Neuroscience Seminar Series Calendar – Fall 2018

**Tuesday, September 11**th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4-5 pm

**Matt Lattal**, Professor, Department of Behavioral Neuroscience, Oregon Health & Science University, Portland, OR.

*Extinction, memory erasure, and the problem of relapse*

Work in my lab is focused on two very broad questions: How do memories form? And once they are formed, how can they be eliminated or inhibited? We know a lot about the behavioral conditions and cellular and molecular processes that are involved in normal memory formation. In my lab, we are especially interested in the mechanisms that are involved in the development and inhibition of particularly salient memories – those involving trauma or drugs of abuse. We have found that repeated exposure to cues associated with trauma or abused substances results in a weakening of the learned behavioral responses (fear or drug-seeking) through a process known as extinction. I will discuss theoretical (extinction and memory erasure) and molecular (particularly epigenetic) mechanisms that may operate to cause the persistent loss of behavior and will describe how this research may inform current treatments for disorders such as PTSD and addiction that involve the inability to suppress invasive memories.
Tuesday, September 25th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4-5 pm

Christine Brennan, Assistant Professor, Departments of Speech, Language, and Hearing Sciences, University of Colorado Boulder, CO

The Cognitive Neuroscience of Language and Reading Processes

The study of the brain and language has a long history, but in the last twenty years the use of neuroimaging methods has greatly advanced our understanding of the neural basis for language processes. Using a physiologically constrained model of word processing as a framework, this talk will review the cognitive and neurological bases for language and reading processes. Results from neuroimaging studies focused on phonological, semantic, and syntactic processes for language inputs and outputs (i.e., receptive and expressive modalities) will be discussed. This talk will also address the overlap between language and reading processes and cross-linguistic comparisons of language from cognitive and neuroscience perspectives. Finally, differences in the cognitive and neural bases of language processes in special populations (such as developmental learning disabilities and acquired impairments) will be presented.
Tuesday, October 9th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4-5 pm

Stan Floresco, Professor, Department of Psychology, University of British Columbia, BC, Canada

Prefrontal-subcortical circuits mediating different components of risk/reward decision making

We routinely face decisions requiring evaluation and choice of different actions may or may not yield different types of rewards. These situations trigger competitive decision biases that reflect interplay between different prefrontal cortical, amygdalar, striatal and habenular nodes within the brain’s dopamine system, which plays a critical role in action selection and reward processing. This lecture discusses some of the interactions between these circuits that shape decision biases and underlie conflicting urges when evaluating options that vary in terms of potential risks and rewards. Optogenetic studies revealed that phasic activity in subcortical circuitry linking the amygdala and the ventral striatum appears to promote choice towards more preferred rewards, and modify choices after non-rewarded actions. These biases are modified by prefrontal regions that serve to temper these urges when riskier options become less profitable via top-down control over the amygdala. Dopamine transmission within these regions also makes dissociable, yet complementary, contributions to risk/reward judgments, promoting either exploitation of current favorable circumstances or exploration of more profitable ones when conditions change. These findings provide insight into the dynamic competition between these cortical/subcortical circuits that shape our decision biases and underlie conflicting urges when evaluating options that vary in terms of potential risks and rewards.
Tuesday, October 23rd

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4-5 pm

Brent Myers, Assistant Professor, Department of Biomedical Sciences, Colorado State University, Fort Collins, CO

Prefrontal cortical integration of behavioral and physiological responses to stress

