

## Symposium for Young Neuroscientists and Professors of the SouthEast

March 25, 2017

Presbyterian College

Clinton, South Carolina

SYNAPSE 2017

## Saturday March 25, 2017Presbyterian College, Clinton, SC

### **Location: Registration and Lectures: Edmund Hall**

### **Posters and Workshops: Harrington-Peachtree Academic Center**

7:30-8:25am Poster Set-up (**Odd** numbers only)

*Location:* ***Harrington-Peachtree Academic Center***

7:30-8:25am Registration, Coffee & Light Breakfast

*Location:* ***Edmund Hall***

### 8:30-9:45am **Welcome & Opening Keynote Address**

*Location:* ***Edmund Hall***

**Medicinal Cannabis: Science, Politics and Medicine**

## Mark Wallace, MD

Pain Management Specialist, Chair, Division of Pain Medicine, Department of Anesthesiology, University of California, San Diego, CA

### **Webpage:**

<https://healthsciences.ucsd.edu/som/anesthesia/research/faculty-research/Pages/wallace.aspx>

### 9:45-10:30am **Student Platform Presentations – Session I**

*Location:* ***Edmund Hall***

### 9:45-10:00am **Effects of the CB1 neutral antagonist AM4113 on palatable food motivation**

***Awardee Speaker:* Rose Ying**

*Department of Biology, Wake Forest University*

10:00-10:15am Effects of epigenetic modifications in X-linked adrenoleukodystrophy

***Awardee Speaker:* Sarah Steadman**
*Department of Biology, Program of Neuroscience, College of Charleston; Department of Pediatrics, Medical University of South Carolina*

10:15-10:30am The role of rostromedial tegmental nucleus afferents in suppression of reward seeking under punishment

***Awardee Speaker:* Samantha Black**
*Department of Biology, Program of Neuroscience, College of Charleston; Department of Neuroscience, Medical University of South Carolina*

10:30-10:45am Coffee Break

*Location:* ***Harrington-Peachtree Academic Center***

10:45-12:00pm Poster Session I (**Odd** numbers present)

*Location:* ***Harrington-Peachtree Academic Center***

12:00-12:30pm Lunch pickup

*Location:* ***Harrington-Peachtree Academic Center***

12:00-12:30pm Poster Session II Set-up (**Even** numbers only)
*Location:* ***Harrington-Peachtree Academic Center***

### 12:30-1:00pm Lunchtime Workshop Session I

### *Location:* ***Harrington-Peachtree Academic Center***

**Preparing for Graduate Study in Neuroscience**

*Keynote speakers: Dr. Mark Wallace and Dr. Axel Nimmerjahn*

*Location:* ***Bennett A. Brown Conference Suite - Amphitheater***

**SYNAPSE Steering Committee Meeting***Location:* ***Conference Room 204***

### 1:00-1:30pm Lunchtime Workshop Session II *Location:* ***Harrington-Peachtree Academic Center***

**Careers in Neuroscience**

*Dr. Adam Franssen, Associate Professor of Biology, Longwood University
Location:* ***Bennett A. Brown Conference Suite - Amphitheater***

### 1:30-2:45pm **Poster Session II (**Even **numbers present)**

*Location:* ***Harrington-Peachtree Academic Center***

2:45-3:00pm Coffee Break

*Location:* ***Harrington-Peachtree Academic Center***

### 3:15-3:45pm **Student Platform Presentations – Session II**

*Location:* ***Edmund Hall***

3:15-3:30pm Regulation of behavioral flexibility by ventral hippocampus
***Awardee Speaker:* Kathleen Bryant**

*Department of Biology, Program in Neuroscience, College of Charleston; Department of Neuroscience, Medical University of South Carolina*

3:30-3:45pm Mechanisms Underlying Cognitive Deficits Following Repeated Methamphetamine Use

***Awardee Speaker:* Jordan Costello**

*Department of Biology, Program of Neuroscience, College of Charleston; Department of Neurosciences, Medical University of South Carolina*

3:45-4:45pm Closing Keynote Address

 *Location:* ***Edmund Hall***

 **How do glial cells control CNS function?**

 **Axel Nimmerjahn, PhD**

Assistant Professor

Waitt Advanced Biophotonics Center

Salk Institute for Biological Studies. La Jolla, San Diego, CA

Webpage:

[https://www.salk.edu/scientist/axel-nimmerjahn](https://www.salk.edu/scientist/axel-nimmerjahn/)





**Medicinal Cannabis: Science, Politics and Medicine**

**Mark S. Wallace, M.D.**

Pain Management Specialist

Chair of Division of Pain Medicine

Department of Anesthesiology

University of California

San Diego, CA

Mark Wallace, MD, is a board-certified anesthesiologist who specializes in multi-modal pain management. He has been the program director of UC San Diego Health’s Center for Pain Medicine since 1996. Under his leadership, the Center for Pain Medicine was named a Clinical Center of Excellence in Pain Management in 2010 and 2014 by the American Pain Society. Dr. Wallace is also chair of the Division of Pain Medicine within UC San Diego School of Medicine’s Department of Anesthesiology and has co-authored 119 peer-reviewed articles and five books on pain medicine. He is active in clinical trials of investigational drug and techniques for managing chronic pain. He received the Leonard Tow Humanism in Medicine Award in 2012 and is consistently elected as one of San Diego’s Top Doctors in *San Diego Magazine*'s "Physicians of Exceptional Excellence" annual survey. Dr. Wallace is currently a member of the Board of Directors of the American Pain Society and serves on scientific planning meetings for both national and international meetings, including the World Congress of Pain, World Institute of Pain, American Academy of Pain Medicine and American Society of Regional Anesthesia and Pain Medicine.





**How do glial cells control CNS function?**

**Axel Nimmerjahn, Ph. D.**

Assistant Professor

Watt Advanced Biophotonics Center

Salk Institute for Biological Studies

La Jolla, San Diego, CA

Axel Nimmerjahn, Ph.D., is an Assistant Professor with Waitt Advanced Biophotonics Center at Salk Institute. Dr. Nimmerjahn’s research focuses on elucidating the role of microglia - resident immune cells - and astroglia - key regulatory cells - in the healthy and diseased central nervous system through development of novel imaging tools and approaches. He has spearheaded the development of new microscopy techniques to visualize the dynamics of glial cells and their functional cellular interactions in the living CNS. He has also created new innovations for cell type-specific staining and genetic manipulation and for analysis of large-scale imaging data. This has improved our understanding of CNS function and how to treat neuroinflammatory and neurological disorders. He has been the recipient of many awards and honors including the NIH EUREKA Award, NIH Director’s New Innovator Award, Whitehall Foundation Award, Rita Allen Scholar Award, Human Frontiers Science Program (HFSP) Postdoctoral Fellowship, Du Bois-Reymond Award and Otto Hahn Medal and Award.



Poster Session Abstracts

(Listed Alphabetically by Author)

1. AFFUL DK, SIEBELS AA, CHILDS AM, SCHMIDT JX, ZEHER AM, CLELAND CL

**Sensory Mechanisms Underlying the Escape Response to Looming Stimuli in Crickets**

*James Madison University*

**ABSTRACT:**

Animals respond to aversive stimuli with escape or withdrawal responses. In crickets, wind, which might normally be produced by an approaching predator, has been shown to evoke an escape response in which the cricket turns 180 degrees from the wind and then runs or jumps away. We have shown that crickets (Acheta domesticus) largely utilize the same turning strategy for looming stimuli, which provides both wind and visual sensory cues. However, there is lack of literature on the sensory modalities that underlie the escape. Our specific aim is to identify and compare the role of visual, cercal, filiform, and antennal sensory cues in the escape of the cricket from looming stimuli. Crickets (n=90) were stimulated with a 2.5” ball (1 m/s) projected at 45 degrees using an air cylinder and stopped 20 mm from the initial position of the cricket. Crickets were stimulated at 8 angles, in 45-degree increments around the body in random order. Above the platform was positioned a high-speed video camera (650 fps) and a LED ring light. Prior to stimulation, a primary sense organ (eye, cerci or antenna) or a source of sensory information was ablated or removed to test necessity, or isolated to test sufficiency for the escape response. Crickets were blinded with lacquer nitrocellulose, or deprived of cercal or antenna information via lesions applied at the base of the appendage. A glass panel was placed between the animal and the stimulus to block wind cues to intact crickets. Crickets’ compound eyes are only receptive to light below red wavelengths so 660nm red light was used to eliminate visual cues in intact crickets. Finally, a white ball against a white background was used to eliminate visual but retain wind cues. Results showed that both eyes and cerci, but neither filiform hairs not antennae, contribute to the escape response. The contributions of both eyes and cerci to the escape depend largely on the direction of incoming stimuli. The eyes mediate escape responses to anterior looming stimuli while the cerci mediates escape responses to posterior stimuli. In addition, our results showed that escape responses mediated by the cerci had a shorter latency but similar magnitude as compared to escape responses mediated by the eye. Taken together, our results suggest that crickets use both vision and wind to program escape responses, and that the resulting movements differ in at least latency

1. ARJUNE K, BOLTON P, BRASINGTON A, BUNGE T, PADULA S, PHILLIPS T, STEINMETZ KM

**Examining Effect of Motivation, Arousal, and Valence on the N1: An event-related potential (ERP) study**

*Department of Psychology, Wofford College*

**ABSTRACT:**

In this study, researchers examined whether attentional differences can be attributed to motivation, arousal, or valence. The neural response to different levels of motivation and valence in regard to an attention-based waveform (N1) were investigated. To prime participants into processing items on a local or global level, they were asked to identify Navon letters. Participants were then shown stimuli that varied on levels of motivation, arousal, and valence while their brain waves were recorded using an electroencephalogram (EEG). After the recording was completed, participants gave individual valence, arousal, and motivation ratings for each image. Results showed that differences in the N1 only occurred when high enough levels of arousal were reached. In addition, significantly higher N1 amplitudes were found in anxious participants, regardless of motivational or valence level. Thus, contrary to prior research, this indicates that motivation level alone does not influence the N1. Furthermore, people with high levels of anxiety may be hypervigilant in reaction to all stimuli.

