

MONDAY
February 10, 2020

PHYSIOLOGY FACULTY CANDIDATE SYMPOSIUM

Luskin Conference Center
8:30 AM – 4:10 PM

SYMPOSIUM SCHEDULE

START	END	DESCRIPTION	LOCATION
8:30 AM	9:00 AM	Mingling with Candidates	Nutrition Hub
9:00 AM	9:10 AM	Welcome	Illumination Room
9:10 AM	10:00 AM	Ambre Bertholet, Ph.D.	Illumination Room
10:00 AM	10:50 AM	Bhagirath Chaurasia, Ph.D.	Illumination Room
10:50 AM	11:10 AM	Break	Nutrition Hub
11:10 AM	12:00 PM	William Giardino, Ph.D.	Illumination Room
12:00 PM	1:30 PM	Break for lunch	
1:30 PM	2:20 PM	Mary Mohrin, Ph.D.	Illumination Room
2:20 PM	3:10 PM	Hume Stroud, Ph.D.	Illumination Room
3:10 PM	4:00 PM	Weizheng Zeng, Ph.D.	Illumination Room
4:00 PM	4:10 PM	Conclusion	Illumination Room

AMBRE BERTHOLET

Instructor, UC San Francisco

“Controlling Mitochondrial Thermogenesis to Combat Metabolic Syndrome”

Mitochondrial thermogenesis (also referred to as mitochondrial uncoupling) is one of the most promising targets for increasing energy expenditure to reduce fat deposition and combat metabolic syndrome. Mitochondrial thermogenesis results from H⁺ leak across the inner mitochondrial membrane, leading to uncoupling of substrate oxidation and ATP synthesis with consequent production of heat. By employing the novel mitochondrial patch-clamp assay combined with modern cellular and molecular techniques, my future research projects will provide new insights into the mechanisms that control the thermogenic ability of mitochondria and how they can be targeted to combat metabolic syndrome.



BHAGIRATH CHAURASIA

Research Assistant Professor, University of Utah

“Ceramide Induced Lipotoxicity in Metabolic Diseases”

The ectopic deposition of lipid molecules in tissues not suited for fat storage drives the tissue dysfunction that underlies diabetes and heart disease. Of the numerous lipid subtypes that accumulate, ceramides and dihydroceramides, show particularly tight associations with these metabolic disorders. Studies in rodents further suggest that these sphingolipids play causative roles in the pathologies. The presentation will review the history of research on these enigmatic molecules, exploring (a) their mechanism of action, (b) the evolutionary pressures that gave them their unique attributes, and (c) the potential of ceramide-reduction therapies as treatments for cardiometabolic disease.



WILLIAM GIARDINO

Instructor, Stanford University

“Neurocircuitry of Emotion: Molecular Mechanisms for Maintaining Homeostasis in Behavioral Arousal”

Sleep is essential for optimal physiological function, and major features of wakefulness are governed by Hypocretin (Hcrt), a key arousal neuropeptide. I recently characterized synaptic and functional connectivity between Hcrt neurons and the bed nuclei of stria terminalis (BNST; a heterogeneous region of the “extended amygdala”), providing a model for BNST dysregulation in sleep/wake disruption, anxiety, and addiction. Next, I developed a circuit-specific CRISPR/Cas9 gene editing system, revealing a mechanism driving BNST-Hcrt interactions in excessive binge alcohol drinking. My laboratory will focus on BNST subcircuits underlying addiction-related sleep disturbances, sex differences in stress and reward, and emotional arousal in narcolepsy.



MARY MOHRIN

Scientist, Calico Labs

“Linking Longevity Regulators and Stem Cells of the Immune System”

Hematopoietic stem cell (HSC) transplantation (HSCT) cures blood and immune system defects, but also has the potential to treat cardiovascular disease, cancer, and HIV. Despite HSCT’s promise, most patients who would benefit from HSCT –or other cell-based therapies like cancer immunotherapy– are of old age and their cells are unresponsive to engineering and transplantation. To improve access to HSCT, I study two aspects of adult stem cell (ASC) biology: (1) how long-lived ASCs like HSCs and mesenchymal stromal cells protect themselves, so we can exploit those mechanisms and (2) how things go wrong with age in ASCs, so we can reverse those changes.



HUME STROUD

Postdoctoral Fellow, Harvard Medical School

“Neuronal Dna Methylation in Brain Development and Disease”

Much of brain development occurs after birth, but the molecular mechanisms that orchestrate this intricate developmental process are unclear. I found that in the brain during early life, the DNA methyltransferase DNMT3A transiently binds the neuronal genome, and its binding specifies the pattern of DNA methylation at CA sequences (mCA). Once deposited, mCA functions in a rheostat-like manner to fine-tune the cell-type-specific transcription of genes that are critical for brain function. In continuing studies, I am investigating the mechanisms that control the mCA pathway, and exploring how disruption of neuron-specific DNA methylation leads to neurological disorders.



WEIZHENG ZENG

Scientist, Janssen Pharmaceuticals

“Molecules and Cells for Sensing Blood Pressure and Heartbeat”

Blood pressure is tightly regulated to ensure the body is ready for daily activity. Although it has been known for decades that baroreflex is a homeostatic loop to sense and compensate blood pressure changes, the identity of baroreceptor mechanosensitivity remains unknown. I discovered that mechanically activated ion channels PIEZO1 and PIEZO2 are the long-sought baroreceptor mechanosensors. In today’s talk, I will review the discovery of Piezo channels and then introduce my study on exploring their physiology roles. By the end, I will present my plan and share some exciting preliminary data with you.

