Neuroscience Seminar Series schedule

All seminars are currently scheduled from 4-5 pm mountain time (this may have to change for some of our speakers on the East Coast!)

FALL 2020

September 8, 2020 – Dr. Jamie Peters

Associate Professor, Department of Anesthesiology, University of Colorado, Anschutz Medical Center, CO

Title: **In search of a neural circuit that limits opioid addiction**

Abstract: Dr. Jamie Peters from the Anschutz Medical Campus at the University of Colorado Denver will be presenting her research on opioid addiction in a talk entitled “In search of a neural circuit that limits opioid addiction.” Her work led to the identification of a neural circuit that functions as a limiter of cocaine seeking in preclinical rodent models of addiction. This addiction ‘limiter’ circuit originates in a specific part of the prefrontal cortex, namely the infralimbic cortex in the rat, which equates to Brodmann Area 25 in the human neocortex. Now working on opioid addiction, Dr. Peters has been searching for a common limiter circuit capable of diminishing a broad spectrum of addictive behaviors, spanning multiple drug classes. Her recent work suggests this limiter circuit may also be capable of regulating opioid choice and relapse, although this therapeutic function may be masked by opposing ‘driver’ circuits emanating from the infralimbic cortex after opioid exposure. Her talk will focus on these opposing circuits that regulate drug seeking and taking behavior, and their potential to be exploited for the treatment of drug addiction.

September 22, 2020 – Alexis Stranahan
Associate Professor, Medical College of Georgia

Title: **It’s not the fat, it’s where it’s at: Adipose tissue distribution determines the cognitive effects of obesity**

Abstract: Many human studies suggest that obesity impairs memory and cognition, but this finding is not consistent and other groups have failed to find similar associations. Because the ‘apple-shaped’ anatomical pattern that accompanies visceral obesity has different physiological consequences than the 'pear-shaped' distribution that reflects subcutaneous adiposity, the variable cognitive outcomes reported in obese humans may be due to the use of weight/height ratios to define obesity. Visceral obesity is accompanied by peripheral inflammation and impairs memory, while subcutaneous adiposity does not activate the innate immune system and is metabolically protective. The beneficial metabolic effects of subcutaneous fat are mediated by a unique population of 'beige' adipocytes that promote energy expenditure, similar to brown adipose tissue. We hypothesized that subcutaneous fat may protect against obesity-induced cognitive dysfunction by limiting inflammation. The outcome of these experiments indicates that beige adipocytes in subcutaneous fat actively oppose obesity-induced cognitive impairment via Th2 cytokine signaling, and implicate recipient-derived lymphocytes in communication between fat and brain.

October 6, 2020 – Dr. Dayu Lin

Associate Professor, Institute of Neuroscience & Department of Psychiatry, New York University Langone Medical Center, NY

Title: **Neural mechanisms of aggression**

Abstract: Aggression is an innate social behavior essential for competing for resources, securing mates, defending territory and protecting the safety of oneself and family. In the last decade, significant progress has been made towards an understanding of the neural circuit underlying aggression using a set of modern neuroscience tools. Here, I will talk about the history and recent progress in the study of aggression.

October 20, 2020 – Dr. Erik Oleson
Associate Professor, Department of Psychology, University of Colorado Denver

Title: **Transient dopamine release events and behavior: Reward seeking, avoidance, fear, and timing.**

Abstract: Dopamine is a chemical that produces many different physiological and behavioral effects throughout the body. The brain contains three dopamine pathways, each of which plays unique roles in behavior. This talk will focus on the behavioral correlates of transient dopamine release events within one of these—the mesocorticolimbic dopamine pathway. While dopamine within this pathway is typically referred to as a reward molecule, the speaker will argue that this perspective is neither sufficient nor complete. To show that mesolimbic dopamine is not simply a reward molecule, the speaker will provide experimental evidence demonstrating that transient, or quick temporary,dopamine release events are associated with a wide array of important and survival-relevant behaviors, including: reward seeking, reward valuation, avoidance, avoidance valuation, conditioned fear, and interval timing. From these observations, it will be inferred that transient dopamine release events within the mesolimbic dopamine pathway were naturally selected to guide an array of advantageous behaviors. The focus of the seminar will then shift to dopamine transients and addiction. Based on the logic that the mesolimbic dopamine pathway is the brain's reward system, it is commonly assumed that abused drugs hijack it by producing artificially high dopamine release events. Data will be shown demonstrating that a wide variety of abused drugs all increase the frequency, but differentially influence the amplitude of dopamine transients within the mesolimbic pathway. Drugs that will be compared include: methamphetamine, MDMA, heroin, buprenorphine, WIN55,212-2, diazepam and zolpidem. From these observations, it will be argued that abuse liability does not form a simple linear relationship with dopamine—at least dopamine transients. Alternatively, these data might demonstrate that drug-induced changes in dopamine transients occur in drug class-specific patterns. And these class-specific patterns of dopamine release events could, in turn, be related to class-specific changes in reinforcement learning. While these primary drug-induced patterns of release could be important during the development of addiction, we observe striking conditioned responses in addiction models that should be considered as well. Data will be shown demonstrating the powerful control that withdrawal-associated stimuli and conditioned internal responses exert on dopamine release in drug-experienced rats. These enduring conditioned responses suggest that dopamine plays a multifaceted and complex role in addiction. From these observations, it will be inferred that dopamine's role in both advantageous behavior and addiction is much more nuanced than current dogma suggests.

November 10, 2020 – Dr. Catharine Winstanley

Professor, Department of Psychology, The University of British Columbia, BC, Canada

Title: **Cued up for addiction- the surprising effects of sound and light cues on decision making.**

Abstract: In addicted people, exposure to things like drug paraphernalia (known as contextual cues) is believed to trigger cravings, drug use and relapse. Exactly how cues promote such outcomes remains poorly understood. One possibility is that cues influence decision-making and tip the scales in favor of the maladaptive addictive behaviour, making it seem more appealing and worthwhile. Our work in both rats and humans suggests that adding sensory reward cues - similar to the lights and sounds in gambling settings - results in riskier and more disadvantageous choice on gambling-like tasks. Computational modeling data suggest that, contrary to what we assumed, pairing wins with sounds and lights doesn't enhance learning from rewards, but instead stunts learning from punishments, at least in rats. The rodent data also indicate that the addition of sensory cues not only makes performance more sensitive to dopaminergic drugs, but also changes the response to noradrenergic, cholinergic, and serotonergic compounds in perhaps unexpected ways. Understanding how win-paired sensory cues alter the brain systems which support cost/benefit decision making may offer new insight into possible treatments for addiction disorders.

December 1, 2020 – Dr. Jasper Heinsbrock

Assistant Professor, Department of Anesthesiology, Anschutz Medical Campus, University of Colorado

Title: **Striatopallidal regulation of drug seeking**

Abstract: Dr. Jasper Heinsbroek's research focuses on the processing of reward and motivation by the interconnected nuclei of the ventral basal ganglia and the regulation of motivated states by distinct subcircuits within this network. The ventral basal ganglia are an integral part of the brain reward system and these systems malfunction in psychiatric disorders of motivation, such as addiction and depression. Our previous work has identified two distinct striatopallidal circuits in the ventral basal ganglia that drive or limit motivated states during the relapse of drug seeking. A history of drug use reduces the functioning of synapses in the limiter pathway, which potentiates the motivation to seek drug due to relative increased activity of the driver pathway. This talk will focus on the mechanisms through which striatal synapses differentially recruit pallidal neurons to promote divergent motivated states in rodent models of addition.