Neuroscience Seminar Series schedule

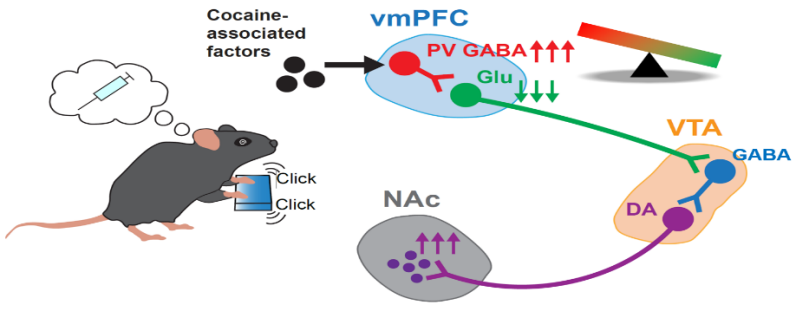
All seminars are currently scheduled Tuesdays from 4-5 pm mountain time. All speakers will be in-person unless noted.

Fall 2025

September 9th, 2025 – Dr. Byungkook Lim
Professor, Neurobiology, Division of Biological Sciences, University of California at San Diego, CA

Title: **Distinct Interneuronal Dynamics Selectively Gate Target-Specific Cortical Projections in Drug Seeking**

Abstract: Drug craving persists after prolonged abstinence, posing a major challenge in treating substance use disorders. The ventral medial prefrontal cortex (vmPFC) plays a critical role in impulsivity and decision-making, making it a promising target for mitigating drug-craving by orchestrating downstream brain-wide activity. However, the dynamics of vmPFC sub-circuits during the progression of drug addiction remain unclear. Here, we uncover a circuit-level mechanism by which distinct vmPFC sub-circuits, defined by cell-type-specific interneurons and projection-specific cortical outputs, differentially modulate mesolimbic pathways to drive drug-seeking behavior. Our results reveal that distinct interneuron subtypes display unique activity dynamics and exert selective modulation over projection-specific cortical outputs. Notably, Parvalbumin-positive (PV) interneurons exhibit target-specific synaptic remodeling with pyramidal neurons projecting to distinct downstream targets, which is crucial for modulating mesolimbic circuits and driving persistent cocaine seeking after abstinence. These findings provide compelling insights into vmPFC microcircuit mechanisms underlying substance use disorders.



September 23th, 2025 – Dr. Todd Sacktor
Distinguished Professor of Physiology & Pharmacology, Anesthesiology and Neurology,
State University of New York (SUNY) Downstate Medical Center, NY

Title: **LTP and memory maintained by persistent synaptic tagging of PKMzeta**

Abstract: Does the molecular mechanism maintaining synaptic long-term potentiation (LTP) sustain long-term memory? The induction of both late-phase LTP and long-term memory requires new protein synthesis. During LTP, the newly synthesized proteins that potentiate synaptic transmission selectively target active synapses by synaptic tagging. The proteins in synapses, however, turn over and are replaced through new synthesis and trafficking, raising the question of how potentiation of specific activated synapses is maintained over time. We propose that a mechanism of “persistent synaptic tagging” continually directs newly synthesized, synapse-potentiating molecules to previously activated synapses.

PKMzeta is an autonomously active, atypical PKC isoform that is synthesized in late-LTP and memory formation and persistently enhances AMPAR-mediated synaptic transmission in the absence of second messenger stimulation. We find that KIBRA (KIdney/BRAin protein/WWC1), a postsynaptic scaffolding protein genetically linked to human memory performance, continually anchors PKMzeta to active synapses. The interaction is necessary to maintain established late-LTP and sustain long-term memory for at least one month. Inhibitors of KIBRA-PKMzeta interaction disrupt late-LTP and memory in wild-type mice but do not affect the compensatory mechanisms that sustain LTP or memory in PKMzeta-knockout mice. These pharmacogenetic experiments show that PKMzeta is crucial for maintaining late-LTP and memory under normal physiological conditions and suggest that persistent synaptic tagging may be a general mechanism targeting other plasticity-related proteins to active synapses. Modeling KIBRA-PKMzeta interaction predicts that formation of KIBRA-PKMzeta dimers is the essential step seeding larger multimeric complexes, the components of which can be perpetually exchanged leading to stable synaptic potentiation and long-term memory.

