19th Annual
Student Research Symposium

Celebrating 35 years
1978-2013

Thursday, April 25, 2013

Stiteler Hall
208 South 37th Street

SCHOOL OF ARTS AND SCIENCES
UNIVERSITY OF PENNSYLVANIA
Biological Basis of Behavior Program

invites you to a

Graduation Reception and Awards Program

Honoring the Class of 2013

Saturday, May 11, 2013
2-4 p.m

University of Pennsylvania
Penn Museum
Chinese Rotunda
3260 South Street, Philadelphia, PA

If you haven't done so yet, please
RSVP by April 6th
E-mail: mholton@sas.upenn.edu
Phone: 215-573-3163
Welcome

On behalf of the Biological Basis of Behavior Program, we welcome you to this very special day, our 19th Annual Student Research Symposium. It has been the goal of the program from its inception 35 years ago, for BBB majors to benefit from our faculty's commitment to multidisciplinary teaching and research. Such a commitment is clearly evident in the research accomplishments of our majors showcased today. Whether you spend the entire day with us or just a portion of it, we are sure you will be impressed.

Dr. Ted Abel, Director
Schedule of Activities

8:30 a.m.  Continental Breakfast

9:15 - 11:15 a.m.  Honor Thesis Presentations  
                 Stiteler Hall B21 & B26

11:30 - 12:30 p.m.  Poster Session and Lunch  
                  Stiteler Hall Forum

12:45 - 3 p.m.  Honor Thesis Presentations  
                Stiteler Hall B21 & B26
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Stitler Hall, Room B21,

9:15
**Katsiaryna Malykhina**
Title: “Comparing Spatial Language and Gesture Production in Younger Controls, Older Controls, and Stroke Patients”
**Supervisor:** Dr. Anjan Chatterjee, Center for Cognitive Neuroscience

9:30
**Geena Ianni**
Title: Metaphor Comprehension in Brain-Injured Patients
**Supervisor:** Ianni, G., Cardillo, E., McQuire, M., Chatterjee, A.

9:45
**Amanda Bader**
Title: The effects of Transcranial Direct Current Stimulation (tDCS) on language recovery in chronic stroke patients with Aphasia
**Supervisor:** Dr. Roy Hamilton

10:00
**Jeffrey Desilets**
Title: Relationships between Maternal Diet & Inattention
**Supervisor:** Teresa Reyes, Ph.D. Research; Mentor: Nicola Grissom, Ph.D.

10:15 – 10:30
- BREAK-

10:30
**Christina Johns**
Neurocognitive performance measures as predictors of psychosis risk in children and adolescents.
**Supervisor:** Monica Calkins, Ph.D, Department of Neuropsychiatry

10:45
**Mark Barry**
Title: Effects of Social Defeat on Cognitive Flexibility and CRF-modulated Systems in Female Rats
**Supervisors:** Dr. Rita J. Valentino; Kevin Snyder

11:00
**Madeleine Byrd**
Title: Neuronal lipid regulation in long-term potentiation and neuropsychiatric disorders
**Supervisor:** Sangwon F. Kim, Department of Psychiatry

11:15
**Daniel Ramos**
Title: Understanding the developmental mechanisms of mecahnoreceptive neurons
**Supervisor:** Dr. Wenxin Luo

11:30 – 12:30
- POSTER SESSION & LUNCH-
12:45

Julia Katz
Title: Effect of glutamatergic modulation on sociability in juvenile protocadherin-10 gene haploinsufficient mice, a genetic mouse model of autism
Supervisor: Dr. Edward S. Brodkin, Assistant Professor of Psychiatry

1:00

Rachel Gates
Title: Liquid control may not determine performance in visuo-motor tasks in Rhinus Macaques
Supervisor: Joshua Gold, Department of Neuroscience

1:15

Giulia Porcari
Title: Effect of atypicality on coordination in behavioral variant frontotemporal dementia
Supervisor: Dr. Murray Grossman, Cognitive Neurology, Perelman School of Medicine

