

Neuroscience Seminar Series schedule

All seminars are currently scheduled from 4-5 pm mountain time. Currently, all the speakers are scheduled to present in-person in Muenzinger E214, but this may change; I will keep you posted.

Spring 2024

January 30th, 2024 – Dr. Kimberley Bruce (in person)

Associate Professor, Division of Endocrinology, Metabolism, and Diabetes, University of Colorado Anschutz Medical Campus, Aurora, CO

Title: Targeting microglial metabolism in neurodegenerative disease

Abstract: The way the brain uses energy is quite different from the rest of the body. For example, the brain is mostly composed of fat, and therefore fat metabolism in the brain needs to be precisely regulated to maintain brain health and prevent disease. In fact, some of the strongest genetic drivers of brain disorders such as Alzheimer's disease involve genes that are critical to maintaining fat metabolism in the brain. Many of these genes are expressed by microglia, the key immune effector cells of the brain, putting these cells center stage in the study of how changes in brain metabolism may drive neurodegenerative disease. The Bruce lab has highlighted specific fat processing enzymes expressed by microglia that may regulate the metabolism and function of microglia, and therefore may be a therapeutic target for treating Alzheimer's disease. In addition, the Bruce lab is interested in factors that trigger changes microglia metabolism and has recently highlighted that the age-associated decline in estrogen shifts microglial metabolism, to a more 'disease-associated' state. Overall, this talk will highlight how the metabolism of microglia is a viable therapeutic target for the treatment of neurodegenerative disease.

February 13th, 2024 – Dr. Maya Opendak (in person)

Assistant Professor, Department of Neuroscience, Johns Hopkins University School of Medicine and Investigator, Kavli Neuroscience Discovery Institute, Baltimore, MD.

Title: **Building a Social Brain: Genes, circuits and behavior across development**

Abstract: Disrupted social behavior is a core feature of compromised mental health, including anxiety and depression, and is a long-standing early diagnostic marker of disorders that emerge in later-life. Yet, we have little understanding of the ontogeny of social behavior neural circuits or how environmental perturbation at different stages of development impacts infant behavior. Using the infant rodent pup, my lab studies the specific infant neuroanatomical circuits that generate developmentally-appropriate social behavior and how these systems go awry following adversity. My team's work examines how ethologically-relevant social challenges, such as early caregiving adversity, produce profound changes in brain structure and function to modify behavior. This talk will focus on specific microcircuits in the mesocorticolimbic complex that promote typical transitions in social behavior and genes and cell-types that are impacted by adversity to disrupt function.

February 27th, 2024 – Dr. Bret Smith (in person)

Department Head and Professor, Department of Biomedical Sciences, Colorado State University, Fort Collins, CO

Title: Synaptic information processing in the vagal complex: Implications for metabolic regulation.

Abstract: Diabetes mellitus is a major health concern, affecting over 30 million people in the United States. Serious complications resulting from diabetes including include heart disease, stroke, hypertension, blindness, nervous system damage, and autonomic dysfunction. A major impediment to developing successful diabetes treatments (versus treating symptoms) is the relative knowledge gap regarding the multifaceted and redundant systems that are affected by and contribute to control of metabolic homeostasis. This seminar will discuss disease-related plasticity of central neural circuitry controlling autonomic function. Second-order viscerosensory neurons in the nucleus tractus solitarius (NTS) are also sensitive to glucose and circulating hormones that are known to be involved in metabolic regulation. These neurons contribute significantly to autonomic regulation of glucose homeostasis by signaling integrated visceral and humoral signals to brain areas that directly regulate systemic glucose levels, including the dorsal motor nucleus of the vagus nerve (DMV), which contains vagal motor neurons. Vagal motor function is altered in diabetes, leading to autonomic dysregulation, including excess hepatic glucose production and gastric motility dysfunction. We have found that changes in activity of GABA neurons in the NTS affects systemic [glucose], and GABA receptors are reorganized in the vagal complex after a few days of hyperglycemia. Vertical sleeve gastrectomy rapidly improves glycemic index rapidly in patients and animal models of diabetes, independent of weight loss, and convergent data suggest the brainstem vagal complex is integral to this response. The broad hypothesis discussed in this seminar is that altered neural function in the vagal complex reflects a neurogenic component of diabetes pathology. Modulating specific neural functions in the vagal system may be used to address diabetes-related glycemic dysregulation.

March 12th, 2024 – Dr. Scott Thompson

Professor, Department of Psychiatry, Director of the Center for Novel Therapeutics,
University of Colorado School of Medicine, Anschutz Medical Campus, Aurora, CO

Title: The hope and the hype of psychedelic medicine: man, mouse, and public policy

Abstract: You've read Michael Pollen's book, all your friends have tried 'shrooms, you voted for Colorado Prop 122, but what actually is the deal with psychedelics and mental health? Do they really work? How good is the evidence? Does microdosing work? How do they work? How and when will they be available in Colorado? Come learn the latest in clinical and basic research on the use of psychedelics for treating psychiatric illness and the underlying neurobiological mechanisms, and get caught up on the status of Colorado's Natural Medicine Healing Act policy implementation.

April 9th, 2024 – Dr. Carole Menard (in person)

Associate Professor, Department of Psychiatry and Neuroscience, Faculty of Medicine & CERVO Brain Research Center, Université Laval, Quebec, Canada

Title: Neurovascular adaptations underlie stress vulnerability vs. resilience in mice and human depression.

Abstract: Our research program aims to shed light on the biological mechanisms underlying stress vulnerability vs resilience, with help of state-of-the-art photonic technology, in order to develop innovative treatments and identify biomarkers of mood disorders. Our multidisciplinary approach combines behavioral experiments to functional, cellular, molecular, and imaging studies and validation of our rodent findings in human samples. We showed that chronic stress exposure promotes blood-brain barrier hyperpermeability leading to passage of circulating inflammatory mediators into the brain and the establishment of depressive behaviors. These changes occur in a sex-specific manner which may contribute to sex differences in depression prevalence, symptoms and treatment responses.

April 23th, 2024 – Dr. Matthew Hill (in person)

Professor, Department of Cell Biology & Anatomy and Psychiatry, Hotchkiss Brain Institute, University of Calgary, Alberta, Canada

Title: **Cannabinoids, the Amygdala and the HPA Axis**

Abstract: Cannabinoids have long been known to regulate stress responses and emotional behavior, and reductions in stress are often cited as the primary reason as to why individuals use cannabis in a recreational manner. Both rodent and human work have identified that the amygdala is a primary region of action within the brain for the anti-stress actions of cannabinoids. In further exploring how cannabinoids may act within the amygdala to regulate neuroendocrine responses to stress, we first needed to further understand the manner in which the amygdala itself regulates the HPA axis. To this extent, we performed a series of anatomical and functional studies to understand the extent to which the amygdala regulates the stress response, for which we can then overlay endocannabinoid function to establish the mechanisms by which it acts within the amygdala to suppress stress.