Seminar/Talk Calendar

Tuesday, September 15th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

Jing Wang, Professor and Director of Research, Center for the Study and Treatment of Pain, NYU School of Medicine

Central Glutamate Signaling in Pain Regulation

Pain has both sensory and affective components. How pain is represented and regulated in the brain, however, is not well understood at the molecular and circuit levels. We are employing a combination of optogenetics, in vivo recording in freely moving rodents, behavior testing, and imaging to study how the cortex connects with subcortical regions to regulate complex pain behaviors. Our data show that excitatory glutamate outputs from the prefrontal cortex to the nucleus accumbens, a key projection within the brain's reward circuitry, provides powerful control over both sensory and affective pain experiences. In addition, our most recent data have identified distinct patterns of neuronal activities in the somatosensory cortex and the prefrontal cortex in response to an acute pain stimulus, suggesting that sensory and affective experiences of pain are differentially coded in the brain.

(For additional information, please contact Serge Campeau at: Serge.campeau@colorado.edu)

Tuesday, September 29th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

Mike Saddoris, Assistant Professor, Department of Psychology & Neuroscience, University of Colorado Boulder

Higher-Order Conditioning Reveals Dissociable Neural and Dopaminergic Signals for Learning, Motivation and Addiction

Learning is essential for animals to adapt to new conditions and seek out rewarding items in their environment, and much research has been dedicated to understanding how cells and circuits in the brain encode information essential for flexible and adaptive motivated behaviors. Recent theories suggest that dopamine is critical for allowing the brain to learn about new and rewarding stimuli, but there has been considerable disagreement about how this neurotransmitter system contributes to these processes, whether via encoding of rewarding properties of valued outcomes (the "hedonic" model), or instead via the acquired predictive value (Reward Prediction Error) or motivational properties (Incentive Salience) of cues and actions, and whether any of this

information is necessary for decision making. Using both single-unit electrophyiology and fast-scan cyclic voltammetry in higher-order conditioning tasks, we have been able to show that neural encoding and real-time dopamine release parsimoniously encode all of these critical aspects of learning, but do so in a manner that sharply differs by the specific regions of the striatum. For example, we have shown that nucleus accumbens shell is biased towards the encoding of motivational properties of stimuli and the hedonic value of rewards, while the nucleus accumbens core is more tuned to reward prediction error encoding and subjective devaluation of rewards by factors like effort, delay and risk. Chronic self-administration of drugs of abuse, such as cocaine (which directly acts upon the dopamine system), appears to fundamentally augment these dopaminergic pathways, even months after abstinence and in a manner highly specific to nucleus accumbens subregions. The cognitive impairments in these animals is consistent with the long-term rewiring of the mesolimbic dopamine system and indeed resemble the effects of optogenetic manipulation of dopamine circuits in normal animals. These findings are discussed in terms of their implications for learning, action and potential treatment targets for addiction.

(For additional information, please contact Serge Campeau at: serge.campeau@colorado.edu)

Tuesday, October 13th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

David Hovda, (Additional info to be added at a later date, subject to change)

(For additional information, please contact Serge Campeau at: serge.campeau@colorado.edu)

Tuesday, November 3rd

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

Marie Banich, Director, Institute of Cognitive Science
Executive Director, Intermountain Neuroimaging Consortium
Professor, Dept. of Psychology & Neuroscience, University of Colorado Boulder

Differences Amongst Individuals in The Neurobiological Substrate That Supports Executive Function

Executive function (EF) is an umbrella term for those abilities that allow us to engage in goal-directed behavior. Such abilities are commonly compromised in individuals with psychiatric and/or neurological disorders. Using the model of Miyake, Friedman and colleagues suggesting individual differences in EF can be usefully considered as comprising three underlying latent factors (common EF, updating-

specific EF, switching-specific EF), our laboratory has examined how differences in EF amongst individuals are associated with static aspects of brain anatomy and function. Our results demonstrates that higher levels of common EF are associated with extensions of the executive control network beyond the fronto-parietal regions typically identified in group studies. Moreover, individual differences in updating-specific EF and switching-specific EF are each associated with unique neuroanatomical and functional characteristics that are distinct from each other as well as those associated with common EF. In addition to shedding light on the neural underpinnings of individual differences in EF, these findings also provide converging evidence for the psychological structure of EF proposed by Miyake and colleagues.

(For additional information, please contact Serge Campeau at: serge.campeau@colorado.edu)

Tuesday, November 17th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

John Forssayeth, Professor, UC San Francisco

Rethinking Parkinson's Disease

Recent studies on the molecular basis of Parkinson's Disease (PD), the second most common neurodegenerative disorder, suggest a radically different explanation for the onset and progression of this relentless disease. Our studies suggest that PD represents in part a functional failure in the signaling of a key growth factor, Glial Cell-derived Neurotrophic Factor (GDNF), required to maintain the integrity of specific classes of synapses. In PD, the development of resistance to GDNF appears to be caused by a deficiency in GM1 ganglioside. In GDNF-responsive cells, and in mice with a partial deletion of a key enzyme in the GM1 ganglioside synthesis pathway, modest decreases in GM1 ganglioside levels lead to a striking reduction in GDNF-stimulated ERK phosphorylation and elimination of autophosphorylated RET kinase, the effector component of the GDNF receptor. Mice, deficient in GM1 ganglioside, develop many Parkinsonian features, such as bradykinesia, but striatal infusions of a viral vector encoding GDNF eliminates this progressive decline in movement and restores RET phosphorylation. In PD patients, absence of Phospho-RET was seen in surviving dopaminergic neurons, and analysis of post-mortem brain (cortex) tissue showed that the GM1 ganglioside concentration was at least a third lower than in age-matched controls. We have concluded from these studies that idiopathic PD is likely triggered by a loss of GM1 ganglioside content in neural tissue that leads to growth factor failure, chiefly GDNF, which in turn brings about synaptic degeneration of specific classes of neurons. The anatomical progression of the disease from the gut to the brain predicts the agency of a pathogenic factor.

(For additional information, please contact Serge Campeau at: serge.campeau@colorado.edu)

Tuesday, December 8th

CU-Boulder – Interdepartmental Neuroscience Seminar Series

Muenzinger Psychology, Room E214, 4 pm

Bruce Pennington, Professor, Psychology Department, University of Denver

An Integrated Neuroscience Understanding of Dyslexia

Advances in genetics and cognitive neuroscience are helping us trace how neurodevelopmental disorders actually develop, thus giving us glimpses of how physical changes at the molecular level change our conscious experience (the formidable body-mind problem). One of the best understood neurodevelopmental disorders is dyslexia, also called reading disability (RD), in which affected individuals have a fairly specific problem with accurate and fast printed word recognition and spelling, sometimes despite being extremely talented in other skills (e.g. several Nobel prize winners have dyslexia). In this talk I will review progress in understanding dyslexia at several levels of analysis, including A) the etiological level(i.e. genetic and environmental causes), B) the pathophysiological (i.e. how the causes of dyslexia change brain development), C) the neurocomputational and neurocognitive levels (i.e. how these changes in brain

development affect neural networks and cognitive precursors to dyslexia), and D) the clinical level (i.e. how we diagnose and treat dyslexia and how might we prevent it). I will also discuss other disorders, like ADHD, that co-occur with dyslexia because of shared genetic and cognitive risk factors.

(For additional information, please contact Serge Campeau at: serge.campeau@colorado.edu)