1:30

Stephanie Golob
Title: Impaired Coordination and Theory of Mind in a Coordination Game in Patients With bvFTD
Supervisor: Dr. Murray Grossman, Cognitive Neurology, Perelman School of Medicine

1:45 – 2:00

- BREAK-

2:00

Jennifer Gilbert
Title: Endogenous Leptin in the Nucleus Tractus Solitarius Reduces Motivation of Reward Seeking Behavior
Supervisor: Harvey Grill, Psychology

2:15

Yael Burla
Title: Behavioral and Eye-tracking Studies on how Consumer Decision-making Strategies Change with Increasing Options
Supervisor: Joseph W. Kable, Psychology

2:30

Anand Tekriwal
Title: Modulation of dopaminergic circuitry using deep brain stimulation
Supervisor: Dr. Tracy Bale.

2:45

Chelsea Aleo
Title: Acute EEG response predicts restoration of cognitive function following constitutive NMDAR-hypofunction
Supervisor: Steven J. Siegel

3:00

Kyle Henson
Title: Exploring the Role of mGluR5 and TrkB in Sleep Homeostasis
Supervisor: Dr. Marcos Frank; Neuroscience Department
Abstracts - Room B21

Katsiya Malychina
Title: “Comparing Spatial Language and Gesture Production in Younger Controls, Older Controls, and Stroke Patients”
Supervisor: Dr. Anjan Chatterjee, Center for Cognitive Neuroscience
Spatial language is used to convey information regarding relationships between objects and their environments, and is often accompanied by gesture. The connection between spatial speech and gesture, as well as the neural mechanisms behind the process, are not well understood. This study compares spontaneous speech and gesture production in book reading and map directing tasks in young people, elderly people, and patients with focal brain injuries. The purpose of this study is to understand the relationship between spatial speech and gesture, with the hypothesis that increased gesture can aid communication of spatial information, especially for patients with brain lesions. The preliminary results show an increased number of verbs used in prepositional phrases used by older controls than younger controls. In our current analyses we examine language differences and gesture disparities between the three groups and how self-awareness of gesture use influences their gesture production.

Geeta Ianni
Title: Metaphor Comprehension in Brain-Injured Patients
Supervisor: Ianni, G., Cardillo, E., McQuire, M., Chatterjee, A.
Despite the prevalence of metaphor in everyday language, the neural basis of this powerful communication device remains poorly understood. The purpose of the current study was to test the hypothesis that traditional aphasia assessments are not sensitive to deficits in metaphor comprehension. Stimuli consisted of moderately novel metaphors of three different types: predicate metaphors based on action verbs (The divorce was a scarf woven through the paperwork), nominal metaphors based on event nouns (The divorce was a hard fall), and nominal metaphors based on entity nouns (The coffee was a caffeine bullet). For each metaphor (n = 60), a closely matched literal sentence with the same base word was also generated. Sentences were presented visually in a randomized order, followed by four adjective-noun answer choices (target + three foil types). Participants were instructed to select the phrase that best matched the meaning of the sentence. Results with healthy adults indicated comparable accuracy across conditions. By contrast, focal lesion patients showed different comprehension profiles, including good comprehension on some conditions, a pervasive metaphor comprehension impairment, and selective impairment with particular types of metaphor. Six of the seventeen patients tested demonstrated a metaphor deficit of some kind. We conclude our task captures deficits and subjective difficulties in metaphor comprehension not captured by traditional language assessments and, as such, allows for critical testing of neural hypotheses about metaphor.

Amanda Bader
Title: The effects of Transcranial Direct Current Stimulation (tDCS) on language recovery in chronic stroke patients with Aphasia
Supervisor: Dr. Roy Hamilton
Aphasia, the loss or impairment of language ability, is one of the most common consequences of a unilateral left hemisphere stroke. Previous research suggests that noninvasive brain stimulation techniques specifically transcranial direct current stimulation (tDCS), may help to facilitate neuroplastic changes in chronic stroke patients with nonfluent aphasia in order to recover language ability. One ongoing research study administers individually-tailored tDCS by identifying the brain region that shows best transient response to tDCS in measures of language function. This is accomplished through the use of varying electrode montages in order to determine the most responsive cortical area. My study extends this research to see if tDCS, applied without identification of an individualized target area, will still result in statistically significant improvements in language ability, as measured by a battery of standardized language tests. Based on the results from previous studies, subjects are likely to exhibit improvements in fluency after undergoing tDCS at a previously determined, not subject specific, site.

