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 2026

September 8th, 2026 – Dr. Charles Hoeffer
Professor, Institute for Behavioral Genetics and Integrative Physiology, University of Colorado Boulder, CO.

Title: **Beyond Neurons: AKT2-Dependent Astrocyte Signaling in Nicotine Responses and Reward**

Abstract: Nicotine addiction has traditionally been studied primarily through its effects on neuronal nicotinic acetylcholine receptors. Increasing evidence, however, suggests that astrocytes actively sense nicotine and may contribute to the cellular and circuit adaptations associated with nicotine exposure. This study examines the role of AKT2, an AKT-family kinase highly enriched in astrocytes, in regulating astrocytic responses to nicotine. Using mouse models, primary astrocyte cultures, morphometric analysis, biochemical assays, transcriptomics, and conditioned place preference, Lombardi et al. show that astrocytes respond differently depending on the duration of nicotine exposure. Acute nicotine increases astrocyte morphological complexity and area, whereas chronic nicotine exposure produces a different, generally reduced morphological state. These responses are altered when Akt2 is selectively removed from astrocytes, indicating that AKT2 helps regulate how astrocytes adapt to nicotine exposure. In cultured astrocytes, nicotine-induced morphological changes require nicotinic acetylcholine receptor signaling: pharmacological inhibition of either α7- or α4β2-containing nAChRs prevents the normal morphological response to nicotine. Loss of AKT2 also disrupts this response, placing AKT2 downstream of, or functionally coupled to, astrocytic nicotinic receptor signaling. Importantly, the consequences extend beyond astrocyte morphology. Mice lacking AKT2 specifically in astrocytes show reduced nicotine-conditioned place preference, suggesting that astrocytic signaling contributes to the rewarding or associative properties of nicotine. Transcriptomic analyses further show that nicotine elicits a molecular response distinct from classical lipopolysaccharide-driven inflammatory activation, supporting the idea that astrocytes can adopt stimulus-specific functional states rather than simply becoming generically "reactive." Together, these findings support an expanded view of nicotine neurobiology in which astrocytes are active participants in drug-induced plasticity. The study raises broader questions about how glial signaling interfaces with neuronal circuits during the transition from acute drug exposure to repeated use, dependence, and withdrawal.

September 22nd, 2026 – 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 dynamics**

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 computations in networks to control motivation and affect relevant to mental health and disease.**

October 6th, 2026 – Dr. Jacqueline Giovanniello

Assistant Professor, Center for Substance Abuse Research, Department of Neural Sciences, Temple University Lewis Katz School of Medicine, Philadelphia, PA

Title: **Neural mechanisms for stress-potentiated habit formation.**

Abstract: Behavioral flexibility is the capacity to adapt our actions as circumstances change. It is a hallmark of adaptive decision making. This flexibility depends on a dynamic balance between two fundamental modes of behavioral control: goal-directed actions, which are guided by expected outcomes, and habits, which are more automatic and resistant to change. Disruptions in this balance can drive maladaptive decision-making and are a feature of many neuropsychiatric conditions. Yet the neural mechanisms that regulate the transition between flexible actions and rigid habits remain poorly understood. Our recent work shows that chronic stress alters amygdala–striatal circuitry to promote premature habit formation, revealing one mechanism through which environmental experience can bias behavioral control. Building on these findings, our research program broadly investigates how stress, drugs of abuse, and genetic alterations reshape neural circuits to promote inflexible, habitual behavior, toward the goal of improving our approach to treating a range of neuropsychiatric conditions.

October 20th, 2026 – Dr. Anna Gillespie

Assistant Professor, Dept of Neurobiology & Biophysics, Dept of Lab Medicine and Pathology, University of Washington School of Medicine, Seattle, WA

Title: **Exploring how aging degrades neural mechanisms of memory encoding and consolidation.**

Abstract: Unfortunately, aging is often accompanied by memory impairment, even for healthy individuals. The hippocampus – a brain region central to memory processes – is particularly vulnerable to age-related changes. Spatially-tuned hippocampal neurons, called place cells, fire in time-compressed sequences that play an important role in memory encoding, consolidation, and memory-based decision-making. These sequences occur during movement (referred to as theta sequences) as well as during immobility (referred to as replay). To determine how these neural sequences are affected by the aging process, we collect high-density hippocampal recordings from young and aged rats while they performed a complex memory-guided spatial task. Decoded sequences of place cell firing exhibited multiple age-related changes during both movement and immobility, some of which correlate with behavioral performance. These results shed light on how age-related changes in hippocampal patterns of neural activity may contribute to memory impairment in advanced age.

November 3rd, 2026 – Dr. Alberto Cruz Martin

Associate Professor, Department of Anesthesiology, University of Colorado Anschutz Medical Campus, Aurora, CO

Title: **Neuroimmune Signaling Across Scales: From Synapse Loss to Network Vulnerability**

Abstract: How do genetic risk and environmental challenges alter the developing brain in ways that create vulnerability to disease? Our laboratory investigates this question across multiple biological scales, from individual molecules and synapses to specific neuronal populations and distributed brain networks. A major focus is the complement system, a collection of immune-related molecules that also functions within the brain and can profoundly influence neuronal connectivity. I will discuss our work showing that altered complement signaling can disrupt synapses through both immune-cell-dependent and unexpected neuron-intrinsic mechanisms, and how these changes affect the function of specific cortical circuits. I will also highlight our emerging efforts to map how genetic susceptibility and environmental perturbations reshape neuroimmune and cellular programs across the brain using spatial transcriptomics. Together, this work aims to understand how relatively localized molecular changes can propagate across scales to produce broader network vulnerability in neuropsychiatric and neurological disease.

December 1st, 2026 – Dr. Kenneth Wright
Professor, Department of Integrative Physiology, University of Colorado Boulder, CO.

Title: **TBD**

Abstract: TBD