Stress, defined as a real or perceived threat to homeostasis or well-being, has a considerable role in the pathogenesis of mood and anxiety disorders. Moreover, prolonged stress and mood disorders are significant risk factors for numerous cardiometabolic conditions that further burden health-related quality of life. The neurobiological mechanisms of stress-related health detriments remain elusive; however, we have identified a specific population of chronic stress-activated glutamate neurons in infralimbic cortex that integrate behavioral and physiological responses to stress. Large-scale quantitative circuit mapping in rats identified a network of projections from these cells to numerous behavioral and homeostatic regulatory regions. Furthermore, knockdown of vesicular glutamate transporter 1 (vGluT1) in infralimbic cortex was employed to reduce presynaptic vesicular glutamate packaging and excitatory outflow from infralimbic cortex in rats undergoing chronic variable stress. This produced a marked divergence of behavioral and physiological stress responding. In terms of behavior, rats with vGluT1 knockdown were protected from the effects of chronic stress on avoidance, depression-like, and anxiety-related behaviors. In contrast, rats experiencing chronic stress with reduced infralimbic outflow exhibited enhanced physiological reactivity to chronic stress. In fact, vGluT1 knockdown and chronic stress interacted to increase basal and stress-induced corticosteroid secretion, enhance heart rate reactivity to stress, elevate chronic blood pressure, and induce vascular endothelial dysfunction. In order to test the hypothesis that infralimbic neurons coordinate the behavioral and physiological aspects of stress responding, we utilized an optogenetic approach to stimulate infralimbic glutamate neurons and investigate mood-related behaviors as well as physiological stress reactivity. These studies uncovered a role for infralimbic cortex in the concordance of behavior and physiological status. Specifically, optogenetic activation of infralimbic glutamate neurons induced place preference and increased social motivation. This was coupled with reduced autonomic and endocrine stress reactivity. Collectively, these studies highlight infralimbic cortical neurons as a critical component of the neural networks mediating stress chronicity and the adaptive integration of behavioral strategy with physiological status.
Keri Martinowich, Associate Professor, Department of Psychiatry and Behavioral Sciences, The Johns Hopkins University School of Medicine

**Complex regulation of brain-derived neurotrophic factor (BDNF) gene expression controls pleiotropic effects of BDNF-TrkB signaling in brain circuits that control behavior**

The critical importance of brain-derived neurotrophic factor (BDNF) signaling in numerous brain processes, including cognition, learning and memory is undisputed. Indeed, intact BDNF signaling is deemed critical for normal brain function and is disrupted in many neuropsychiatric and neurodevelopmental disorders. Stimulating BDNF production is proposed therapeutically in a number of disorders, but since BDNF and its cognate receptor TrkB are widely expressed in multiple cell types and brain regions, global stimulation strategies suffer from a lack of specificity. The *BDNF* gene has nine 5’ untranslated region (UTR) exons, each containing a unique promoter. These promoters drive transcription of *BDNF* variants that contain different regulatory sequences, but encode a similar protein. Isoforms show distinct expression patterns suggesting localization in discrete circuits. Since they produce a similar protein, individual transcripts may serve similar molecular roles, but be activated in cell populations that are dedicated to distinct brain functions. This has led to the hypothesis that *BDNF* isoforms may have discrete, rather than redundant, roles in brain function. This *BDNF* transcriptional “code” would provide tight temporal and spatial control of BDNF signaling and downstream plasticity in discrete subpopulations of neurons, allowing a single cell-signaling pathway to mediate a myriad of functions. If distinct transcripts are expressed in circuits that control specific brain functions, those transcripts may represent more selective therapeutic targets.
**Tuesday, December 4th**

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Muenzinger Psychology, Room E214, 4-5 pm

**Diego Restrepo**, Professor, Department of Cell and Developmental Biology, University of Colorado, Anschutz Medical campus, Denver

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*Shedding light on the olfactory cocktail party effect*

Linda Buck and Richard Axel won the Nobel prize because of their discovery that the olfactory input takes place through a large number of olfactory receptors (~1,200 in mice, ~220 in humans). This raises the question of how the brain can make rapid decisions based on the sense of smell given this massive parallel input. Studies in our laboratory have shown that processing the input to the olfactory bulb, the first relay station that receives the parallel olfactory input, is filtered by modulation by centrifugal fibers from modulatory centers and downstream processing brain areas. My laboratory is studying whether this process of filtering the incoming information is analogous to the cocktail party effect where auditory processing focuses on the relevant conversations in a crowded room. I will discuss background on modulation in the olfactory bulb, a study of the role of interneurons in plasticity in the basolateral amygdala, a candidate brain area for gating olfactory information, and efforts for development of new tools to record and modulate neural activity.