Jeffrey Desilvia
Title: Relationships between Maternal Diet & Inattention
Supervisor: Teresa Reyes, Ph.D. Research Mentor: Nicola Grissom, Ph.D.
Human children of mothers with certain adverse health conditions tend to maintain gestational sizes that are either larger or smaller than average. These offspring tend to develop attention deficits/hyperactivity disorder later in life. This experiment used a mouse model to determine the relationship between gestational size, gene expression, and attention in the offspring. Individuals that were small for gestational age exhibited more abnormal attentional focus and less restlessness in the 5-choice serial reaction time test (5C-SRT) of attention. By contrast, individuals that were large for gestational age exhibited less motivation and executed more premature responses in the 5C-SRT attention test. Both of these deficits resulted in less correct responses in the 5C-SRT test, but their neurochemical causes may be different.

Christina Johns
Neurocognitive performance measures as predictors of psychosis risk in children and adolescents.
Supervisor: Monica Galkins, Ph.D., Department of Neuropsychiatry
Risk for schizophrenia is characterized by both sub- psychotic symptoms and deficits in neurocognition. In order to establish a more detailed risk profile for psychosis, I evaluated predictive power of working memory, verbal memory, and attentional capacity as measured using a neurocognitive battery. Participants were drawn from a large cohort of 9,200 young people aged 11-21, among whom 164 returned for follow-up assessment. While neurocognitive performance at Time 1 was not significantly predictive of risk designation at the follow-up approximately two years later, there was a small but significant deficit in all three cognitive domains for higher psychosis risk participants. It is possible that our sample size at follow-up was not large enough to detect such a small difference in performance. With repeated analysis, it is hoped that a more reliable screening method for psychosis risk including both sub-psychotic and cognitive symptoms can be developed.

Mark Barry
Title: Effects of Social Defeat on Cognitive Flexibility and CRF-modulated Systems in Female Rats
Supervisors: Dr. Rita J. Valentino, Kevin Snyder
Corticotropin releasing factor, a stress-related peptide that acts as a neurotransmitter to affect autonomic and behavioral components of stress, has been shown to modulate activity in the dorsal raphe and locus coeruleus, nuclei that project to forebrain regions involved in cognitive flexibility. To study the effects of chronic social stress on cognitive flexibility and these brain regions in female rats at different developmental stages, a social defeat procedure is used here. Performance on reversal learning and strategy set shifting has been assessed using an operant procedure, and immunohistochemistry is being used to assay c-fos expression as an indirect measure of neuronal activity in the prefrontal cortex and dorsal raphe. Early adolescent (EA) rats with long latencies to defeat performed worse on strategy shifting tasks than both FA controls and EA defeat rats with short latency. Regional analysis of c-fos expression will help to identify neural correlates of
behavioral performance.

**Madeleine Elizabeth Byrd**

Title: Neuronal lipid regulation in long-term potentiation and neuropsychiatric disorders

**Supervisor:** Sangwon F. Kim, Department of Psychiatry

Long-term potentiation (LTP) forms the cellular basis of learning and memory. LTP involves activity-dependent persistent increase in synaptic transmission. N-methyl-D-aspartate receptor (NMDAR) trafficking plays an essential role in regulating synaptic efficacy and remodeling, which, when dysregulated, can contribute to a number of neuropsychiatric disorders including schizophrenia. NMDA channel activation has also been found to result in rapid transcription of various genes called immediate early genes, such as Activity-Regulated cytoskeletal-associated protein (Arc). My laboratory previously showed that SREBP, the master transcription factor regulating lipid synthesis, activation is required for Arc activation, both of which are impaired in the animal model of schizophrenia. Here, we report that TFA, an NMDA-dependent chemical inducer of LTP, fails to activate both SREBP and Arc transcription in the mouse model of schizophrenia. This study implicates abnormal lipid synthesis in the pathogenesis of schizophrenia, which may provide a potential target for the development of therapeutics treating this neuropsychiatric disorder.