1. BLACK SL, LI H, JHOU TC, VENTO PJ

 **(Travel Award Winner)**

**The role of rostromedial tegmental nucleus afferents in suppression of reward seeking under punishment**

*Department of Biology, Program of Neuroscience, College of Charleston; Department of Neuroscience, Medical University of South Carolina*

**ABSTRACT:**

An inability to avoid aversive stimuli is a common symptom observed in patients with neuropsychiatric disorders such as depression and addiction. The rostromedial tegmental nucleus (RMTg) has been shown to encode aversive stimuli and sends an inhibitory efferent signal to the ventral tegmental area (VTA). Given this inhibitory influence on dopamine (DA) neurons in the VTA, the RMTg has been proposed to serve as a brake to reward circuitry in response to negative events. Recent data from our lab demonstrates that RMTg inactivation causes resistance to the suppressive effect of foot shock on food seeking. The RMTg also receives afferent projections from multiple structures, such as the medial prefrontal cortex (mPFC), associated with decision making and executive function. We recently found that prelimbic cortex (PL) inactivation in rats, via intracranial infusion of the GABA receptor agonists baclofen/muscimol, results in increased food seeking under punishment in rats. This effect has also been demonstrated in our previous studies following inactivation of the RMTg. Given the projections from the PL to the RMTg, and the finding that PL inactivation mimics the effects of RMTg inactivation on punished reward seeking, we hypothesized that the connection from the PL to the RMTg is important for suppressing reward seeking under punishment. The present study aims to further assess the role of these afferent projections from the mPFC to the RMTg. Eight Sprague Dawley male rats were operantly trained to press a lever to receive a food reward, which was paired with a foot shock of increasing intensity. Terminals from the mPFC projecting to the RMTg were inhibited using designer receptors exclusively activated by designer drugs (DREADDs) and microinjections of clozapine-n-oxide (CNO) administered locally to the RMTg through bilateral cannulas. A within subject design was used such that each rat received both conditions (infusions of CNO or vehicle) on alternating days. Preliminary data indicates that inactivation of PL terminals in the RMTg caused subjects to endure significantly higher shock intensities to receive food reward. These results are consistent with our previous studies and support our hypothesis that the mPFC-RMTg connectivity plays an important role in mediating aversion responses. This study gives us further insight into a broader circuit as it pertains to aversion and the suppression of reward-seeking under punishment.

1. BLUMENTHAL SA, PRATT WE

**The Effects of Serotonergic Agonists on Mu-opioid Induced Feeding**

*Department of Psychology, Wake Forest University*

**ABSTRACT:**

The consumption of palatable foods is selectively increased in rats following the stimulation of mu-opioid receptors in the nucleus accumbens. In contrast, certain serotonin agonists reduce feeding by causing increased satiety and hypophagia in rats. Despite this knowledge, there has been little prior research examining the potential interactions between serotonin and opioid systems on regulating feeding. In order to assess the effects of simultaneous opioid and serotonin receptor stimulation on feeding in rats, two experiments were conducted measuring 2-h intake of high-fat vegetable shortening. In the first experiment, rats received nucleus accumbens microinfusions of a mu-opioid receptor agonist (DAMGO; 0.025 microg/0.5 microl/side) with or without peripheral injections of the 5-HT releaser and reuptake inhibitor fenfluramine (0.0, 0.6, or 3.0 mg/kg) across six testing days. In a second experiment, rats received nucleus accumbens microinfusions of a mu-opioid receptor agonist (DAMGO; 0.025g/0.5l/side) with or without peripheral injections of a 5-HT2c receptor agonist (lorcaserin; 0.0, 0.3, 1.0 mg/kg). Rats in each group received all combinations of drugs, with a 48-h washout period between injection days. The order of the drug treatments in both experiments was counterbalanced across rats. Preliminary analyses of our data suggest that stimulation of a mu-opioid receptors in the nucleus accumbens through DAMGO microinfusions alone resulted in a significant increase in consumption of high-fat vegetable shortening, while both fenfluramine and lorcaserin injections resulted in dose dependent decreases in shortening intake. Fenfluramine and lorcaserin were also found to cause a dose-dependent decrease in DAMGO-induced feeding. This data suggests that serotonergic receptor activation inhibits normal intake of a fat diet, as well as binge-like feeding elicited by opioid receptor stimulation in the nucleus accumbens. These data support prior research suggesting the effectiveness of serotonin-promoting drugs for assisting in appetite control.

1. BRYANT KG, BARKER JM, CHANDLER LJ

 **(Travel Award Winner)**

**Regulation of behavioral flexibility by ventral hippocampus**

*Department of Biology, Program in Neuroscience, College of Charleston; Department of Neuroscience, Medical University of South Carolina*

**ABSTRACT:**

Habitual behavior may play a role in the development and maintenance of addiction and a greater understanding of the neuroanatomical substrates of habits can inform the development of new treatment strategies. Habits are inflexible behaviors that are no longer sensitive to changes in the action-outcome relationship, while goal-directed actions are behaviors which are sensitive to changes in this relationship. Thus, addiction can be explained in terms of a behavioral inflexibility caused by an inability to control one’s actions. One structure implicated in the expression of habits and regulation of behavioral flexibility is the nucleus accumbens shell (NAcS). The dysregulation of glutamatergic signaling in the NAcS is involved in the development of addiction and other substance abuse disorders. The NAcS receives glutamatergic input from a number of structures, notably including the ventral hippocampus (VH). We investigated the role of glutamatergic projections of the VH in habits, focusing on the VH to NAcS pathway. This was done using a chemogenetic strategy that allows for the investigation of specific neural pathways. Using this strategy, we injected a virus expressing an inhibitory Gi-coupled designer receptor into the VH of mice prior to operant habit training. In order to silence VH projection neurons, we administered the designer receptor agonist, clozapine N-oxide (CNO) either systemically via intraperitoneal injection or locally to the NAcS via cannula infusion prior to testing in order to silence either all VH projections or selectively VH-NAcS projections, respectively. We tested for the expression of goal-directed or habitual behavior by degrading the relationship (contingency) between behavior and its outcome in mice that received either CNO or saline. Both systemic and locally-infused mice that received CNO showed reduced responding when the contingency was degraded as compared to a non-degraded test (systemic p=0.03; prelim. local p=0.09), indicative of goal-directed behavior. In contrast, mice receiving a saline injection responded at the same rate when the contingency was degraded in both conditions, indicative of habitual behavior. This suggests that VH glutamatergic projection neurons are necessary for the expression of habits and that those neurons projecting to the NAcS specifically may be mediating this effect. Furthermore, this circuit may represent a future target for pharmacotherapeutic strategies to reduce addictive behavior.

1. COSTELLO JT, LAVIN A, PENA-BRAVO J

 **(Travel Award Winner)**

**Mechanisms Underlying Cognitive Deficits Following Repeated Methamphetamine Use**

*Department of Biology, Program of Neuroscience, College of Charleston; Department of Neurosciences, Medical University of South Carolina*

**ABSTRACT:**

Methamphetamine is a highly addictive psychostimulant that targets the pre-frontal cortex (PFC). This compound produces intense feelings of euphoria and increases concentration levels by flooding the synapses of PFC neurons with dopamine. Previous studies believe the impetus behind these physiological alterations to be methamphetamine’s ability to bind to the vesicular monoamine transporter 2 causing the release of dopamine from synaptic vesicles in the pre-synaptic neuron, and its ability to prevent the reuptake and recycling of dopamine from the synapse back into the pre-synaptic neuron. Methamphetamine has been shown to elicit working-memory deficits in individuals subjected to repeated exposure. The physiological actions of methamphetamine on dopamine transporters is well understood, however the mechanisms in the PFC that underlie working-memory deficits remain unclear. This study will test the hypothesized mechanism that working-memory deficits are produced with repeated methamphetamine administration (7 days) due to the over-excitation of interneurons in the PFC which innervate GABA pyramidal neurons, which results in PFC hypo-frontality. Rats (n=10) were exposed to a Y- maze memory paradigm in which their working-memories were tested via the association of a positive reinforcer (food) with the correct selection of a particular side of the maze. Correct selection percentages were compared to a baseline measurement, and across meth and control treatment groups. We detected a significant decrease in correct selection percentages in rats injected with methamphetamine (1 mg/kg), as compared to the baseline condition (p=.020). Whereas, the control treatment (saline) did not differ significantly from the baseline condition (p=.067). To better understand the mechanisms driving the short- term memory deficits, we are currently examining the potential electrophysiological changes in the PFC of ten mice using whole-cell clamp in voltage mode. The short-term memory deficits indicated by the behavioral trials, in addition to our hypothesized data we hope to yield from the electrophysiology experiments, will hopefully advance the development of novel therapeutics to alleviate short-term memory abnormalities in methamphetamine-addicts. NIH R25 DA033680 (AL)

1. COWEN MH, LIZARRAGA SB

**Understanding the Role of Rab3Gap1 in Neuronal Development**

*University of South Carolina*

**ABSTRACT:**

Mutations in the exocytosis regulatory gene RAB3GAP1 are associated with a rare disorder known as Warburg Micro Syndrome. This syndrome is characterized by severe mental retardation, slowed development, hypoplasia of the corpus callosum, and postnatal microcephaly, or failure of the brain to grow after birth. Understanding the function of Rab3Gap1 is of particular interest in studying neurodevelopmental disorders due to the symptomatic intellectual disability and developmental delay. The overall objective of this project is to understand the function of Rab3Gap1 in neuronal development. In order to accomplish this goal, we plan to use neuroblastoma SH-SY5Y cells, deficient for Rab3Gap1. We have optimized the neuronal differentiation conditions for SH-SY5Y cells. In particular, we used retinoic acid (RA) and brain derived neurotropic factor (BDNF). Successful differentiation was shown by immunostaining for TAU, an axonal marker. In addition, we have optimized the transfection protocols of differentiated cells and found higher transfection efficiency when conducting a double transfection over a 48-hour period. To determine the role of Rab3Gap1 in neuronal development, we are using short hairpin RNA (shRNA) to knockdown Rab3Gap1. The level of Rab3Gap1 knockdown was assessed using HEK293 cells transfected with shRNAs against Rab3Gap1. Western blot analysis indicates that Rab3Gap1 protein levels are reduced by 30% in transfected HEK293 cells. Using the reagents and protocols we have established, we plan to analyze changes in the morphology (size, axon growth, and branching) of differentiated neurons, as a result of Rab3Gap1 knockdown. Additionally, alterations in the autophagy pathway have recently been related to developmental processes and neurological disorders, making it of interest in the study of Warburg Micro Syndrome. We plan to investigate the relationship between Rab3Gap1 and the autophagy pathway by conducting co-localization studies in human neurons derived from stem cells. We hypothesize that postnatal microcephaly characteristic of Warburg Micro Syndrome arises from altered neuronal morphology, which is caused by impaired autophagy mechanisms.

