, as well as internal challenges, including those linked to neurodevelopmental disorders. More broadly, I am interested in using zebrafish as a model to uncover circuit principles by which sensory coding propagates through brain-wide networks to shape behavior.*

## **Abstract:**

Resolving competition between simultaneously available stimuli is a fundamental problem for brains engaged in action selection. Prey hunting provides a natural context for studying this problem at the level of whole-brain dynamics, as animals must select a single target from competing sensory inputs to generate a coherent behavioral response. Here we use free-swimming hunting behavior and 2-photon neural imaging in zebrafish larvae in response to prey-like stimuli to investigate the role of multiple brain regions in resolving sensory competition during prey selection. When fish encountered competing prey, capture success was reduced and initial orienting turns were altered compared to the single-stimulus case, indicating impaired target selection. Brain-wide calcium imaging revealed widespread suppression of target-evoked activity under competition and altered network structure. The strongest effects were found in the habenula and nucleus isthmi which showed the greatest separation of population trajectories for competition and the highest decoding accuracy for stimulus context. Habenula activity preceded choice-related signals elsewhere in the brain, and targeted ablation impaired hunting and attenuated competition-dependent representational differences. By linking behavior, whole-brain dynamics, and causal perturbation in a freely moving animal, our findings provide a systems-level account of how vertebrate brains transform competing sensory inputs into coherent, goal-directed actions.

# Bruna Costa

**Affiliation:** Champalimaud Foundation, Portugal

**Title:** *The zAvatar model as a surrogate for patient response to chemotherapy and metastatic potential in colorectal cancer*

## **Short-bio:**

*I am a cancer researcher specializing in translational oncology. I am currently a senior postdoctoral researcher in the Fior Lab at the Champalimaud Foundation (Portugal). My work focuses on zebrafish patient-derived xenografts, colorectal cancer biology, therapy screening, and radiation oncology. My research has been recognized through competitive fellowships and awards, including the Raquel Seruca Prize (2024) and the Pfizer Award in Clinical Research (2025).*

## **Abstract:**

We conducted a clinical study to validate the zebrafish patient-derived xenograft model (zAvatar) as a fast predictive platform for personalized treatment in colorectal cancer. By individually comparing the clinical responses of 55 patients with their zAvatar-test, we developed a decision tree model integrating tumor stage, zAvatar-apoptosis, and zAvatar-metastatic potential. This model accurately forecasted patient progression with 91% accuracy (Costa et al, Nat Commun 2024). Beyond treatment response, zAvatars derived from patients who later developed disease progression showed a significantly higher incidence of micrometastases. In stage III tumors, absence of micrometastases in zAvatars was sufficient to identify non-progressive patients, whereas the presence of micrometastases indicated that clinical outcome depends on the efficacy of adjuvant chemotherapy. These findings suggest that the zAvatar model captures the intrinsic metastatic potential of patient tumors. To elucidate the molecular basis underlying this phenotype, we performed RNA sequencing on stage III CRC tumors and identified a metastatic gene signature composed of five overexpressed genes associated with micrometastatic behavior in the zAvatar model (unpublished). Together, these results position the zAvatar model as a dual functional platform capable of predicting chemotherapy response and revealing early metastatic potential, offering a powerful strategy for personalized therapy and improved patient stratification in CRC.

# Cassia Michael

**Affiliation:** Albert Einstein College of Medicine, USA

**Title:** *Deciphering the Mechanisms that Regulate Neutrophil Migration and Prioritization in Polytraumatic Injury*

**Short-bio:**

*My name is Cassia, and I am a fourth-year PhD student in the laboratory of Sofia de Oliveira at Albert Einstein College of Medicine in New York City. When I'm not in the lab, I love exploring museums and trying new restaurants around the city with friends.*

**Abstract:**

Traumatic injuries are the leading cause of death for individuals under the age of 50, and a significant subset of patients suffer from polytraumatic injury involving multiple organs. While advances in emergency medicine and surgical intervention have dramatically improved survival after severe trauma, many patients experience prolonged recovery and chronic complications. This burden is amplified in modern patient populations, where rising rates of obesity, diabetes, and cardiovascular disease create a background of chronic low-grade inflammation that predisposes individuals to immune-driven complications. Neutrophils, the most abundant leukocytes in the peripheral blood, rapidly migrate toward sites of injury to initiate defense and repair. Although neutrophil dysregulation in trauma has been broadly studied, how neutrophils immediately interpret and prioritize multiple, spatially distinct injuries in vivo across organs remains unknown, largely due to technical limitations in capturing these events at the whole-animal scale. To address this, we developed zebrafish polytrauma models enabling real-time, whole animal imaging of neutrophil migration and behavior. We discovered a previously unrecognized mechanism that regulates immune homeostasis after complex injury, in which neutrophils prioritize signals from anatomically and physiologically vital organs (e.g., liver and spinal cord) over simpler epithelial injuries. This prioritization is not dictated by proximity to specific hematopoietic niches or neutrophil maturation state. Furthermore, neutrophils display distinct behavioral signatures, including differences in speed, volume, and directionality, when responding to polytrauma versus single injuries. We are integrating single-cell RNA-sequencing and other omic approaches to define the transcriptional programs underlying neutrophil prioritization and organ-specific behaviors. Finally, we show that diet-induced meta-inflammation disrupts neutrophil prioritization mechanisms and significantly reduces survival, particularly following LPS-induced sepsis challenge, mirroring high-risk clinical trajectories. Together, these findings reveal that neutrophils deploy a context-dependent, organ-specific response program during polytrauma and highlight zebrafish as a powerful platform to dissect systemic inflammatory dynamics relevant to trauma pathology and outcomes.