**Daniel Ramos**

Title: Understanding the developmental mechanisms of mecamlocreptive neurons

**Supervisor:** Dr. Wenqin Luo.

The study of nerve outgrowth has implications in understanding not only the basic biology behind development, but also in better treatment opportunities for disease. The goal of this study is to determine the molecular mediators of proper mechanocrepetitive development. Using immunohistochemical and cell culture techniques we investigate the signaling pathways that occur both peripherally and centrally that are required for proper mechanocrepetive development. Peripherally, we find that a transcription factor called FFR1 is required for Pacinian corpuscles to develop. We continue our research to investigate the cues that mediate the third order central projections of rapidly adapting mechanoreceptors that innervate layers III and IV of the dorsal spinal cord. We are actively looking into the Ret receptors GFRα1 and GFRα2 to elucidate their roles in this developmental process.

**Julia Katz**

Title: Effect of glutamatergic modulation on sociability in juvenile protocadherin-10 gene haploinsufficient mice, a genetic mouse model of autism

**Supervisor:** Dr. Edward S. Brodkin, Assistant Professor of Psychiatry

Various cadherin and protocadherin genes, including protocadherin 10 (PCDH10), have been implicated in the etiology of autism spectrum disorders (ASD). Studies in the mouse have demonstrated that Pcdh10 is involved in prenatal brain development (axon outgrowth), is expressed in glutamatergic synapses postnatally and binds to PSD-95. The Brodkin lab has found reduced sociability in Pcdh10 haploinsufficient (Pcdh10<sup>-/-</sup>) male mice. Given the expression of Pcdh10 in glutamatergic synapses, we hypothesized that the social behavior deficits in Pcdh10<sup>-/-</sup> mice could be rescued by modulation of glutamatergic neurotransmission. D-cycloserine (DCS), a partial agonist at the glycine-binding site of N-methyl-D-aspartate receptors (NMDARs) was used for the modulation of the glutamatergic neurotransmission. A significant increase in sociability was found in Pcdh10<sup>-/-</sup> mice that were treated acutely with 32 mg/kg DCS intraperitoneally. Future studies will determine whether glutamatergic neurotransmission is altered in specific circuits in Pcdh10<sup>-/-</sup> mice.

**Rachel Gates**

Title: Liquid control may not determine performance in visuomotor tasks in Rhesus Macaques

**Supervisor:** Joshua Gold, Department of Neuroscience

Our growing understanding of the brain has been based, in part, on studies that involve neurophysiological recordings in awake, behaving monkeys. These studies typically use standard operant techniques, in which liquid is given as a positive reward for desired behaviors. In these cases, nearly all monkeys are kept at a carefully controlled liquid intake schedule to ensure adequate motivation. These schedules, which mirror conditions that monkeys sometimes encounter in the wild (Desimone et al., 1992), can be both effective and applied humanly within a laboratory setting (Newsome and Stein, 1999). Nevertheless, minimizing water control, as long as the scientific integrity of the study is not compromised, is always laudable goal. This study endeavors to show that water control should not be taken as the sole factor driving motivated behavior in these experiments, but rather one of numerous factors that should be considered. By using data collected over the past seven years on eight separate monkeys, we document that the daily rate of water intake can vary across a broad range while the monkeys are still performing effectively. In fact, even monkeys that have seen a relatively abrupt increase in daily liquid intake have maintained or in some cases improved performance over previous sessions when under a more strict liquid-control regime. These results suggest that identifying other factors like task design, husbandry techniques, and monkey-human interactions that also can affect motivated behavior might allow for an easing of water control and thus help promote the health and well being of on-study monkeys.