1. DANIEL MA, QUINTERO G, GERHARDT G

**Ganging Enzyme-Coated Microelectrode Sites Produces Greater Sensitivity to an Analyte in a Biosensor**

*University of Kentucky*

**ABSTRACT:**

When neurons are excited and an action potential occurs, vesicles containing neurotransmitters are fused with the neuronal membrane and released into the synaptic cleft. Enzyme-based biosensors can be implanted at specific sites to measure these chemical signals. By studying a biosensor’s sensitivity to peroxide and to a particular analyte, it is possible to predict how responsive the biosensor is. In the current biosensor design, each of the four platinum sites on the microelectrode’s tip is wired to a recording channel in a miniaturized preamplifier via a connector. This study sought to examine the effects of ganging microelectrode sites together using modified connectors on the microelectrode’s analyte sensitivity, limit of detection, and level of noise; the two experimental connectors developed for this study either ganged all four sites into one recording channel in the miniaturized preamplifier or ganged all four sites into two recording channels. The peroxide and glucose calibration results showed that the slopes of the electrodes when connected to the modified connectors were much greater than the slopes of those same electrodes when connected to the control connector. The limits of detection for the two modified connectors were usually less than those of the standard connector, with the 4-in-1 connector generally having the lowest limits of detection. The noise level measurements did not show a consistent trend for the connectors, but the experimental connectors usually had slightly higher levels of noise than the control connector. An ANOVA test done on the hydrogen peroxide data confirmed that there was a statistically significant difference between the three connectors for both hydrogen peroxide slope and LOD, while the difference between groups for noise was not statistically significant. The ANOVA test done on the glucose calibration data found that only the difference in glucose slope was statistically significant between the three connectors.

1. DEMARCO JD, JONE T, CONNER WE

**Triggering jamming signals during the search and approach phase results in similar rates of capture in Eptesicus fuscus**

*Department of Biology, Wake Forest University*

**ABSTRACT:**

Bats rely on high-frequency echolocation to detect and capture their prey (Griffin et al. 1960). Echolocation calls contain three distinct phases that occur throughout prey to give the bat accurate information about its target (Schnitzler et al. 2003). These phases are distinguishable by the interval between the emitted calls (search > 5 ms; 12 ms < approach < 49 ms; buzz < 12 ms). Bat echolocation is susceptible to sinFM jamming signals from other bats (Corcoran and Conner, 2014) and there is a critical window (~2 ms) prior to onset of the buzz during which the signal is thought to be most effective (Miller 1991). One of the most common ways to test hypotheses concerning sonar jamming are playback experimentation, where the jamming call is broadcasted to a foraging bat. The inherent drawback of timing playback, as bat echolocation signals can vary too much to allow for automatic triggering parameters to be reliable, and manual triggering can cause the signal to be broadcasted too early (i.e. during the search phase). This experiment proposed that since all signals overlapped the critical window, there would be no difference in capture success if the signal was played during the search phase than if it was played during the approach phase. Ultrasonic microphones and infrared cameras were utilized in an outdoor flight cage to record the interaction between 3 male big browns attempting to capture a tethered moth while simultaneously being exposed to the jamming call of Tadarida brasiliensis.

1. DIXON K, ROTE A, FOO P

**The Effect Of Soccer Related Concussions On The Emotional Health Of Student Athletes**

*Department of Health and Wellness and Department of Neuroscience, University of North Carolina at Asheville*

**ABSTRACT:**

Traumatic brain injuries, specifically concussions, frequently occur in contact sports and may result in devastating effects on the cognitive ability and mental health of the athlete. This brain damage can cause down-regulation of the brain’s metabolism (hypometabolism) or structural damage to microtubules, resulting in reduced grey matter, which can cause symptoms such as: depression, emotional regulation difficulties and confusion. Less well-studied are the emotional deficits associated with concussions. Soccer is not usually thought of as a sport with a high risk of concussions, yet studies have shown that the risk of concussion is significant at the collegiate and professional level. The NCAA has a specific concussion protocol with guidelines for pre and post-concussion treatment and testing. When collegiate student athletes are freshman they are required to undergo baseline testing using the ImPact test, balance testing, and report any symptomatology and concussion history. These baseline tests are then compared with the same tests taken once an athlete has had a concussion. Post-concussion athletes are given medical treatment, assessments and a definitive diagnosis. They are then treated and follow return to play protocol. However, within concussion testing there is a lack of a thorough emotional test to determine if the athlete has any emotional deficits. This could lead to athletes being allowed to return to play with the potential risk of mental and emotional health problems. Our study examines male and female college soccer players from Universities along the East Coast and Tennessee to investigate emotional deficits due to their last sports related injury using a survey consisting of questions from the DERS and Beck Depression Inventory as well as compare these survey scores to their baseline and post-concussion Impact test scores. Our findings will potentially identify a positive correlation between concussions and the emotional symptoms in college soccer players. The research’s aim is to help add to concussion research to help prevent the risk of depression and future negative consequences of disruption of daily life due to concussions for this population. This study is currently ongoing, preliminary results and conclusions will be discussed as obtained.

1. FRITH ME, LEUNG K, RAMIREZ JJ

**Spatial Working Memory Performance on a Radial Arm Maze After Bilateral Fimbria-Fornix Transection in Rats**

*Department of Psychology, Davidson College*

**ABSTRACT:**

The fimbria-fornix is a major fiber pathway by which the hippocampal formation sends information regarding spatial working memory to the rest of the brain. This pathway also carries signals from the septum to the dentate gyrus. When the fimbria-fornix is intact, hippocampal signals bound for various structures in the associational cortex facilitate spatial navigation based on memories about locations of objects and features of the surrounding environment; the hippocampus simultaneously integrates information it receives from the septum. In this study, rats were trained on an 8-arm radial arm maze (RAM) before undergoing either a bilateral fimbria-fornix transection or sham craniotomy. Postoperative testing on the RAM was performed five days per week, for six weeks. In a delayed win-shift task, spatial working memory errors were quantified as reentries into an arm already visited during the trial. After six weeks of postoperative testing, animals were perfused with formalin and their brains sectioned. Acetylcholinesterase staining density in the outer molecular layer and supragranular zone of the dentate gyrus was assessed using optical densitometry analysis. Compared to control animals, rats with bilateral fimbria-fornix transections made significantly more spatial working memory errors and took longer to acquire the task. Analyses of subjects’ choice behaviors on the RAM, and insights into the behavioral consequences of dorsal versus ventral hippocampal deafferentation, will be discussed. This study follows up previous research in our laboratory, which suggested the possible presence of memory compensation mechanisms after bilateral hippocampectomy and entorhinectomy, respectively. The comparative absence of recovery by animals with bilateral fimbria-fornix transections in the present study provides interesting insights for disconnection approaches to studying hippocampal functioning.

1. GEIGER RE

**Cognition Enhancement Using Transcranial Direct Stimulation (tDCS)**

*Tennessee State University*

**ABSTRACT:**

Noninvasive brain stimulation called transcranial direct-current stimulation is a method for manipulating brain activity that is growing in prominence. This method allows cognitive scientists to increase neural activity in the vicinity of the anodal electrode and suppress activity in the vicinity of the cathodal electrode. This bi-directional control of neural activity makes this method a unique tool for understanding what different regions of the brain do. It is also safe and inexpensive, with effects lasting up to five hours. Transcranial direct-current stimulation uses a device powered by a 9 millivolt battery connected to electrodes that connect to the skin through saline soaked sponges or electrode jelly. Here I explain how transcranial direct-current stimulation can be used to understand how the brain receives, processes, and responds to external stimuli, specifically related to attentional tasks.

1. HARILL SA, HARTMANN DA, NOONAN T, SHIH AY

**Can Pericytes Regulate Blood Flow?**

*Department of Neuroscience, Medical University of South Carolina and Department of Biology, College of Charleston*

**ABSTRACT:**

Pericytes are small cells with processes that extend around the capillary endothelium, providing pericytes the ideal anatomical location to regulate blood flow by constricting capillaries. In the current study, we tested the longstanding hypothesis that pericytes have the ability to regulate blood flow by capillary constriction. We used ChR2-containing pericytes to stimulate pericyte constriction because ChR2 stimulation has been shown to cause constriction in smooth muscle cells. The control group had pericytes containing only YFP. Using two-photon microscopy, we optogenetically stimulated ChR2 and imaged hemodynamics. In the mice with ChR2, a large number of capillaries stopped flowing (17/42) upon optogenetic stimulation of pericytes. Importantly, almost no vessels stopped flowing (2/46) in the control group, which ensured it was ChR2 that was responsible for this change (p < 0.001). These data show pericytes can play a significant role in regulating blood flow. We intend to present data on capillary diameter and velocity changes to elucidate how pericytes mechanistically regulate blood flow. If pericytes have the ability to constrict and regulate blood flow, this may have implications for neurological diseases characterized by changes in capillary blood flow such as Alzheimer’s Disease and stroke. A potential therapeutic option would prevent pericytes from constricting, thus increasing blood flow.

1. HOK E, GESLAIN R

**Evaluating the neurotoxicity of unused transfer ribonucleic acids**

*Department of Biology, Neuroscience Program, College of Charleston*

**ABSTRACT:**

Transfer ribonucleic acids (tRNAs) are small non-coding RNA that carry specific amino acids to the ribosome for protein synthesis. Specific base pairing between tRNA anticodons (triplets of nucleotides) and codons on messenger RNAs (mRNAs) is central for accurate protein synthesis. Mispairing between tRNA codons and anticodons has been reported to cause protein misfolding and neurodegeneration. Developmental biology has shown low levels of mistranslation has a phenotype in the brain, but is virtually silent in the body. To investigate mistranslation effects in neurons, we looked at less frequently occurring anticodon. Their rare occurrence in the genome implies that some tRNA anticodons may have been selected against throughout evolution. For example, genes encoding tRNA alanine, proline, threonine, serine, arginine, and leucine, harboring GGC, GGG, GGU, GGA, GCG and GAG anticodons respectively, are virtually absent from the genome of vertebrates. Our collaborators in Strasbourg, France demonstrated, using non-cellular translation assays, that these six tRNAs species decode both corresponding and non-corresponding codons. Hybridization of non-corresponding codons is potentially toxic due to the incorporation of amino acid substitutions in the proteome. We hypothesize that these rare species named impossible tRNAs (itRNAs) have negative impact on the translation machinery. To validate our hypothesis, we synthesized in vitro six itRNA transcripts and their respective control and evaluated their toxicity by a colorimetric cellular assay in HeLa cells. HeLa cells were cultured in a 96-well plate, lipofected with tRNA, and incubated for 48 hours. The itRNA presented varying degrees of toxicity that correlate with the amino acid substitution they induced. This is consistent with our hypothesis, suggesting selective pressure against their presence in the genome. Future experiments will explore the toxic effects of mistranslation in additional cell lines, such as gliolblastoma and HEK293 cells, to explore tRNAs as potential therapeutic agents.

15. ISHINO Y, SOSSI S, CUSTER S

**Development of Molecular Tools to Study the Subcellular Synapse, Specificity of Cortical Interneuron Subtypes**

*Max Planck Florida Institute for Neuroscience*

**ABSTRACT:**

Chandelier cells (ChCs) are one of the most anatomically striking subtypes of interneurons (INs) and their particular morphology suggests that they have the most power to gate action potentials compared to any other cell type. A singular ChC forms synapses specifically on the axon initial segments (AISs) of multiple excitatory pyramidal neurons (PNs). Thus, ChCs are thought to play a critical role in learning, memory and in diseases such as epilepsy and schizophrenia. As a result, gaining further knowledge about the molecular and cellular mechanisms by which ChCs establish specific innervation of PN-AISs is necessary in order to understand how functional cortical circuits arise and for developing therapeutic strategies for neurological disorders.

