Neuroscience Seminar Series schedule

All seminars are currently scheduled from 4-5 pm mountain time, with one exception this semester (Tracy Bale). Currently, all the speakers are schedule to present in-person, but this may change; I will keep you posted.

Fall 2023

September 5th, 2023 – Dr. Sara Aton (in person)
Associate Professor, Department of Molecular, Cellular and Developmental Biology,
The University of Michigan, Ann Arbor, MI

Title: Network and cellular mechanisms for disruption of memories by sleep loss

Abstract: Post-learning sleep plays an important role in memory processing. In this presentation, I will discuss recent work from our lab aimed at understanding the sleep-driven changes that occur in the mouse neocortex and hippocampus during memory consolidation, and how sleep disruption interferes with these changes. I will describe our recent work on the effects of sleep and sleep disruption on the offline reactivation of memory-encoding "engram" neurons in the sensory cortex and the hippocampal dentate gyrus (DG) in the hours following learning. I will also discuss our recent findings on the effects of sleep and sleep disruption on the activity of hippocampal interneurons, which actively gate post-learning information processing by principal neurons in the DG. Finally, I will discuss our use of hippocampal subregion-specific spatial profiling of transcripts and proteins, with the aim of identifying cellular pathways affected by learning and subsequent sleep or sleep disturbance in specific parts of the hippocampal circuit. These data provide evidence for a number of cellular- and microcircuit-level mechanisms by which sleep loss can interfere with hippocampal memory consolidation mechanisms.
Title: Extracellular vesicles serve as stress signals important to brain development and function

Abstract: Exposure to traumatic events and stress across the lifespan is a risk factor for negative physical and mental health outcomes. However, we know little about what the signals are that produce these lasting effects or the mechanisms as to how this occurs. Extracellular vesicles (EVs) are nanoparticles secreted by all tissues in the body, including the brain, and serve as a novel signaling mechanism involved in important developmental and maturational processes. EVs carry protein and RNA cargo to local or distant cells to communicate information vital for maintaining homeostasis, and are highly dynamic in response to disruptions to homeostasis. Interestingly, our recent studies found that stress significantly alters the type and composition of EVs secreted into circulation in both humans and mice. EVs produced following periods of high stress or traumatic events can be used as biomarkers – indicating lasting changes in brain function, cellular processes, or point to novel tissues involved in disease. Our studies are working to identify the important EV components and develop synthetic nanoparticles that could be used to intervene in negative health outcomes.
October 3rd, 2023 – Dr. Erica Glasper (in person)
Associate Professor, Department of Neuroscience, The Ohio State University, OH

Title: Breaking Bonds: Neurobiological consequences of loss across the lifespan

Abstract: Our brains are highly plastic and respond both structurally and functionally to a host of experiences – those beginning in the early days of life and stretching well into our older years. In this talk, I will discuss how the maintenance, or loss, of social bonds occurring throughout life (e.g., early-life adverse parental experiences, pair bonding) can modulate neural plasticity, hormones, and immune function. I will discuss data suggesting the loss of social bonds (e.g., mate bond separation, early-life parental loss) can have deleterious, sex-specific consequences on brain and behavior, highlighting the potential for central inflammation and peripheral stress hormones as potential mediators.
Abstract: Negative emotional (affective) experience is commonly multi-modal, with different types of sensory information (auditory, somatic, visual) contributing to our affective state. Research suggests that our brains contain representations (circuitries) for processing negative affect that are modality-specific and such that span across modalities. However, how these representations are integrated in the brain and related to our subjective experience of ‘unpleasant’ or ‘bad’ is not well understood. In this talk, I will focus on our recent work combining fMRI and predictive modeling to offer evidence for parallel brain systems encoding modality-specific negative affect in early sensory pathways and cross-modal negative affect in a distributed set of limbic, midline, forebrain, insular, and somatosensory regions. Further evidence will be presented for the importance of both types of representation in predicting subjective experience, the specificity of these brain representations to negative affect rather than general salience or arousal, and for the robustness and generalizability of brain models to predict negative affect in independent test samples. Together, this talk will provide an integrated account of how negative affect is constructed in the brain and offer preliminary predictive neuromarkers for future studies.
Abstract: Clinical disorders arising from maladaptive emotion regulation present a large burden on society worldwide and many of these disorders show comorbidity, for example, addiction with anxiety disorders. Even though there has been much research on reward and fear processing, the majority of studies have been conducted in parallel, investigating the neuronal circuitries separately. Our lab uses a behavioral paradigm designed to assess how safety cues can regulate fear and reward seeking behaviors in male and female rats. During this behavior we record neural activity to track changes in individual neurons as the animal learns about safety, and we manipulate specific neural connections to determine which ones are necessary for this safety learning. We hope by investigating how safety, fear and reward circuits integrate their functions to influence behavior, we will be able to better understand and treat disorders resulting from maladaptive emotion regulation.
Title: **Defining molecular, cellular, and circuit programs that regulate behavioral and sensory thresholds**

**Abstract:** Our sensory environments are complex: we must decide, on a moment to moment basis, which stimuli require our attention and which should be ignored. We are able to successfully navigate that complexity through appropriately establishing, maintaining, and modulating sensory thresholds. In particular, habituation is a simple behavioral plasticity mechanism through which animals dynamically increase their response thresholds for repetitive sensory stimuli. Mechanisms that promote plasticity must be balanced by those that maintain consistent responding. Whereas learning to ignore repeated innocuous sensory stimuli is adaptive, it is crucial that certain stimuli elicit reliable responses (e.g. persistent cries of hungry offspring, lethal or toxic stimuli, etc.). In addition to their essential role in everyday life, properly tuned sensory thresholds are essential for healthy brain functioning. Sensory threshold establishment, maintenance, and plasticity are disrupted in a variety of human neurological disorders including ASD, schizophrenia, ADHD, Huntington’s disease and migraine. We have recently conducted a forward genetic screen in the larval zebrafish to identify basic mechanisms underlying sensory processing. In addition, we completed a pilot screen of putative migraine-associated genes to identify mechanisms relevant for disease. Using the powerful larval zebrafish system, we are able to link the function of individual genes with cellular processes, and ultimately circuit assembly and function, as well as animal behavior. This work provides an integrative perspective on sensory processing that spans genes, circuits, and behavior.