# 9th ANNUAL SIERRA NEVADA CHAPTER OF THE SOCIETY FOR NEUROSCIENCE & NV COBRE NEUROSCIENCE RESEARCH SYMPOSIUM

**Thursday, October 26th, 2017, 12:00pm – 5:00pm**  
Pennington Health and Science Building Room 102, UNR

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<td>12:20 – 12:30</td>
<td><strong>Jacquie Snow</strong> (President SNC-SfN) Welcome and Opening Remarks</td>
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| 12:30 - 1:15 | **KEYNOTE:** Jeffrey Cummings, Lou Ruvo Center for Brain Health, Las Vegas  
Alzheimer’s Disease Drug Development: An Insider’s Perspective |
| 1:15 – 1:30   | **Jeff Kinney** (Psychology, UNLV)  
Role of GABA<sub>B</sub> in Inflammation and Alzheimer’s disease       |
| 1:30 – 1:45   | **Robert Renden** (Physiology and Cell Biology, School of Medicine, UNR)  
Presynaptic loss of dynamin-related protein 1 profoundly alters synaptic vesicle  
release and recycling at the calyx of Held                          |
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Visual neural circuits underlying prey-capture behavior in mice       |
| 2:00 – 2:45   | Posts & Refreshments (Business Meeting – SfN Board)                   |
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A broader vision of object recognition: beyond ventral cortex         |
| 3:30 – 3:45   | **Francesco Marini** (Postdoctoral Scholar, Psychology, UNR)  
Using HD-EEG to compare neural signatures for real objects vs. their images |
| 3:45 – 4:00   | **Mark Lescroart** (Psychology, UNR)  
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| 4:00 - 4:15   | **Eelke Folmer** (Computer Science & Engineering, UNR)  
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| 4:15 – 4:30   | **Paul MacNeillage** (Psychology, UNR)  
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| 4:30 – 4:40   | **Jacquie Snow** (President SNC-SfN) Closing Remarks                 |
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KEYNOTE SPEAKERS

Jeffrey Cummings, MD, Lou Ruvo Center for Brain Health, Las Vegas

Alzheimer’s Disease Drug Development: An Insider’s Perspective

Dr. Cummings is experienced in Alzheimer’s disease (AD) drug development and clinical trials. He will describe how development of the Neuropsychiatric Inventory (NPI) led to participation in planning and interpretation of clinical trials. The current landscape of AD clinical trials, treatment targets, and biomarkers will be described. The utility of developing new quantitative approaches in neuroscience to advance a field and create career opportunities will be described.

Marlene Behrmann, Ph.D., Department of Psychology, Carnegie Mellon University

A broader vision of object recognition: beyond ventral cortex

The neural correlates of object recognition are typically assumed to be under the purview of the ventral pathway of the cortical visual system. Decades of empirical studies using neuroimaging as well as single unit recording in awake behaving non-human primates have supported this conclusion. I will describe some recent studies from my lab that examine the nature of these ventral neural representations including investigations that permit the reconstruction of the images displayed to the observer using previously acquired fMRI data from ventral cortex. I will then go on to argue that signals associated with object recognition extend beyond ventral cortex and that representations in the dorsal visual pathway and even in subcortical regions are tuned to represent shape and identity properties of objects. I will describe several studies using fMRI and psychophysics in both normal and brain-damaged individuals that support the role of these other regions in the recognition of visual objects. I will suggest that objects are widely represented in the brain and the challenge is to understand the necessity and sufficiency of these representations.
Jeff Kinney (Dept. of Psychology, UNLV)

Role of GABA\textsubscript{B} in Inflammation and Alzheimer’s disease

Alzheimer’s disease (AD) is a neurodegenerative disorder characterized by a progressive loss of neurons and cognitive function. Pathological hallmarks include amyloid plaques (A\textbeta plaques) and neurofibrillary tangles (NFTs) that are implicated in the neurodegeneration. Additional data demonstrate that inflammation in the brain occurs in AD and that activated microglia surround regions containing A\textbeta and NFTs. Chronic inflammation has been found to exacerbate AD pathologies (A\textbeta deposition and NFT) and to accelerate the disease progression. Given the evidence that inflammation plays a substantial role in AD pathogenesis the elucidation of mechanisms driving microglial activation as well as mechanisms capable of reducing inflammation is needed. Data indicating that microglial activity is suppressed by endogenous transmitter activity and these same transmitters are lost in AD suggest an avenue to address this problem. In particular, reductions in GABAergic signaling have been reported in clinical populations and animal models of AD. A growing literature indicates microglia express GABA\textsubscript{B} receptors (GABABR) and that activation of GABABR is associated with reduced microglial response and reductions in pro-inflammatory cytokines. These findings suggest that GABA loss in AD may exacerbate the inflammatory response and by extension accelerate AD deficits and pathology. It may also indicate that increased GABABR activation in AD could reduce inflammation and serve as a therapeutic target. Our laboratory is currently utilizing several related approaches to determine the role of GABA\textsubscript{B} in AD and inflammation. These projects include investigations for changes specifically in GABA\textsubscript{B} in well-established animal models of AD, examination of GABA\textsubscript{B} ligands to decrease inflammatory signaling, and the investigation of the effects of the selective loss of GABA\textsubscript{B} on microglia and impact on immune function and AD related pathology.
Robert Renden (Dept. of Physiology and Cell Biology, School of Medicine, UNR)