Due to the highly specific connectivity of mature ChCs, we hypothesized that developing ChCs recognize AISs cellular substrates. To test this hypothesis we developed molecular reagents to disrupt the normal development of AISs in PN. The expression of Neurofascin (NF) and Ankyrin G (AnkG), two crucial proteins needed for the proper formation of AISs, was eliminated using the CRISPR/Cas9 system. The reagents were introduced into cortical PNs via in utero electroporation and the knockout was confirmed using the Surveyor system and immunostaining.

We found that using CRISPR/Cas9 against NF and AnkG successfully eliminated their expression in transfected PNs (labeled with EGFP). These results demonstrate that our molecular reagents were efficient in altering AIS structures and also in modifying ChC innervation. Therefore, these reagents can be utilized in future experiments to assess the involvement of PNs’ AISs in forming synapses with ChCs.

16. JACKSON J, TURCHAN M, VAN WOUWE N

**Action Control in Essential Tremor**

*Tennessee State University*

**ABSTRACT:**

Essential Tremor (ET) is one of the most common neurological disorders but the development of the disease is not quite understood. ET is a movement disorder that is usually classified by its action tremor, although more recently cognitive and emotional dysfunction have been reported as well, including impairments in executive control, dementia, depression and changes in visual reaction time (Benito Leon 2015). This suggests that ET is not just a motor disorder, however, there is still a limited understanding of changes in executive control with regard to ET. In this study examine differences in action control between subjects with Essential Tremor (ET) and Healthy Controls (HC) to increase our understanding of the cognitive impairments in ET. Specifically, we focus on action control as measured by the Flanker task. This task measures the ability to control impulsive responses that compete with goal-directed behavior. We found that the Flanker effect (measured as the difference in reaction times between corresponding and non-corresponding stimuli), was larger in ET patients compared to HC. That is, it takes ET patients more time to suppress irrelevant information as presented in non-corresponding stimuli. This suggests that the impairments in ET are not limited to simple motor deficiencies but extend to limitations in the ability to control behavior when conflicting information is present.

17. KREIBICH E, ASEMANI D, ROBERTS D

**WinSCAT scores as a Reflection of Functional and Neurocognitive Changes Following Long-term Spaceflight**

*Department of Psychology and Program in Neuroscience, College of Charleston; Department of Radiology, Medical University of South Carolina*

**ABSTRACT:**

As the National Aeronautics and Space Administration (NASA) continues to plan future space missions, many of which will last for several months or longer, it is important to understand the health consequences for the astronauts involved. Previously, it was not believed that a zero gravity environment greatly impacted the central nervous system of astronauts. As a result, NASA lengthened the duration of space missions, an action which has resulted in an emergence of neurological symptoms among long-term crew members, including visual changes and increased intracranial pressure. However, little is known about the impact of long-term spaceflight, spaceflight lasting for several months, on cognition. NASA has developed a computerized test to assess astronauts for changes in cognition that reflect underlying neurological problems. The Space Flight Cognitive Assessment Tool for Windows (WinSCAT) consists of five tasks performed on a computer before, during, and after spaceflight that test a variety of essential cognitive functions, including working memory, sustained attention, basic calculations, visual search, verbal learning, pattern recognition, and recall. For each of the five tasks, there are subscores that represent response time, accuracy, and cognitive efficiency. In this study, we analyzed the WinSCAT scores of astronauts that underwent long-term spaceflight. We expected to see a decrease in WinSCAT scores, reflecting impairment of various cognitive functions. We averaged three of the subscores for each category together to get an overall score for each WinSCAT task. Statistical comparisons between before, during, and after spaceflight scores were performed for each task. We found that the composite score for each WinSCAT session, the code substitution task score, and the continuous processing task score increased over the course of the mission. This indicates that extended time in space is actually associated with enhanced cognition, specifically on tasks that involve concentration, visual search, verbal learning, recall, and working memory. These results contradict our hypothesis that cognition declines over the course of extended spaceflight, indicating that long-duration missions may be pursued without great harm to the nervous systems of the astronauts.

18. LAHIRI S, BAKER CA, BOLTON MM

**Dendritic Morphology in Mice with Defective Cleavage of the Autism Spectrum Disorder-Linked Protein Neuroligin-1**

*Disorders of a Neural Circuit Lab, Max Planck Florida Institute*

**ABSTRACT:**

Currently there is a growing list of genes that are potentially susceptible to autism spectrum disorders (ASD). Dr. Mclean Bolton’s Disorders of a Neural Circuit lab aims to characterize the neuronal alterations in ASD animal models. Studies have shown that copy number variation or point mutation in neuroligin proteins are linked to ASD. Neuroligin is a postsynaptic cell adhesion molecule which plays a role in neural connectivity. Increased neuronal activity leads to neuroligin cleavage, which could affect synapse formation and elimination in neural circuits. Functional structure analyses have shown linkage between ASD and hyperconnectivity in the amygdala. The objective of this project was to examine changes in dendritic morphology in the amygdala of the SD3 mouse, in which normal cleavage of Neuroligin-1 is inhibited.

19. LEE DC, SHARKO AC, KAIGLER KF, FADEL JR, WILSON MA

**Activation of Hypothalamic Orexin Neurons during Extinction of Fear Memories**

*Department of Physiology, Pharmacology, and Neuroscience, University of South Carolina - Columbia*

**ABSTRACT:**

Post-traumatic stress disorder (PTSD) is a prevalent anxiety disorder that can occur after a serious traumatic event such as serving in the armed forces or a natural disaster. However, not all people who experience a traumatic event develop PTSD which indicates that some neurobiological mechanisms may make some individuals more or less susceptible to the disorder. Long Evans rats have been shown to exhibit individual differences in cue-induced freezing during extinction of fear memories suggesting this outbred strain could serve as a useful model for PTSD. The neuropeptide orexin (hypocretin) has been shown to preserve fear responses during extinction of fear memories. Although orexinergic neurons are located in the hypothalamus, they project to areas of the brain associated with fear extinction (e.g., amygdala and prefrontal cortex). This study tested the hypothesis that individual differences in fear extinction will lead to differential activation of orexin neurons. Three cohorts of rats were exposed to three tone-shock pairings, followed by extinction training two days later in a novel environment with twenty cue (tone) presentations. Brains were collected either after the extinction learning trial or after an extinction recall trial two days later. Rats were divided into good and poor extinction groups based on their freezing during the last ten minutes of the extinction learning trial. The percentage of activated orexinergic neurons in the lateral and medial hypothalamus were examined through dual label immunohistochemistry for orexin-A and cFos. Rats showing resistance to extinction learning (high freezers) had a significantly greater percentage of orexinA neurons with cFos in the medial hypothalamus than low freezers following the extinction recall trial. Interestingly, no individual differences in the activation of orexin neurons were observed in the lateral hypothalamus, or in the medial hypothalamus following extinction learning. These data suggest that orexinergic neurons in the medial hypothalamus may contribute to differential extinction learning.

20. LEE DM, HYUN JH, JUNG K, HANNAN P, KWON HB

**Calcium- and Light-gated switch of neuronal activity for gene expression**

*Department of Cellular Basis of Neural Circuit Plasticity*

**ABSTRACT:**

The identification and manipulation of an active population of neurons during a specific action or perception have been a long-standing challenge in neuroscience. We developed a dual protein switch system named Calcium and Light-Induced Gene Handling Toolkit, “Cal-Light”, that drives neuronal activity-dependent gene expression in response to light and calcium. This technique targets individual neurons for specific gene expression and fine-scale labeling, allowing the single cell activity profile of a given neuronal population to be mapped out. In vivo viral injection of Cal-Light into the motor cortex successfully labeled a subset of excitatory and inhibitory neurons related to lever-pressing motor behaviors. Silencing the activity of the labeled neurons impaired the learned behaviors, indicating that the Cal-Light system can effectively assess a behaviorally relevant neuronal population in a genetically identifiable manner. Thus, the Cal-Light system greatly improves the ability to dissect neural circuits underlying complex mammalian behaviors with high spatiotemporal precision.

21. LEROY V

**Validation of siRNA against LPA receptor 4 to analyze retinal ganglion cell guidance**

*Winthrop University*

**ABSTRACT:**

The optic nerve fully develops during the embryonic stages of growth, and once damage has been done to the optic nerve, regeneration in adult mammals does not occur naturally. We are using the chicken embryonic system to study the developing nervous system, specifically the developing optic nerve. The optic nerve is made up of axons from retinal ganglion cells, or RGCs, which are neurons that transmit visual information from the eye to the brain. RGCs reach their destination in the tectum (which is a structure of the chicken brain that is analogous to the mammalian superior colliculus) by relying on a growth cone to detect environmental cues. Lysophosphatidic acid (LPA) may be one such environmental cue that has a role as a repulsive cue to guide the axon to its target in the tectum. LPA binds to six known LPA receptors, which are G-protein coupled receptors (GPCR) that work through the G12/13 pathway to induce growth cone collapse. Previous work has suggested that 5 of the receptors are not involved in axon guidance, leaving lpar4 as the remaining receptor that likely mediates this growth cone collapse. By designing siRNAs (small interference RNA) against lpar4 we can reduce its expression, which allows us to determine its role in axon guidance. We are testing our constructs by transfecting the siRNA into a neuroblastoma cell line, which overexpresses lpar4 with a myc protein tag. In order to validate the siRNA, RNA will be isolated from the transfected B103 cells and quantified through qRT-PCR. A decreased concentration of lpar4 RNA compared to the non-transfected B103 cells would suggest that our siRNA was successful in knocking down the RNA responsible for generating lpar4. The lpar4 protein levels are analyzed via Western blot against the myc tag to further demonstrate lpar4 siRNA knockdown. After successful validation, we will inject our siRNA into the developing eye of chicken embryos and investigate growth cone response to LPA in the absence of lpar4.

22. LONG ME, JONES GC, WARREN JW, MOTT DD

**Cortical and Subcortical Projections to the Amygdala**

*School of Medicine, University of South Carolina*

**ABSTRACT:**

Post-traumatic stress disorder is a fear-related disorder that afflicts nearly 24 million adults. The amygdala is a brain structure that plays a vital role in fear processing involved in disorders like PTSD. A detailed understanding of the anatomy and connectivity of the amygdala provides critical information for advancements in therapeutic research. The prelimbic cortex and the thalamus are brain regions important in the fear processing that have projections to the amygdala. In this study, we used anterograde tract tracing with an adeno-associated virus (AAV) that encodes yellow fluorescent protein (YFP), to define the termination fields of these neuronal projections in the amygdala. Preliminary studies defined stereotaxic injection sites and compared expression and transport of different serotypes of the virus (AAV2 vs. AAV5). These studies also determined the time frame needed for optimal YFP expression. Data demonstrated that AAV5-CAG-YFP was better contained to the injection site than AAV2-CAG-YFP and that the optimal expression of YFP in the amygdala was at approximately 5 weeks. Two groups of adult, male B6129SF2/J mice were then injected in either the prelimbic cortex or the thalamus with AAV5-CAG-YFP. The injected YFP traveled down axons that terminated in anatomically defined amygdalar nuclei. After 6 weeks, the extent of the axonal projections was quantified in each nucleus. Results demonstrate that projections from different brain regions terminate in distinct subregions of specific amygdalar nuclei. These results support a differential role for amygdalar subregions in fear and emotional processing. Supported by NIH R01 MH104638 and the Magellan Scholars Program.