**Giulia Porcari**

Title: Effect of atypicality in coordination in behavioral variant frontotemporal dementia

**Supervisor:** Dr. Murray Grossman, Cognitive Neurology, Perelman School of Medicine

Social interactions requiring that individuals reach the same conclusion without explicit communication are formalized in game theory as “coordination”, and rely on the establishment of a focal point which transcends the structure of the game. A recent study indicated that patients with behavioral variant frontotemporal dementia (bvFTD) have a decreased ability to perform coordination tasks. To confirm that this is due to coordination specific deficits we designed an alternative task in which an “oddball” response could serve as a focal point. Unexpectedly, controls (N=11) did not coordinate on oddballs; however, bvFTD patients (N=15) did show strikingly different behavior, selecting oddballs significantly more on baseline, but significantly decreasing oddball selection in coordination, mirroring control levels. Furthermore, regression analysis on MRI images related oddball selection in coordination with atrophy in the ventromedial PFC. Further investigation is necessary, but these results may suggest a possible dissociation between executive and social
Stephanie Golob
Title: Impaired Coordination and Theory of Mind in a Coordination Game in Patients With bvFTD
Supervisor: Dr. Murray Grossman, Cognitive Neurology, Perelman School of Medicine
Behavioral- Variant Frontotemporal Dementia (bvFTD) is a neurodegenerative disease that has been associated with progressive frontal and anterior atrophy, resulting in inappropriate social behavior and difficulty with executive decision-making. bvFTD patients typically exhibit limitations in social coordination and theory of mind. Coordination is the process by which individuals reach the same conclusion with minimal communication. In this study, coordination and theory of mind were quantified in the form of a Coordination Game. The goals of this study were to test theory of mind deficits in bvFTD, use coordination to quantify social decision-making deficits in bvFTD, and identify the neuroanatomic locus of this deficit. It was found that bvFTD subjects performed significantly worse than control subjects in the coordination condition but not the control condition due to impaired coordination and theory of mind. This poor performance was correlated with atrophy in the right inferior frontal cortex, which is responsible for executive functions.

Jennifer Gilbert
Title: Endogenous Leptin in the Nucleus Tractus Solitarius Reduces Motivation of Reward Seeking Behavior
Supervisor: Harvey Grill, Psychology
Signaling of the adipose tissue derived hormone leptin in the nucleus tractus solitarius (NTS) of the hindbrain has been implicated in food intake driven by caloric deficit (“need-based feeding”). The current study examines whether NTS leptin signaling is also involved in food reward seeking behavior, and if behavior differs with caloric status. Rats maintained on ad libitum or food deprived diets were trained in conditioned place preference (CPP) or progressive ratio reinforcement (PR) paradigm. We found leptin (0.3 µg) administration into the NTS significantly reduced the motivation to eat under PR conditions, independent of energy status. Leptin administration also significantly reduced food-seeking in the CPP paradigm. This effect was food specific, as there was no effect on morphine in either paradigm, and dependent on energy status. These findings suggest a novel role for NTS leptin signaling in the control of food reward seeking behavior.

Yael Buda
Title: Behavioral and Eye-tracking Studies on how Consumer Decision-making Strategies Change with Increasing Options
Supervisor: Joseph W. Kable, Psychology
On a daily basis, consumers are presented with options from which they must make a decision. Previous research has shown not only that people face a choice paralysis or behave sub-optimally when they are presented with too many options, but also that they use a simplifying heuristic to compensate for the difficult decision-making task. Research in our lab specifically suggests people use an adaptive reduction strategy when selecting a gamble from an increasing number of options. This study was adapted and externally validated to assess the strategies used to make decisions on consumer goods as the number of choice options increases. Due to the increased option complexity, such as number of attributes and brand loyalty, the results determine whether the same change in strategy is elicited as when choosing from a gamble.

