



We had a very exciting BACLIF meeting on April 8. We had 19 professors from Buck Institute, UC Davis, UC Berkeley, UCSF, Stanford, UC Santa Cruz, and UC Merced attended the talks (16 people in person and 3 via Zoom). The talk covers diverse topics with good amount of background introduction aiming to help colleagues from different fields to understand and cultivate potential cross-disciplinary collaborations, which is a key purpose of BACLIF meetings. The BACLIF meeting encourages questions and discussions, and that's why each talk always went overtime and every member enjoyed the meeting more than just listening to some talks.

We had 14 members attend the dinner and discuss issues related to science, policy, grants, and mentorship, etc.

Time:

Saturday April 8, 2023 from 2-5pm (for the talks/meeting), with dinner afterwards.

Speakers:

2:00 – 2:30: Bin Chen, "Cell fate specification in the developing cerebral cortex"

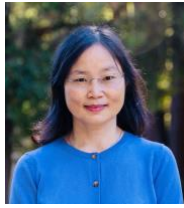
2:30 – 3:00: Xuecai Ge, "Understanding cell signaling in the cilium of the developing brain" 3:00 – 3:30: Break/discussion

3:30 – 4:00: Yadong Huang, "Neuronal ApoE4 and Alzheimer's

Diseases" 4:00 – 4:30: Qili Liu, "Programming the Protein Intake

Setpoint in Drosophila" 4:30 – 5:00: Break/discussion

Bin Chen, UC Santa Cruz



Title: Cell fate specification in the developing cerebral cortex

Bio:

Bin Chen is a professor in the department of Molecular, Cell and Developmental Biology at UC Santa Cruz. She received her Bachelor's degree in Cell Biology and Genetics, and her Master's degree in Cell Biology from Beijing University. She received a Ph.D. degree in Cellular and Molecular Pharmacology from University of Stony Brook. She received her postdoctoral training in the laboratory of Susan McConnell at Stanford University. Dr. Chen's laboratory investigated neural stem cell lineage and neuronal cell fate specification in the developing cerebral cortex.

Abstract:

The cerebral cortex is critical for cognition, perception, emotion, and complex behaviors. Glutamatergic projection neurons comprise the majority of cortical neurons and occupy a central position in neural circuits, serving both as the principal input units and the sole output system.

Therefore, proper development and function of these neurons is essential for normal brain functions. I will talk about the molecular mechanisms underlying the generation of different cortical projection neuron subtypes and how mutations associated with neurodevelopmental diseases affect their development.

Xuecai Ge, UC Merced



Title: Toward understanding of cell signaling in the cilium in the developing brain

Bio:

Xuecai Ge is an assistant professor in the department of Molecular and Cell Biology at University of California Merced. She studied embryonic neurogenesis during her PhD training at Harvard University, where her work revealed how human mutations in psychiatric disorders impact neuronal production in the brain. Her postdoctoral research at Stanford University focused on the regulation of Hedgehog signaling, the first signaling pathway found to rely on the primary cilium. At UC Merced, her

lab studies the fundamental mechanisms of cell signaling in the primary cilia and how signaling errors lead to developmental disorders, such as birth defects and pediatric brain tumor. Research from her lab provides unique insights into the regulatory mechanisms of signaling pathways in development and diseases.

Yadong Huang, UCSF



Title: Neuronal ApoE4 and Alzheimer's Diseases

Bio:

Yadong Huang, MD, PhD, is a senior investigator at the Gladstone Institutes, where he is also the director of the Center for Translational Advancement and an investigator in the Roddenberry Stem Cell Center. In addition, he is a professor of neurology and pathology at UC San Francisco.

Dr. Huang earned an MD from Qingdao Medical University in China, and a PhD in biochemistry and pathology from Peking Union Medical College and Chinese Academy of Medical Sciences in Beijing. He was trained as a postdoctoral fellow at the Arteriosclerosis Research Institute at the University of Muenster, Germany. Huang joined Gladstone Institutes in 1995 as a postdoctoral fellow, and became a staff research investigator in 1999. In 2015, he was promoted to senior investigator.

Abstract:

Dr. Huang and his team focus on the causes and progression of Alzheimer's disease. Specifically, they study a variant of apolipoprotein E, called apolipoprotein E4 (apoE4). Approximately 60–75 percent of Alzheimer's patients carry the apoE4 variant, making it the most important genetic risk factor for Alzheimer's disease. The team uses mouse models and induced pluripotent stem (iPS) cells made from skin cells of patients carrying apoE4 or other mutations related to Alzheimer's to study their effects on the development, survival, and degeneration of neurons and glial cells. In addition, Huang's lab is working to identify drug targets, develop therapeutic strategies, and repurpose existing drugs for Alzheimer's disease and other neurodegenerative disorders.

Qili Liu, UCSF



Title: Programming the Protein Intake Setpoint in *Drosophila*

Bio:

I got my bachelor's degree in biology from Beijing Normal University in 2005 and then attended graduate school at Shanghai Institute of Physiology and Ecology, Chinese Academy of Sciences. In graduate school I identified molecules required for maintaining stem cell competence in *Arabidopsis* and obtained a PhD degree in genetics in 2010. I then carried out my postdoctoral work in the laboratory of Dr. Mark Wu at the Johns Hopkins University, where I dissected and characterized key circuit mechanisms underlying two different motivated behaviors: sleep and protein feeding. I joined UCSF faculty in 2019. Employing a multidisciplinary approach including large-scale genetic and behavioral analyses, immunohistochemistry, functional imaging, and patch-clamp electrophysiology, my lab seeks to understand the fundamental principles underlying the organization and modulation of motivated behaviors, including the determination of homeostatic setpoint.

Abstract:

All animals share motivated behaviors to fulfill their basic needs for survival, including food, water, sleep, and social interactions, etc. The homeostatic regulatory system energizes behaviors to defend a target level for these needs (the homeostatic setpoint). What defines the homeostatic setpoint and how is it modulated remain unanswered questions for all motivated behaviors. Protein is a crucially important macronutrient, and behavioral studies indicate that a wide range of species, including humans, seek to consume a fixed amount of protein: the protein intake setpoint. In *Drosophila* fruit flies, protein consumption is different in males, virgin female and mated female flies, due to the differences in protein needs for protein in egg production, thus provides an elegant model to study the homeostatic setpoint for motivated behaviors. We have previously identified a group of dopamine neurons named DA-WED in *Drosophila* that encode protein-specific hunger. Here, we tested the hypothesis that the protein intake setpoint is encoded in the hunger neurons by the resting membrane potential (RMP). We identified two GPCR signaling pathways, FMRFa-FMRFaR-PKC and MS-MSR2- PKA, that modulate the RMP of DA-WED neurons in opposite directions and program the protein intake setpoint. Identification of FMRFa+ and MS+ neurons that act upstream of DA-WED neurons revealed the dynamics of these signaling pathways in male, virgin female and mated female flies.