23. MARINELLI NA, GAMZIS DE, DOUGLAS A, MCGUIRE A, COWEN MH, MORROW EM, LIZARRAGA SB

**A human neuronal transcriptome study on and ASD environmental risk factor**

*Department of Biological Sciences, University of South Carolina*

**ABSTRACT:**

Autism Spectrum Disorders (ASD) affect 1 in 68 children, often resulting in impaired language development, social interactions, and increased repetitive behaviors. ASD is challenging to study because it is a highly heterogeneous and heritable disorder, yet the known genetic etiology constitutes only 25%. These suggest an association between environmental risk factors and ASD pathology. Whole exome analysis showed high risk variants of genes encoding chromatin and transcriptional regulatory proteins associated with ASD, suggesting a potential role for epigenetic regulators as mediators of environment risk factors for ASD. Previous studies have identified prenatal exposure to the anti-epileptic and mood enhancement drug Valproic Acid (VPA) as an ASD environmental risk factor. VPA is a Histone Deacetylase (HDAC) inhibitor. HDACs are important in regulating both gene transcription and the differentiation of neural stem cells. However, the molecular mechanisms underlying VPA associated ASD are largely understudied. The primary objective of this study is to unravel the molecular mechanisms underlying ASD pathology associated with in utero exposure to VPA. We conducted RNA sequencing (RNAseq) on VPA and control treated 65 day old iPSC-derived human neurons. Using differential expression analysis, we found 1048 genes significantly upregulated, and 1777 genes significantly downregulated in human neurons treated with VPA. To confirm the results of our RNAseq studies we used Quantitative PCR (QPCR) on 12 differentially expressed genes that have been previously identified as high risk autism candidate genes. RNA isolated from human neurons was converted into cDNA and subjected to SYBR green QPCR. Preliminary results confirmed that these 12 candidate genes are in fact downregulated when neurons are exposed to VPA. Differentially expressed genes were further analyzed for pathway enrichment using DAVID gene ontology. We find that upregulated genes clustered on biological processes that were primarily associated with mitochondrial function, while downregulated genes primarily clustered in chromatin regulation and transcriptional cellular processes. To further assess the relevance of our differentially expressed genes to ASD pathology we conducted a comparison analysis with previously published VPA studies on human stem cells. We identified a cohort of 200 neuronal genes that were significantly downregulated in our study as well as in 11 other studies.

24. MCDOUGLE MJ, GLOVER EJ, CHANDLER LJ

**Effect of adolescent intermittent ethanol exposure on choline acetyltransferase expression**

*Department of Biology and Program in Neuroscience,*

*College of Charleston; Department of Neuroscience, Medical University of South Carolina*

**ABSTRACT:**

Adolescence is characterized by increased risk-taking and the pursuit of novel stimuli, including drugs and alcohol. Binge-drinking and heavy alcohol use during adolescence has been linked to long-term cognitive deficits and increased risk of developing alcohol use disorders in adulthood. The cholinergic system serves to regulate cognitive function, sleep, wakefulness, reward and aversion, all of which previous studies have shown to be altered by prolonged alcohol exposure. The present study was designed to investigate the effects of adolescent alcohol exposure on the adult cholinergic system. Pair-housed male Long-Evans rats received intermittent ethanol vapor exposure in a binge-like, two-day-on, two-day-off pattern for a total of four cycles between postnatal day 28 and 42 (n=11), the period corresponding to early adolescence in rats. Control rats were exposed to air in a similar fashion (n=11). Rats were subsequently sacrificed in adulthood between postnatal days 90-120 and brains were processed for choline acetyl transferase (ChAT) visualization, an enzyme involved in synthesis of the neurotransmitter acetylcholine and which serves as a marker of cholinergic neurons, using standard avidin-biotin immunohistochemistry and immunofluorescence methods. ChAT expression was measured in brain regions expressing dense ChAT-positive cell bodies or fiber staining including the medial septum, nucleus basalis, pedunculopontine tegmental nucleus, laterodorsal tegmental nucleus, medial habenula, interpeduncular nucleus, basolateral amygdala, striatum, and prefrontal cortex. In rats exposed to ethanol during adolescence, ChAT expression was significantly reduced in the laterodorsal tegmental nucleus (p≤0.01), an area involved in signaling reward and aversion, compared to controls. Significant between group differences were not observed in any of the other regions measured. These results differ from previous work reporting global reductions in ChAT expression in adult male Sprague Dawley rats following intragastric ethanol exposure during adolescence. Thus, adolescent ethanol-induced changes in ChAT expression may depend on rat strain or route of ethanol administration. Future studies will focus on parsing out the effect of rat strain on adolescent ethanol-induced changes in ChAT expression, as well as understanding the functional consequences of adolescent ethanol exposure on cholinergic pathways involved in signaling reward and aversion.

25. MEDINA NIETO T, TRAWINSKI A, FAHRBACH SE

**The effect of queen pheromone exposure on the growth of the worker honey bee brain**

*Department of Biology, Wake Forest University*

**ABSTRACT:**

Queen mandibular pheromone (QMP) is a pheromone produced by the queen honey bee that suppresses ovary development in worker honey bees. When exposed to QMP, the ovaries of worker honey bees are not only suppressed, but they can also produce ecdysteroids. The amount of ecdysteroids produced is influenced by the age of exposure to QMP. Ecdysteroids are steroid hormones responsible for development and reproduction in insects. During the metamorphosis of the honey bee, ecdysteroids promote neuronal growth. In this study, we focused on a central region of the insect brain critical for learning and memory called the mushroom bodies to ask if QMP, ecdysteroids, or a combination of both signals shape the adult brain. We used a competitive enzyme immunoassay to determine ecdysteroid levels in caged honey bees exposed to QMP and/or injected with ecdysone. We then used an antibody to a presynaptic protein to assay changes in the synaptic structure in the brain. Studies are ongoing, but we predict honey bees injected with ecdysone and responders to QMP will have high ecdysteroid levels and an increase in synaptic connections.

26. MURPHY AJ, SATTERFIELD R, YOUNG JR. SM

**Development of a Helper-Dependent Adenoviral vector for the expression of Munc13 proteins**

*Research Group of Molecular Mechanisms of Synaptic Function, Max Planck Florida Institute for Neuroscience*

**ABSTRACT:**

Helper-Dependent Adenoviruses (HdAd) are the ideal viral vector choice for expression of Munc13 proteins due to the large packaging capacity needed (~7.7 kb) for the dual expression cassettes of Munc13 and EGFP. HdAd vectors are useful for a wide range of applications in neuroscience, including rapid onset and high neuronal specificity of transgene expression in vivo. HdAd vectors are devoid of viral genes which increases the packaging capacity of the vector up to 37kb of foreign DNA. The increased carrying capacity is not obtainable for other recombinant viral vector technologies such as rAAV and LVV. The lack of viral genes also eliminates the possibility of chronic toxicity as there is no chance for residual viral gene expression. The large carrying capacity, neuronal specificity and lack of toxicity in conjunction with the ability for rapid onset of transgene expression and easy scalability making HdAd vectors the ideal choice for CNS applications. Development of a HdAd vector for the expression of Munc13 gene family proteins allows for the rapid expression of molecules located at the synaptic active zone critical for neurotransmitter release. The Munc13 HdAd viral vector can be used to express proteins of the munc13 gene family in the calyx of Held allowing for the elucidation of the molecular mechanisms employed by the calyx of Held in the process of encoding the onset and modulation of sound production of an HdAd vector for the expression of Munc13 proteins occurs in five parts; cloning, transfection, amplification, purification and tittering. After production of the HdAd vector, Munc13 protein expression was analyzed by western blot.

27. MURPHY JE, KIRKLAND A, YANES JA, KIRBY LAJ, JANTZEN B, REID MA, ROBINSON JL

**Left, right, or bilateral amygdala activation? How the effects of smoothing and motion correction on ultra-high field, high-resolution functional magnetic resonance imaging (fMRI) data alter inferences**

*Department of Psychology, Auburn University*

**ABSTRACT:**

Background: Functional magnetic resonance imaging (fMRI) data is incredibly complex and heavily influenced by subjective decisions in preprocessing, possibly resulting in vastly different inferences to be drawn from the same data. Further complicating the issue, very little is known about the effects of preprocessing on submillimeter fMRI data, especially at ultra-high field strengths greater than 3 Tesla. Spatial smoothing and motion correction are normal preprocessing steps with many published recommendations for standard or greater than 1mm resolution data.

Method: We analyzed fMRI data from 30 healthy individuals collected at sub-millimeter in plane resolution and used a field standard preprocessing pipeline with different combinations of smoothing kernels and motion correction options to determine the impact they have on activation patterns associated with a well-documented affective task.

Results: Brain activation patterns change significantly with different combinations of smoothing and motion correction. These differences have implications in the interpretation of the data, particularly in small structures where we found that preprocessing choices led to either unilateral or bilateral amygdala activation.

Comparison with Existing Methods: Common recommendations for smoothing kernels with lower resolution fMRI data based on voxel size are not applicable for submillimeter data. However, recommendations made for choosing smoothing kernel size based on size of brain structure of interest remain valid but must be tempered with the total effect preprocessing steps have on the data.

Conclusions: These results suggest synergistic effects of spatial smoothing and motion correction, and highlight the importance of rigorous investigations on the effects of preprocessing.

28. NOONAN TE, HARTMANN DA, HARRILL SA, SHIH AY

**Optogentically Stimulated Pericytes have Transient Effects on Blood Flow**

*Department of Neuroscience, MUSC and College of Charleston*

**ABSTRACT:**

The brain has an extremely high demand for blood supply. The cerebrovasculature, covered by smooth muscle cells and pericytes, is responsible for the maintenance of blood flow. Smooth muscle cells are known to regulate blood flow through arterioles, but it is unknown if pericytes on smaller vessels called capillaries modulate blood flow. In this experiment, we used transgenic mice expressing channelrhodopsin-2 (ChR2) in smooth muscle cells and pericytes in order to clarify the role of pericytes in blood flow regulation. We stimulated individual pericytes through an acute skull-removed cranial window with two-photon wavelengths that excite ChR2, and measured any resultant changes in capillary blood flow. We insured that we were imaging pericytes and not smooth muscle cells by targeting vessels in the middle of the capillary bed (5-9th branch order), where certain smooth muscle proteins are no longer expressed. We found that optogenetic stimulation of individual pericytes halted blood flow in a substantial proportion of associated capillaries (~30%) when looking ~1 minute after the stimulus was applied. Importantly, control animals expressing YFP and not ChR2 in pericytes were imaged identically but showed negligible stoppages in flow. After 16 to 24 hours, we found that the non-flowing capillaries had re-established blood flow. Long term effects were not statistically significant when compared to the YFP control group. These data are the first suggest that optogenetic stimulation of pericytes produces transient effects on blood flow.