Anand Tekriwal
Title: Modulation of dopaminergic circuitry using deep brain stimulation
Supervisor: Dr. Tracy Bale
Schizophrenia is an undiagnosed neuropsychiatric disorder affecting 24 million people worldwide. Although the majority of schizophrenics benefit from conventional treatments, up to 30% of patients are refractory to care. Deep brain stimulation (DBS) has advanced clinicians’ ability to address several treatment-resistant neuropsychiatric disorders and may prove efficacious for schizophrenia. We hypothesize that modulation of the prefrontal cortex using DBS may attenuate behavior associated with schizophrenia. Mice were implanted in the prefrontal cortex with bipolar electrodes and stimulated. Pre-pulse inhibition (PPI) testing was subsequently carried out, as it is known that schizophrenics have a deficit in sensorimotor gating and accordingly a decreased PPI. In the stimulated group an increase in PPI was found but the change was statistically insignificant. These results may be due to the temporally acute effects of DBS or unavoidable confounding variables resulting from presence of an electrode. The effect of prefrontal cortex stimulation is currently being further evaluated using a hypothalamic-parietal-adrenal axis response to stress.

Chelsea Aleo
Acute EEG response predicts restoration of cognitive function following constitutive NMDAR hypofunction
Supervisor: Steven J. Siegel
Previous research has shown that electrophysiological deficits, in particular gamma (30-80Hz) oscillatory abnormalities, are present in medicated schizophrenia patients who display treatment-resistant cognitive and social deficits. Therefore, we hypothesized that gamma band activity may act as a biomarker for therapeutic development to target the cognitive and social deficits related to schizophrenia. As previous research has shown NMDA hypofunction contributes to cognitive, social, and gamma band abnormalities associated with schizophrenia, this study used pyramidal cell selective NMDA-R1 (NR1) knockout mice. We examined the efficacy of two AMPA/kainate antagonists, ZK200775 and NBQX, on restoring normal electrophysiological and cognitive activity through event-related EEGs and the continuous T-maze paradigm, respectively. We then determined whether normalization of gamma band activity predicted restoration of cognitive function. Preliminary results have shown that though neither ZK200775 nor NBQX significantly restored normal electrophysiological or cognitive function, there is a correlation between gamma band activity and cognitive ability in the NR1 mice.

Kyle Heuson
Title: Exploring the Role of mGluR5 and TrkB in Sleep Homeostasis
Supervisor: Dr. Marcos Franks, Neuroscience Department
With an increasing appreciation for the role of astrocytes in sleep homeostasis, research efforts have begun to focus upon the molecular and cellular mechanisms by which these supporting cells are able to sense and relay sleep pressure signals. An experimental focus of these efforts is the elucidation of the potential role of astrocytic receptors in sensing extracellular chemical concentrations altered by neurons emitting sleep pressure signals. This study explores two such receptors: the metabotropic glutamate receptor mGluR5 and the neurotrophin kinase T1 TrkB. The mGluR5
study failed to demonstrate a sleep phenotype in an inducible knockdown model using a sleep deprivation paradigm. Imaging results, however, were inconclusive in verifying the knockdown's success. Behavioral results for the T1 TrkB knockout study presented promising indications of increased sleep pressure in experimental subjects. These results will lead to the discontinuation of the mGluR5 study and continued exploration of the role of T1 TrkB.
9:15
Jonathan Ferrari
Title: The time course of estrogen-mediated spinogenesis in the VMH in female rats
Supervisor: Dr. Hanagan-Carto

9:30
Kei Okochi
Title: Effects of Aberrant mTOR Signaling on Cell Migration in TSC and PMSF
Supervisor: Dr. Peter Crino, Department of Neurology, Shriners Hospitals Pediatric Research Center

9:45
Benjamin Freedman
Title: cAMP-Dependent Protein Kinase Signaling in Sensory Neurons Inhibits Sleep in Caenorhabditis elegans
Supervisor: David Raizen, Neurology Department

10:00
Allison Letica
Title: Behavioral and neuronal responses to affiliative social behavior in mice
Supervisor: Dr. Sheryl Beck, Department of Critical Care and Anesthesiology

