

We had a very exciting BACLIF meeting on June 3rd. This is usually a hard time to gather a lot of PIs because of their summer travelss and family vacations. We had about 13 professors from Buck Institute, UC Davis, UCSF, Stanford, and UC Santa Cruz who attended the talks in person or via Zoom. The talks covered diverse topics with a good amount of background introduction aimed helping colleagues from different fields to understand and cultivate potential cross-disciplinary collaborations. The meeting started went 2pm until 5:30pm with a lot of exchanges and discussions The BACLIF meeting encourages questions and discussions, one reason that our talks often go overtime. All in all, we had had seven members attend dinner at Tai Pan and discuss issues related to science, policy, mentorship, etc.

Time:

Saturday June 3rd, 2023 from 2-5pm (for the presenations), with dinner afterwards.

Location:

TopAlliance Biosciences at Meno Park Labs (1440 O'Brien Drive, Suite A-1, Menlo Park, CA 94025)

Speakers:

Each speaker had have 25+5 minutes.

Xinnan Wang (Stanford)	Why do we care about
Biao Wang (UCSF)	TBD
Break/discussion AND relaxin	ng wine tasting
Xiaoke chen (Stanford)	Circuitry and Molecular
Descending Pain Facilitation	-
Xianhua Piao (UCSF)	Microglial mechanism of
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Break/discussion	
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Dr. Xinnan Wang, Stanford



Talk title: Why do we care about mitochondria?

Intro:

Mitochondria are vital organelles with distinct morphological features and functional properties. The dynamic network of mitochondria undergoes structural and functional adaptations in response to cell-type-specific metabolic demands. Mitochondrial heterogeneity supports unique subcellular functions and is crucial to polarized cells, such as neurons. The spatiotemporal metabolic burden within the complex shape of a neuron requires precisely localized energy metabolism. In this talk, I will explore the significance of the multifaceted roles of mitochondria to brain function and disease.

Short Bio:

Dr. Xinnan Wang obtained a PhD in genetics and neuroscience at University of Cambridge and conducted postdoctoral training at Harvard Medical School before she started the faculty position at Stanford University. Dr. Wang's lab focuses on the molecular mechanisms underlying mitochondrial dynamics and clearance in neurons and how even subtle disturbances of those processes may contribute to neurodegeneration. Dr. Wang's lab linked Miro and the mitophagy pathway to Parkinson's disease pathogenesis and discovered the Miro impairment as a convergent theme in PD patients.

Dr. Biao Wang, UCSF



Title: Immunometabolism in adipose tissue

Abstract: The innate immunity by type I interferon pathway orchestrates viral clearance through two-phase responses in triggers (cells sensing viral materials and producing interferon) and amplifiers (interferon-activated immune cells producing more interferon and anti-viral proteins). RNA specific adenosine deaminases (ADARs) suppress the interferon-stimulated gene (ISG) program and innate immunity. In this project, we investigate how Adar1 regulates adipocyte maintenance and insulin sensitivity in adipose tissues, potentially linking common risk variants from genome-wide associated studies to adipocyte dysfunction and metabolic diseases.

Short Bio: Dr. Biao Wang received his bachelor's degree from Wuhan University (1995-1999) in Wuhan, and Ph.D. degree from Institute of Biochemistry and cell biology (1999-2004) in Shanghai. Dr. Wang went to the United States for his postdoctoral trainings at University of Minnesota (2005-2006) and Salk Institutes for Biological Sciences (2006-2102). During this period, Dr. Wang used Drosophila as the genetic model system to interrogate the molecular mechanisms controlling energy metabolism. Dr. Wang became Assistant Professor (2013-2020) and Associate Professor (2020-now) at University of California San Francisco, continuing studies of adipose tissue metabolism. The research projects in Dr. Wang's laboratory are funded by NIDDK/NIH.

Dr. Xiaoke Chen, Stanford



Title: Circuitry and Molecular Mechanisms for Descending Pain Facilitation

abstract: Spinal cord-projecting neurons in the rostral ventromedial medulla (RVM^{SC} neurons) play active roles in pain facilitation. However, the underlying circuitry and molecular mechanisms remain largely unknown. We show that acute activation of OPRM1⁺ RVM^{SC} neurons does not facilitate pain in normal mice, but activity of these neurons is required for both initiation and maintenance of chronic mechanical hypersensitivity in mouse models of inflammatory and neuropathic pain. Excitatory collicular inputs are essential for upregulating pseudokinase CaMKv in the OPRM1⁺ RVM^{SC} neurons after nerve injury and causing mechanical hypersensitivity. Up- or down- regulation of CaMKv is sufficient to drive or reverse mechanical hypersensitivity. Together, our results reveal a collicular-medulla-spinal cord pathway that drives persistent pain and substantiate CaMKv as a key molecular determinant of chronic mechanical hypersensitivity.

Short Biography:

Xiaoke Chen is an associate professor in the Biology Department at Stanford University. He got his PhD. in 2005 at the Institute of Neuroscience, Chinese Academy of Sciences, where he studied the kinetics of vesicle fusion in neuron and astrocyte. From 2006-2012, he did postdoc with Dr. Charles Zuker in UC San Diego and Columbia University, to study the neurobiology of taste. Currently, his lab at Stanford is trying to understand how brain circuits mediate motivated behaviors and how maladaptive change in these circuits lead to chronic pain, addiction and depression.