29. PATEL NV, HUGHES M

**Neuroplasticity and behavioral consistency related to trauma and recovery in snapping shrimp**

*Department of Biology, Program in Neuroscience, College of Charleston*

**ABSTRACT:**

Consistent individual differences in behavior (i.e., “personality”) are found across animal taxa, and thus understanding neural mechanisms underlying animal personality is a central question in behavioral neuroscience. When animals undergo trauma, plasticity in both behavior an underlying neural mechanisms may be required. Snapping shrimp are an ideal model system for studying neuroplasticity and personality through trauma and recovery: these shrimp have a large snapper claw and a small pincher claw, each specialized for different behaviors, and when the snapper claw is lost or autotomized, the small pincher is transformed into a snapper claw, a process requiring extreme nervous system reorganization in both motor control and sensory input. Snapping shrimp exhibit consistent individual differences in reactivity as measured by latency to snap in response to a threat. We found that shrimp show a change in reactivity following autotomy and transformation, with the changed reactivity being maintained through at least 2 molt cycles of recovery. Two hypotheses could account for this change in reactivity: (1) shrimp had not yet fully recovered, predicting that shrimp revert back to their original baseline if allowed more molt cycles to recover from the trauma of claw autotomization; (2) control of reactivity is lateralized, predicting that shrimp revert back to their original personality if the shrimp return to their original handedness following a second autotomization. In this study, we are testing these hypotheses. Animals either autotomized their large snapper once, twice, or served as controls, in which no autonomization occurred. We began by obtaining ten trials of pre-autotomy reactivity latencies for each shrimp (n=60) to determine their baseline snap latency. Reactivity of each shrimp throughout the experiment will be compared to the baseline. Currently, we are in the process of collecting post-autotomy snap latency on both groups, and are continuing to collect data on the controls. Through this research we hope to determine what causes the behavioral differences associated with stress, trauma, and recovery.

30. PATINO EM, ROWAN MJ, CHRISTIE JM

**Activity-Dependent Labeling of Cerebellar Interneurons during Motor Learning**

*Max Planck Florida Institute for Neuroscience; Department of Biology, Florida Atlantic University*

**ABSTRACT:**

The cerebellum is involved in coordinating movement and participates in motor skill learning. Cerebellar dysfunction is associated with loss of balance and motor control, thus it is important to understand the cellular mechanisms underlying learning within its circuits. Motor learning in the cerebellum is thought to be encoded through modification of the synaptic inputs onto Purkinje cells, the sole output neurons of its cortex. Purkinje cells receive both excitatory and inhibitory inputs. Although a long-standing hypothesis posits that excitatory synapses are the main locus of plasticity, inhibitory inputs from molecular layer interneurons (MLIs) are also potential sites for motor memory storage. Therefore, we are using a novel memory-labeling system to identify if MLIs are engaged during motor learning and if so, examine if their synapses are altered to express adaptive behavior.

To study cerebellar learning, we are using a well-known motor-learning paradigm, adaptation of the vestibulo-ocular reflex (VOR). The VOR results in an equal and opposite eye movement in response to head rotation, maintaining image stability on the retina. The flocculus, a specialized region of the cerebellum, is responsible for the proper calibration of the VOR. If motion is detected on the retina, activity in the flocculus results in a corrective motor response generating either an increase or decrease in eye movement.

To tag cells activated during VOR motor learning, we used a genetic approach relying on a reporter construct that uses the activity of the immediate early gene cfos to drive the expression of the fluorescent protein GFP. This system offers the ability to conditionally limit the reporter system by means of doxycycline (Dox) administered in the diet. Dox suppresses cfos-dependent GFP production until administration is discontinued. We used Cre-dependent (FLEX) AAV to limit this reporter construct to MLIs through an MLI-specific Cre mouse line. Quantification of MLIs expressing GFP allowed the analysis of the proportion and identity of MLIs during motor adaptation. After training resulting in a learned increase in eye movement, the majority of AAV-induced MLIs were found to express GFP. This indicates that the fos-GFP system can successfully label MLIs in response to motor learning and that a majority of MLIs are engaged during the learning process.

31. PEGELOW ME, MAVI S, GRIGORYAN DA, CLELAND CL

**What is the Nociceptive Withdrawal Response of Unrestrained Rats when Noxious Stimulation is Delivered to the Tail or Feet?**

*James Madison University*

**ABSTRACT:**

The nociceptive withdrawal response (NWR) is a protective response to a noxious stimulus. The response can vary due to many factors, including stimulus intensity, stimulus location and posture. In previous studies from our laboratory, rats were restrained in an acrylic tube during stimulation. It was observed that rats, when given a heat stimulus to the tail, moved their tails in the opposite direction of the stimulation site. It is unknown however, whether they concurrently exhibit postural body movements. The specific aim of this study was to examine postural changes in the body of rats evoked by a noxious heat stimulus to the tail or foot in an unrestrained, as opposed to retrained, setting. Sprague-Dawley rats (n=7) were first anesthetized with isofluorane and black marks were placed on the feet, tail, and body to target stimulation and track movement of the animal. Rats were then placed on an open glass table with a video camera (60 fps) positioned underneath to capture movement of the tail, feet and body. Localized stimuli were delivered via an infrared laser to one of five points on the tail or the plantar surface of one of the four feet. Using the recorded video and a tracking software (ProAnalyst), tail, foot, and body movement frames were recorded and changes in body angle, initial/final foot positions and number of steps were tracked. We found that when the tail or feet were stimulated, the stimulated body part was withdrawn between 1 and 3 seconds following stimulation nearly 100% of the time; however rats exhibited varying escape strategies depending on if the tail or feet were stimulated. When the tail was stimulated, rats tended to exhibit initial tail movement followed by a 180° rotation to face the stimulus. When the feet were stimulated, there was far less turning and the rats tended to simply shift their body weight with minimal foot movement. Though these responses were relatively consistent among the rats, it is important to note that individual rats responded qualitatively differently. In summary, rats appear to use similar local withdrawal strategies, but different postural strategies. Consequently, testing rats while restrained may miss these associated and potentially functionally important postural strategies

32. PEL AV, LOM BM

**Ethanol Compromises Xenopus laevis Development**

*Department of Biology, Davidson College*

**ABSTRACT:**

The widespread consumption of alcohol has been the focus of many studies attempting/aiming to understand its effect on the body and embryonic development. The central nervous system is particularly vulnerable to alcohol exposure during embryogenesis. Alcohol consumption during pregnancy has been linked to cognitive deficiencies, behavioral problems, and other functional impairments. The mechanisms by which ethanol impairs neuronal development are complicated. Alcohol is known to decrease white matter and damage neural stem cell progenitor pools that supply neurons and glia. In humans, ethanol exposure causes smaller head sizes, below average heights/weights, smaller eye sizes, and narrower eye distances. This distinctive developmental phenotype and associated neurological damage has been termed fetal alcohol syndrome (FAS). Model organisms such as the frog Xenopus laevis are also sensitive to ethanol during development. This experiment investigated the effects of ethanol on tadpole embryogenesis. Tadpoles were reared in 0 - 2.5% (v/v) of ethanol for two or six days. Body lengths and inter eye distances were measured using ImageJ. We observed a negative correlation between ethanol concentration and both body length and eye distance. There was a ~30% decrease in body length at both day two and day six of development. We also observed a ~60% decrease in eye distance at six days of development. Additional observations included bloated abdomens, curled body shapes, and reduced eye sizes. Taken together, these results suggest that ethanol can induce FAS-like gross morphological differences in a developing aquatic vertebrate, a potentially useful model for elucidating mechanisms by which ethanol compromises CNS development.

33. PINEDA G, GHATE P, LIZARRAGA SB

**Modeling gene-environment interaction in autism spectrum disorders with stem cell technology**

*Department of Biological Sciences, University of South Carolina*

**ABSTRACT:**

Autism spectrum disorders (ASD) affects 1 in 68 children(Park et al., 2016), is associated with deficits in verbal communication, social behaviors and the presence of repetitive behaviors. ASD represents a growing public health concern worldwide. The cost of taking care of ASD patients is an average of $1.4 million per individual during their lifetime. Behavioral intervention therapies can help some individuals but are not equally effective in all cases. A large proportion of ASD cases are of indirect genetic etiology; however, the relevance of the gene-environment interaction in the pathogenesis of ASD remains largely understudied. A number of studies point to a role of maternal immune activation in ASD etiology; yet the mechanisms that underlie this correlation are largely unknown. Animal models of maternal immune activation (MIA) exhibit abnormal behaviors reminiscent of ASD (Malkova et al., 2012). The increase in ASD-like behaviors in MIA models is mediated by IL-17A cytokine (Choi et al., 2016). IL-17A is a pro-inflammatory cytokine produced by CD4+T cells (TH17 cells). Despite the available data on the role of inflammation in ASD etiology there is very little known regarding how inflammation affects the development of human neuronal circuitry. We aim to determine the cell autonomous role of neuronal inflammatory mechanisms underlying defects in neuronal connectivity associated with ASD pathology. Our central hypothesis is that increased levels of IL-17A will impair the development of human neuronal connectivity. We are testing this hypothesis by using human stem cell derived neural models (Mariani et al., 2015; Mariani et al., 2012). In particular we are analyzing, the effect of IL-17A in neuronal cell fate determination, neuronal morphogenesis and gene expression in human neurons. We expect that determining the contribution of the different components of the Central Nervous system (CNS) and immune system will allow us to dissect the cellular mechanisms that underlie the role of neuronal inflammation in ASD pathology.

34. RATLIFF KE, FRANSSEN CL, EAGLE AK, ZIMMERMAN CA

**Working in Nature: Behavioral Neuroendocrinological Measures of Stress and Well-Being in Yellowstone National Park**

*Longwood University*

**ABSTRACT:**

When people spend time outdoors, they report feelings of well-being and reduced stress levels. Service providers and clinicians in the field of Outdoor Behavioral Healthcare (OBH) have validated anecdotal evidence with surveys and other behavioral assays; however, analyses of physiological responses to natural elements and environments is extremely limited. To date, while many OBH interventions involve long-term exposure to nature, most experimental work has focused on acute exposure to an outdoor environment. Here, we studied chronic exposure (~3 months) via seasonal employees in Yellowstone National Park (YNP). Employees of the Canyon Lodge in YNP participated by completing a survey of anxiety and well-being measures at the beginning, mid-point and end-point of their 3 month stay. Additionally, participants provided saliva samples at these three time-points. Saliva was frozen and shipped back to Longwood University for analysis. While overall effects are not easily seen in this small sample size, the results of both survey and salivary results suggest that demands of the job and environment may affect cortisol levels more than outdoor exposure, and indicate that different measures and time-points may be more appropriate to answer the question of efficacy of anxiety-reduction resulting from outdoor exposure. Details of conducting an out-of-lab undergraduate research experience will be shared along with the results of this study.