Presynaptic loss of dynamin-related protein 1 profoundly alters synaptic vesicle release and recycling at the calyx of Held

Synaptic transmission at glutamatergic synapses is affected when presynaptic mitochondrial function is impaired, but the underlying mechanism is poorly understood. We investigated the role of mitochondria in synaptic vesicle (SV) recycling by eliminating Dynamin-Related Protein-1 (DRP1), a protein essential for mitochondrial fission, selectively and autonomously in presynaptic terminals of the giant calyx of Held synapse in mice (DRP1-preKO). Using pre- and post-synaptic voltage-clamp recordings, we find that DRP1-preKO exhibited enhanced basal evoked response (response to 0.1Hz stimulation) and increased spontaneous synaptic activity. Standing readily-releasable pool (RRP) size was significantly reduced in DRP1-preKO, suggesting an important role for mitochondria in maintenance of SV modality at presynaptic terminal. Additionally, DRP1-preKO synapses have profoundly altered short-term plasticity, and a significant reduction in synaptic transmission delay. These results indicate that proper functioning of mitochondria regulates synaptic vesicle release during a train of stimuli. Ongoing experiments aim to determine the specific mechanism underlying this defect.

Jennifer Hoy (Dept. of Biology, UNR)

Visual neural circuits underlying prey-capture behavior in mice

Understanding how the brain detects significant information in the environment and drives an appropriate behavioral response is an important goal in neuroscience. We therefore study visually driven predatory behavior in the mouse in order to identify and understand the neural circuits in the mammalian brain that are needed to detect the sudden appearance of simple visual features and drive natural orienting and approach behaviors. I will present our recent work that identifies which cell types in the superior colliculus (SC) are required for prey detection and successful prey-capture. In addition, I will reveal our findings that a subset of these cells encodes the visual information relevant to orientating behaviors during prey-capture. The SC is a conserved subcortical structure that allocates attentional resources and is critical to appropriate orienting and approach behaviors across mammalian species. I will therefore conclude with a discussion of how we intend to expand upon these findings to further understand how the SC and connected structures process visual information in order to support natural visual behavior.
Francesco Marini (Postdoctoral Scholar, Snow Lab, Psychology, UNR)

Using HD-EEG to compare neural signatures for real objects vs. their images

Recent behavioral and fMRI research indicates that real objects may be processed and represented differently to their images, although the underlying neural mechanism for these effects remains unknown. Here, we compared electrophysiological brain responses for real objects with matched pictures to examine whether the underlying temporal dynamics of brain activation differed across the two display formats. We recorded brain responses to real-world graspable objects and 2D photographs of the same items using high-density EEG. We found significant reductions of event-related power for real objects versus pictures over bilateral centro-parietal electrodes in the mu frequency band, consistent with enhanced motor preparation processes. The temporal dynamics of recruitment of sensorimotor regions in the dorsal stream are different for real objects than 2D pictures.

Mark Lescroart (Dept. of Psychology, UNR)

Decoding the 3D spatial structure of scenes

Abstract: The human visual system must take 2D input from the retina and convert it into useful 3D information about the world, including information about scene structure. There are many scene-selective areas in the brain, but it’s still unknown how these areas represent the 3D structure of the local visual environment. In this talk, I will present work modeling fMRI responses in scene-selective areas as a function of 3D structural scene features—specifically, the orientation and distance to large, occluding surfaces. The fit models reveal tuning for scene openness and distance across voxels in scene-selective regions. As a demonstration of the power of these models, I will show how the fit model can be used to predict brain responses to complex naturalistic scenes in independent data, and conversely, how the model can be used to reconstruct the approximate 3D layout of a perceived scene based on brain activity.
Eelke Folmer (Dept. of Computer Science & Engineering, UNR)

How do we move around? Solving virtual locomotion problem.

Moving around freely has been a fundamental appeal of 3D games for decades, but facilitating this in virtual reality (VR) has been a major challenge. Natural walking input using positional tracking on PC VR platforms generally delivers the most natural and immersive experiences while reducing cognitive load and minimizes the occurrence of VR sickness. Natural walking doesn’t scale beyond the confines of available tracking space and users must switch to using an alternative locomotion method (ALT). Popular ALTs include teleportation, full locomotion and vehicle movement. However the usage of ALTs can be detrimental to presence and or induce VR sickness. This talk will provide a brief overview of challenges with existing ALTs and present some novel ALTs developed by Dr. Folmer’s VR lab that aim to solve VR’s locomotion problem.

Paul MacNeilage (Dept. of Psychology, UNR)

Motor contributions to human self-motion perception

Humans glean information about movement through the environment from multiple sources. Motor signals related to eye and head movement compliment visual and vestibular sensory signals that contain information about eye and head movement, respectively. Research in my lab aims to quantify the relative contributions of these various signals in psychophysical experiments with healthy human subjects. For example, recent work characterizes the dependence of visual-vestibular conflict detection on oculomotor fixation behavior. Ongoing work is investigating how neck motor signals contribute. These findings have practical implications for virtual reality technologies where sickness induced by visual-vestibular conflict is a major concern.