10:15 – 10:30 - BREAK-

10:30
Nikolai Zapertov
Title: Tracking sleep-dependent protein translation throughout sleep and wakefulness in the mouse brain.
Supervisor: Dr. Marcos G. Frank, Associate Professor of Neuroscience, Neuroscience Department

10:45
Katherine Liu
Title: Cognitive and Neuroplastic Effects of Chronic Morphine and Morphine Withdrawal in C57Bl/6J mice
Supervisor: Dr. Irwin Lucki

11:00
Sheila Shanmugan
Title: Psychostimulant Effects on PFC Activation and Function in Menopausal Women
Supervisor: Dr. C. Neill Epperson, Penn Center for Women's Behavioral Wellness

11:15
Leah Zuroff
Title: The Effects of DHEA-S and Cortisol on Cognition in a Normal Elderly Population
Supervisor: Dr. Steven E. Arnold, Department of Psychiatry

11:30-12:30 -POSTER SESSION & LUNCH-
12:45
Laura Shook
Title: Sleep Fragmentation and the Unfolded Protein Response in Drosophila Melanogaster
Supervisor: Dr. Nrinjini Naidoo

1:00
Daniel Greenberg
Title: Center for Sleep and Circadian NeurobiologyTranslational Research Laboratories
Supervisor: Dr. Sigrid C. Veasey

1:15
Melissa Riegel
Title: Set-shifting deficits and increased estradiol and gonadotropin levels in female adolescents with anorexia nervosa and low-weight eating disorder not otherwise specified
Supervisor: Dr. Rebecka Peebles, Department of Pediatrics

1:30
Bridget Eilsworth
Title: Epigenetic Regulation of Homer1
Supervisor: Ted Abel, Biology

1:45 – 2:00 - BREAK-

2:00
Ashley Baldwin
Title: Studying Social Reward Pathways Using Multi Variate Pattern Analysis
Supervisor: Dr. Robert Schultz

2:15
Lauren Ambler
Title: Pharmacological modulation of negative endophenotypes of schizophrenia in Brattleboro rats
Supervisor: Dr. Steven Siegel, Psychiatry Department

2:30
Deborah Silber
Title: Examining Neurocognitive Deficits in Children and Young Adults with Chronic Kidney Disease Using the Computerized Neurocognitive Battery
Supervisor: Ruben Gur, PhD
Research Mentor: Tyler Moore, PhD MSc

2:45
Drew Goldberg
Title: An Assessment of Cognitive Performance in Adolescents with Chronic Kidney Disease
Supervisor: Dr. Azuri Collier, Dr. Ruben Gur, Neuropsychiatry Department (Hospital of the University of Pennsylvania)
Jonathan Ferrari
Title: The time course of estrogen-mediated spinogenesis in the VMH in female rats
Supervisor: Dr. Flamangan-Caro

Ovarian hormones act in the ventromedial nucleus of the hypothalamus (VMH) to facilitate female mating behavior in various species, including laboratory rats. Previous work revealed that estrogen action in the VMH alters the organization of dendrites there. In specific, estradiol treatment shortens long primary dendrites and increases the number of spines on short primary dendrites. The mechanisms involved in such dendrite remodeling have not been specified. The present study investigated the time course of estrogen-mediated dendrite changes in the VMH to shed light on the mechanisms of estrogen action. Ovariectomized female rats were treated with vehicle or estradiol and killed 4 or 76 hours later. VMH neurons were visualized with Golgi impregnation. In preliminary analysis, estradiol appears to increase the number of spines on the short primary dendrites by 76, but not 4, hours after treatment. These early results suggest that cytoskeletal changes necessary for estrogen-induced spines in the VMH require more than 4 hours.