Keywords: cortisol, stress, nature, national park.

35. REED E, SILVER WL

**The Search for Chemoreceptors in the Earthworm (Eisenia hortensis)**

*Department of Biology, Wake Forest University*

**ABSTRACT:**

Earthworms are often used as biomarkers for healthy soil as they aerate and enrich the soil providing a favorable habitat for plants. Deviations in the soil’s chemical composition may affect the distribution of worms, which in turn, could greatly impact soil quality. As a first step to determine how earthworms detect environmental chemicals, we have devised several behavioral assays. Virtually nothing is known about how earthworms detect appetitive chemicals. Previous feeding assays suggest that earthworms are attracted to 2 volatile chemicals commonly produced by fungi, ethyl pentanoate (EP) and ethyl hexanoate (EH). In the current study, a T-maze was used to examine the worm’s appetitive behavior to these 2 compounds. One arm contained soil and mineral oil/water (control) and the other arm contained soil, mineral oil/water and the stimulus. Our data clearly show that EP and EH are aversive to the earthworm, Eisenia hortensis, and not attractive as reported in the previous study. As the concentration of EP and EH increased, so did the percentage of worms choosing the control soil.

In terms of aversive behavior, allyl isothiocyanate (AITC) is used to sample the number of earthworms in an area of soil by chemical expulsion. This clearly demonstrates that earthworms have the ability to detect AITC. In our experiment, a simple burrowing assay was used to characterize earthworm responses to AITC. A worm is placed in a cup containing soil and mineral oil/water (control) or soil, mineral oil/water, and 10mM AITC. 100% of the worms placed in the control cups quickly burrowed into the soil, however, 100% of the worms placed in the AITC cups quickly climbed out of the cups. We suspect that TRP channels, which are involved in irritant detection in organisms ranging from C. elegans to humans, are also used by earthworms to detect aversive chemicals. We used the TRPA1 channel blocker, H3-030031 (10μM), to examine whether AITC might be stimulating TRPA1 channels. Worms were submerged in the blocker for 5 minutes and the burrowing assay was repeated. At the end of the 10 minutes, 50% of the worms were found to still be in contact with the AITC-laced soil suggesting that the blocker rendered them insensitive to AITC. We are currently using electrophysiological, immunohistochemical, and molecular biological assays to determine what receptors might be used by earthworms to detect chemicals in the environment.

36. ROBBINS I, ANDERSON RI, BECKER HC, LOPEZ MF

**Habitual Responding for Alcohol in C57BL/6 Mice**

*Department of Biology and Program in Neuroscience, College of Charleston; Addiction Sciences Division, Department of Psychiatry, Medical University of South Carolina*

**ABSTRACT:**

Habitual drug-seeking is one of the fundamental mechanisms underlying alcohol use disorders. While initial drinking behavior is typically goal-directed (driven by the rewarding properties of the drug), prolonged exposure may lead to habitual alcohol seeking. This shift in behavioral control reflects a critical step in the development of alcohol related disorders. Preclinical models of habitual intake can serve as useful paradigms for examining the distinction between habitual vs. goal-directed alcohol seeking and the underlying neural mechanisms modulating each behavior. However, it is important to differentiate habitual from compulsive drinking behavior. Alcohol seeking often persists despite the development of aversive consequences, posing a challenge in the formation of effective therapeutic interventions. The current study sought to evaluate whether altering the taste of an alcohol solution would affect the rate of responding for alcohol reinforcement in mice. In addition, we examined the effect of foot shock as an aversive consequence on the rate of responding. Adult male C57BL/6 mice were trained to self-administer a 12% ethanol/1% sucrose solution on a fixed-ratio schedule during 30 minute sessions in operant chambers. After establishing a stable baseline level of responding and intake, increasing concentrations of quinine (a bitter tastant) were introduced into the ethanol solution daily. As expected, the gradual increase in the bitterness of the alcohol solution resulted in a decrease in the intake of alcohol as compared to baseline (F(5, 115)=5.4, p<0.001). However, the rate of responses remained stable indicating a habitual pattern of responding for alcohol. In a subsequent test, subjects not previously exposed to quinine were exposed to a 0.5mA foot shock (one second in duration) upon every other delivery of the alcohol reinforcer. In this test, both the number of responses and alcohol intake were significantly reduced as compared to baseline (F(4, 92)=10.5, p<0.001, F(4, 92)=5.4, p<0.001). These results suggest that mice trained to respond for alcohol reward over several weeks will show signs of habitual but not compulsive responding for alcohol. Future studies will evaluate whether this is also evident in mice that have developed alcohol dependence and self-administer alcohol at significantly higher levels.

37. ROSALES RIVAS J, MANNS P, ENRIQUEZ C, LOM B

**BPA and BPS do not affect tyrosine hydroxylase or swimming activity in Xenopus laevis tadpoles**

*Davidson College*

**ABSTRACT:**

Bisphenol-A (BPA) is a synthetic compound commonly used to make plastics. As an endocrine disruptor (specifically an estrogen antagonist), BPA has been linked to health problems including obesity, diabetes, schizophrenia, hyperactivity, cancer, and more. As a result, some manufacturers are making “BPA free” products, which frequently contain the BPA analog bisphenol-S (BPS). BPS’s strong structural similarities to BPA allow it to be an effective plasticizer but not necessarily a healthier alternative. Previous experiments in our lab and others have revealed that developing dopaminergic neurons can be compromised by BPA exposure. This experiment compared the influence of BPA and BPS on dopaminergic neurons of Xenopus laevis tadpoles. Embryos were exposed to 0-300 nM BPA or BPS for 96 hours. Immunostaining for tyrosine hydroxylase (TH), an enzyme critical for dopamine production, allowed visualization of midbrain dopaminergic neuronal clusters in the posterior tuberculum via confocal microscopy. Areas of these TH+ neurons were then analyzed using quantitative morphology. Statistical comparisons showed that neither BPA nor BPS had an effect on the area of TH+ neuronal clusters. These results may be due to an inability to quantify 3D clusters appropriately. We also studied the effects of BPA and BPS on the developing tadpole nervous system by examining swimming behaviors and hyperactivity because BPA has been linked to hyperactivity in children and zebrafish. We hypothesized that BPA and BPS would cause hyperactivity, but observations of swimming behaviors revealed no significant differences from controls.

38. RYAN C, HIDALGO-LOPEZ M, SCHUMMERS J

**Astrocyte morphology shaped by placement within functional maps in ferret visual cortex**

*Florida Atlantic University, Max Planck Florida Institute for Neuroscience*

**ABSTRACT:**

Although once considered minor participants in the realm of cortical processing, astrocytes, the most abundant type of glial cell in the CNS, are now recognized for their significance in shaping the functional capacities of the cortex. Here we injected into the ferret visual cortex three “Spaghetti Monster” fluorescent proteins (smFP’s) each containing multiple copies of distinct epitopes that permitted signal amplification for astrocyte visualization. This allowed investigation of the spatial distribution of astrocytes in various cortical layers, relating the position of an astrocyte within a functional visual map as a potential determinant of morphology. In areas of greater functional heterogeneity where multiple orientation columns converged, called “pinwheel centers,” we sought to determine how the shape and spatial placement of these astrocytes was correlated to their relative position to these pinwheel centers. Specifically, we hope to establish how the astrocyte’s shape and its degree of elongation reflects the functional organization of the visual cortical circuit. The elongation of astrocyte shape near the pinwheel centers is hypothesized to reflect the boundaries between regions of various orientation preferences represented by that portion of the cortex.

39. SAMMONS KM, DEAK LC, CLELAND CL

**Selective Stimulation of A-delta Nociceptors in Rat Hind Limb and the Resulting the Nociceptive Withdrawal Response**

*James Madison University*

**ABSTRACT:**

Rats rapidly withdraw their hind limb in response to heat or other noxious stimulation, which is known as the Nociceptive Withdrawal Response (NWR). Two types of nociceptors may mediate the NWR: C-fibers and Aδ nociceptors. Among the differences between these two types of nociceptors, C-fibers have large receptive fields while Aδ nociceptors have much smaller receptive fields. Previous studies have shown that the direction of the NWR does not depend on stimulus location. However, these experiments used a method of heating that may have predominantly stimulated C-fibers. If C-fibers were stimulated, we might expect no dependence on stimulus location due to their larger receptive field compared to Aδ nociceptors. Therefore, it remains possible that Aδ nociceptors could mediate a response that is dependent upon stimulus location. The specific aim of our ongoing experiments is use preferential stimulation of Aδ nociceptors using high intensity, short duration (100ms) pulses of heat to determine if the NWR depends in stimulus location. The length of the pulse was determined based on previous research showing that Aδ nociceptors are selectively activated with short, high intensity pulses of heat. Because Aδ fibers have small receptive fields, we hypothesize that the selective stimulus will result in a response that is dependent on stimulus location. Five small (1 mm) spots (three aligned rostral-caudal, three aligned lateral-medial) were blackened on the plantar surface of the left hind paw. These spots were stimulated in a randomized sequence and the initial and final positions of the paw were recorded with a camcorder (60 fps @ 1080p) placed underneath the rat. When stimulated, the rat picks up its paw and rapidly places it back down on the glass. The difference between the initial and final positions represents the NWR movement response vector. Unexpectedly, stimulus location still did not have an effect on the direction of the NWR. Rather, preliminary results (n=6) suggest that the direction of the response is determined only by the initial position of the paw, as observed in previous experiments where C-fibers were presumably stimulated. These results further substantiate our findings that the NWR is organized around initial posture rather than the details of the noxious stimulus.

40. SHANKS MK, OPRISAN SA

**Functional near-infrared spectroscopy (fNIRS) while performing temporal discrimination tasks**

*Department of Biology, Department of Physics and Astronomy, Program in Neuroscience, College of Charleston*

**ABSTRACT:**

The capability of perceiving and using the passage of time in the seconds-to-minutes range (interval timing) is essential for survival and adaptation, and its impairment leads to severe cognitive and motor dysfunctions. The striatum and its afferent projections from the substantia nigra are thought to be involved in interval timing. In rats, lesions to the caudate-putamen and substantia nigra impair interval timing. Recent studies suggest that this interval timing network could involve cortico-striatal loops, including areas of the prefrontal cortex, such as the dorsolateral prefrontal cortex. This study will use functional near-infrared spectroscopy (fNIRS) to image the prefrontal cortex of humans during temporal discrimination tasks (distinguish between 8s and 21s intervals). It is hypothesized that there will be activity in the dorsolateral prefrontal cortex in response to the temporal discrimination tasks. Previous studies have shown that normal subjects are able to differentiate between two different time intervals, therefore, we expect subjects in the current study will be able to distinguish between 8s and 21s. This study is expected to validate the findings of previous studies, and to determine whether fNIRS is a reliable method of measuring activity in the prefrontal cortex.