October 7th, 2025 – Dr. Zachary Kilpatrick
Associate Professor, Applied Mathematics and Computer Science, University of Colorado Boulder, CO

Title: **The impact of bias on estimation and choice: From behaviors to neural circuits models.**

Abstract: Why does the brain sometimes seem wired to make “mistakes”—and how can that be a good thing? My group uses mathematical and computational models—guided by behavioral experiments—to explore how biases emerge in estimation and choice. We examine tasks where people must remember or accumulate uncertain evidence, revealing consistent patterns in how their answers deviate from perfect accuracy. By building models of neural circuits that can reproduce these patterns, we uncover how the brain’s dynamics and connectivity may shape its handling of uncertainty. I will share how these modeling insights connect back to real-world behavior, and how they point to surprising benefits of noise, diversity, and even bias.

October 21st, 2025 – Dr. Emily A. Gibson

Associate Professor, Dept. of Bioengineering, University of Colorado Denver, Anschutz Medical Campus, Aurora, CO

Title: **Bringing the microscope to the mouse!**

Abstract: Understanding how the brain's complex neural networks perform critical functions and govern behavior, cognition and intuition is a key goal of neuroscience and can lead to improved treatment for various neurological disorders. The development of new tools for studying the brain is critical in this effort. Light microscopy has greatly expanded the capabilities for minimally invasive cellular-level biological studies and in combination with genetically encoded fluorescent indicators allows unprecedented real-time imaging of activity in individual neurons in a network. In this talk, I will discuss recent work in my lab on the development of miniature fiber-coupled microscopes for 3-D imaging using adaptive optics and their applications for studies in freely moving and behaving animals. Additionally, I will discuss how control of light patterning combined with optogenetics in these miniature microscopes makes it possible to modulate neural circuits and study how this can affect behavior.

November 4th, 2025 – Dr. Hugo Tejeda

Stadtman Principal Investigator, Chief , Unit on Neuromodulation and Synaptic Integration, National Institute of Mental Health (NIH), Bethesda, MD.

Title: **Prefrontal cortical neuropeptidergic control of defensive states and circuit function.**

Abstract: Emergent properties of the brain underlie coordinated motivated behavior in response to challenges and reward pursuit. Emergent properties are complex activity patterns in microcircuits and networks not predictable from properties of the individual parts. Dysfunction in emergent properties of limbic circuitry is implicated in mental health disorders. Neuropeptides and monoamines, secreted neuromodulators that signal via G-protein coupled receptors (GPCRs), are expressed in limbic circuits and trigger robust changes in motivation and affect. *There remains a critical knowledge gap in our understanding of how neuromodulators regulate emergent properties of limbic circuits underlying top-down control of coordinated motivation and affective behavior, largely due to technical limitations.* Neuromodulators expressed in limbic circuits include the endogenous opioid peptides dynorphin (Dyn) and enkephalin (Enk), the neuropeptide somatostatin (SST) and the monoamine dopamine (DA). These systems are of relevance as their dysfunction is implicated in mental health disorders and GPCRs, broadly-speaking, represent the largest existing therapeutic space. **The mission of our laboratory is to understand how neuropeptides and monoamines modulate limbic circuit function from cellular action to large scale networks to control motivation and affect relevant to mental health and disease.**

December 2nd, 2025 – Dr. Luke Evans

Assistant Professor, Department of Ecology and Evolutionary Biology, University of Colorado Boulder, CO

Title: **Genome-wide interaction associations identify specific neuronal cell types and molecular pathways implicated in behavioral and aging traits.**

Abstract: Genome-wide association studies (GWAS) have identified numerous genes linked to behavioral and aging traits, including psychiatric traits, measures of substance use and disorders, and neurodegenerative diseases. Yet nearly every single published GWAS of has implemented a model with a central, strong assumption – that every gene affects risk independently of all other genes. This standard model of single-locus associations is computationally efficient, and these GWASs have advanced our understanding of genetics. However, this standard GWAS assumption of independence of all genetic effects leaves critical gaps in our understanding of the biology of complex traits because genes exist and operate together within biological networks and molecular pathways: How do risk genes interact to modify disease risk? How do they operate within key molecular pathways? Do different suites of interacting genes affect disease risk in specific neuronal cell types or brain tissues? Can one risk allele be ameliorated by a protective allele in another part of the same or a different pathway? One productive, alternative approach to standard GWAS assumptions is to incorporate gene-gene interactions (epistasis) into association studies, directly modeling how one gene's effect on risk may depend on the effects of another. Here, I will introduce one way to efficiently and exhaustively test all pairwise gene-gene interaction associations with behavioral traits and identify enriched cell types and molecular pathways, using examples of substance use and Alzheimer's disease.