41. SLEDGE RA, MOORE EJ, JOHNSON HL, KAPLAN ZS, SNOUSE SJ, PAVELKA MN, EVERETT SK, FENNELL CT, ZRULL MC

**The IMPULSE journal: a practical teaching tool for a neuroscience minor**

*The Honors College and Department of Psychology, Appalachian State University*

**ABSTRACT:**

While undergraduate journals are a relatively recent phenomenon, their popularity has grown significantly since the founding of IMPULSE in 2003. The Council on Undergraduate Research (CUR) maintains an online database of most undergraduate journals, and currently they have over 200 listed; 51 focus specifically on natural or health sciences. However, the majority of undergraduate journals limit their submissions to students at their journal’s host university, and reviewing is rarely by undergraduates. These university-specific journals serve as a library database for reviewed research across all disciplines for that particular university. IMPULSE, however, is limited to neuroscience work,

but open for submissions from all undergraduates worldwide. An important and unique feature of IMPULSE is that the reviewers are also all undergraduate students. This student-review process is overseen by faculty at Reviewer Training Sites from 14 institutions (open to others), and they provide reviewer and editor training that is centered on an authentic process of submission review. With the increase in undergraduate neuroscience minors, there has been growing interest in integrating the IMPULSE opportunities into the curriculum of those minors, and tailoring its use to meet the research and curricular needs of each school. In addition, IMPULSE's function as both an academic journal and training tool has been shown to benefit students' writing, editing, and leadership skills regardless of discipline. Thus, the model can be extended to other fields to provide the broader experience of reviewing, editing, and publishing articles that goes far beyond merely publishing a student's work.

42. STEADMAN S, BAARINE M, SINGH I

 **(Travel Award Winner)**

**Effects of epigenetic modifications in X- linked adrenoleukodystrophy**

*Department of Biology, Program of Neuroscience, College of Charleston; Department of Pediatrics, Medical University of South Carolina*

**ABSTRACT:**

X- linked adrenoleukodystrophy (X-ALD), a rare genetic disease, is caused by a mutation in the ABCD1 gene affecting a peroxisomal protein called adrenoleukodystrophy protein (ALDP). ALDP is a peroxisomal membrane protein that facilitates the transport of very long chain fatty acids (VLCFA) from the cytoplasm into the peroxisomal lumen for catabolism by peroxisomal beta-oxidation enzyme synthesis. VLCFA are exclusively catabolized in the peroxisomes. Dysfunction of ALDP leads to a buildup of VLCFA in plasma of the brain, adrenal gland, and testis leading to demyelination and neurodegeneration in affected patients. Two main clinical forms of X-ALD known to date are the asymptomatic form, adrenomyeloneuropathy (AMN) and the fatal cerebral form childhood adrenoleukodystrophy (cALD). Patients with the same mutation can present with either of the two phenotypes of X-ALD. To understand the epigenetic mechanisms in AMN and cALD phenotypes, induced pluripotent stem cells (IPSCs) derived oligodendrocytes (OLs) were used to measure protein acetylation and DNA methylation in AMN vs. cALD. To generate IPSC derived phenotype specific OLs (AMN vs. cALD), neural precursor cells (NPCs) were produced from IPSCs and characterized for specific markers via immunostaining. Next, NPCs were differentiated to OLs and characterized for specific markers. Using these OLs, we investigated a phenotype specific dysregulation in histone acetylation vs. DNA methylation. We observed that OLs differentiated from both cALD and AMN IPSCs had decreased histone acetylation. Treatment of cALD cells with sodium acetate corrected the decreased histone acetylation levels in cALD glial cells. Next, we investigated phenotype specific DNA methylation in IPSC-derived OLs. Highest levels of DNA methylation were observed in cALD OLs and these methylation patterns correlated well with expression of DNA methyl transferases (DNMTs). Treatment of cALD cells with a DNA methyl transferase inhibitor decreases the gene expression of DNMTs. These observations suggest pathological roles of decreased histone acetylation and increased DNA methylation in phenotype specific (AMN vs. cALD) metabolic pathology of X-ALD in OLs. These observations may also be applied to other neurodegenerative diseases with similar epigenetic modifications.

43. UNROE KA, FRUCHTERMAN TC, RIPLEY AO, FRANSSEN RA

**Estrogen Receptor Levels Higher in "Bad" Maternal Rats than in "Good" Maternal Rats**

*Department of Biological and Environmental Sciences, Longwood University*

**ABSTRACT:**

Our lab is interested in maternal behavior in rats and the underlying neurological drivers of behavior. Recently, we discovered that most maternal rats make complex decisions regarding whether to care for a litter of pups that contains alien pups. Additionally, we found that some rats, when presented with a litter of pups, would quickly collect them all regardless of what percentage were her own (Good Moms). Others would do the opposite and be slow to retrieve regardless of percentage of own vs. alien pups in the litter (Bad Moms). This year, we sought to discover the neurological differences between Good and Bad maternal rats. We doubled our n-value from last year in an effort to find clear behavioral and neurological signals. Our preliminary findings suggest that, in addition to differential neuron activation in several brain regions, Estrogen Receptor Alpha levels are significantly different between groups. Here, we discuss these preliminary findings and put them in the context of previous results to help address the question of why a rat might behave as a Good or Bad mother.

44. VALIULIS GJ, WEBER RA, ADKINS DL

**Pharmacological Enhancement of Rehabilitation After Ischemic Stroke in Rats**

*Department of Biology and Program in Neuroscience, College of Charleston; Department of Neurosciences and Health Science Research, Medical University of South Carolina*

**ABSTRACT:**

Approximately 795,000 people experience a stroke each year. An estimated third of stroke survivors have persistent motor deficits. Currently, tissue plasminogen activator (tPA) is the only approved treatment to help reduce stroke-induced brain damage. tPA helps to restore blood flow after ischemic stroke, but must be administered within 4hrs of stroke. Thus, many stroke survivors do not benefit from this treatment, creating a need to develop treatments that can protect the brain during and after stroke. Previous studies have shown that mitochondrial dysfunction is a common cause of neuronal death early after stroke. We also reported that after focal, unilateral strokes to the forelimb area of the motor cortex in rats, there is an increase peri-lesional mitochondrial dysfunction up to 6 days after. Further, we demonstrated that formoterol, a β2 adrenergic agonist, restores some of these mitochondrial functions in peri-injury striatum. After focal motor cortical stroke, administration of formoterol combined with motor rehabilitation significantly improved motor performance compared to rehabilitation alone, suggesting that formoterol may reduce long-term motor deficits by improving mitochondrial function. In the current study, we examined whether formoterol combined with rehabilitation would also be effective after a more severe stroke that produces greater tissue loss and more profound motor impairments. Rats were trained in the single pellet reaching task and received a unilateral middle cerebral artery occlusion stroke. Twenty-four hours post-stroke, animals were injected with 0.1mg/kg formoterol (n=7) or saline (n=8), daily for 18 days. On day 3 post-stroke, all animals began daily motor rehabilitation. There was no significant difference between groups over the 15 days of rehabilitation. After severe ischemic damage to the cortex and striatum, formoterol does not enhance the efficacy of rehabilitation. In fact, rehabilitation alone also failed to improve motor function over the 15 days of training following severe stroke, unlike previous studies involving more moderate strokes. Currently, we are using these data to investigate the relationship between stroke size and behavioral deficits and the potential effects of formoterol on mitochondria number and β2 adrenergic receptor expression.

NIGMS IDeA P20 GM109040, MUSC Neuroscience Institute Pilot Grant

45. WANLISS JA, LIU D, BROWN D, WASHINGTON B

**Human Psychomotor Skills Acquisition**

*Presbyterian College*

**ABSTRACT:**

Existing research on human skills acquisition studies has shown that learning follows a nonlinear pattern, but the exact form remains unknown due to the limitation of traditional experimental methods and lack of systematic

modeling of tasks. We applied a nonlinear fractal analysis on the time series data produced by human subjects on target-tracking motor learning tasks. Tracking of a non-fractal sinusoid-cosinusoid signal was used as the platform. Our preliminary results suggest that fractal models may prove effective in investigating details of the human learning process.

46. YING R, PRATT WE

 **(Travel Award Winner)**

**Effect of the CB1 neutral antagonist AM4113 on palatable food motivation**

*Department of Biology, Wake Forest University*

**ABSTRACT:**

CB1 receptors are found throughout the brain in regions important for regulating food intake and motivation, and have been targets for the development of effective appetite suppressing drugs. However, many tested CB1 antagonists, such as rimonabant, have shown inverse agonist properties, and also produce unwanted side effects such as emesis and anxiety. CB1 neutral antagonists may help with weight loss without producing these side effects. In these experiments, the efficacy of one CB1 neutral antagonist, AM4113, was tested on the intake of palatable food and cue-induced reinstatement. For the feeding experiment, eight rats were placed in feeding chambers for two hours per day, and habituated to a high-fat palatable diet for ten days. Water and food intake were measured by weight, and sensors placed around the cage measured locomotion and rearing. Drug doses (vehicle, 1.0 mg/kg, 2.0 mg/kg, 4.0 mg/kg, and 8.0 mg/kg) were randomized and administered intraperitoneally on each of the five drug testing days. Four days were given between each drug test to allow the drug to wash out of the rat’s system. All rats had ad libitum access to laboratory chow outside of the trials. AM4113 dose-dependently reduced palatable food and water intake significantly without affecting locomotor measures. All doses of AM4113 reduced food and water intake at or above the levels observed when other rats were tested with rimonabant. A separate experiment tested the effects of AM4113 on cue-induced reinstatement. Rats were trained to lever press for sugar pellets on a FI20/PR5 schedule. Each sugar pellet delivery was paired with a concurrent light/tone to establish the reward-related cue. Behavior was then extinguished by removing both the cue and the reward, until rats exhibited 10% of their pre-extinction response. During reinstatement, lever presses produced the audiovisual cue associated with reward, but no sugar pellet was received. Rats were intraperitoneally injected with AM4113 (4.0 mg/kg) or saline prior to two reinstatement sessions. AM4113 significantly prevented cue-induced reinstatement of sugar seeking. These results extend earlier studies by demonstrating that the neutral CB1 receptor antagonist AM4113 is effective at reducing food intake motivated by palatability and that it effectively blocked cue-induced reinstatement, suggesting that it may reduce the incentive properties of cues associated with palatable food.