Kei Okochi
Title: Effects of Aberrant mTOR Signaling on Cell Migration in TSC and PMSE
Supervisor: Dr. Peter Crino, Department of Neurology, Shriners Hospitals Pediatric Research Center

Tuberous sclerosis complex (TSC) and polyhydramnios, megalencephaly, symmetrical epilepsy and seizures syndrome (PMSE) are neurodevelopmental disorders associated with cerebral cortical malformations and intractable epilepsy. Both TSC and PMSE have been linked to aberrant hyperactivation of the mammalian target of rapamycin (mTOR) pathway. We hypothesized that loss of Tsc1, Tsc2, or STRADA would alter neural migration in an mTOR-dependent mechanism. In this study, we define the effects of loss of function of Tsc1 and Tsc2 (TSC) and STE20-related kinase alpha (STRADA) on migration. mTOR is a direct regulator of mTORC1, which in turn regulates the activity of mTORC1, beta or STRADA. mTORC1 targets, including mTOR, mSIP, and mOS, are involved in migration. We also examined the specific function of mTORC1 in migrating neurons, as well as the role of mTOR in regulating migration.

Benjamin L Freedman
Title: cAMP-Dependent Protein Kinase Signaling in Sensory Neurons Inhibits Sleep in Carnehorbatdus elegans
Supervisor: David Raizen, Neurology Department

During larval stage "lethargus," Carnehorbatdus elegans sleep is regulated by molecular signaling pertinent to mammalian sleep—like cAMP-dependent protein kinase (PKA), which promotes wakefulness. We observed hyperactivity and hyper-responsiveness (2-fold/wild-type) during lethargus in-lite-2. C. elegans, which have constitutive PKA activity because they lack the PKA regulatory subunit (KIN-2). To test the hypothesis that PKA activity in specific neurons promotes wakefulness, we made transgenic lines of lite-2:2/nematodes expressing KIN-2 with distinct, cell type-specific promoters. By quantifying response latency to blue light and the extent of locomotion during lethargus, we found that lite-2:2/nematodes were rendered even less responsive (p<0.01) and less mobile (p<0.01) than wild-type nematodes by pan-neuronal (Pesp-119-driven) KIN-2 rescue. However, (Plac-4-driven) KIN-2 rescue in TAC-4 expressing sensory neurons normalized lite-2:2/nematode lethargus responsiveness (p<0.01) to wild-type levels. We conclude that PKA may exert its wake-promoting effects in several neuron types, but physiologically, it seems, in TAC-4-expressing sensory neurons.

Allison Liberta
Title: Behavioral and neuronal responses to affiliative social behavior in mice
Supervisor: Dr. Sheryl Beck, Department of Critical Care and Anesthesiology

Affiliative social bonding experiences, such as sexual relationships, are thought to reduce anxiety in animals. One possible factor in the mechanism of this response is arginine vasopressin, which is implicated in various social behaviors and found in brain regions thought to be important for social behavior. Vasopressin neurons in male mice show neural activation as indicated by c-Fos expression after mating and project to the dorsal raphe, which makes and releases serotonin, a neurotransmitter critical to anxiety-related behavior. We hypothesized that a behavior that activates vasopressin neurons would decrease anxiety. To test this hypothesis, we monitored the activity of mice on an elevated plus maze, expecting to find an increase in exploratory behavior following affiliative interactions. Preliminary results suggest this is the case. We also examined Fos expression in the principle bed nucleus of the stria terminals and the lateral septum and found that affiliation increased Fos expression in both regions. These results suggest that affiliative behavior both may reduce anxiety and selectively activate neurons in brain regions implicated in social behavior.

Nikolai Zapertov
Title: Tracking sleep-dependent protein translation throughout sleep and wakefulness in the mouse brain
Supervisor: Dr. Marcus G. Frank, Associate Professor of Neuroscience, Neuroscience Department

The role of sleep in relation to protein translation mechanisms is currently not well understood. Previous studies identified changes in protein translation following sleep in the cat cortex and in the mouse hippocampus. Our pilot study utilizes the mouse model to further elaborate on the previous studies by expanding upon the time-course and regional specificity of those experiments. Our research paradigm involved sacrificing mice at specific circadian time-points (0, 1, 2, 3 hours after sleep-initiation) following 5 hours of sleep deprivation. The results revealed that protein translation was upregulated in the cortex and downregulated in the hippocampus, with regional specificity that varied across the sleep cycle.

Katherine Liu
Title: Cognitive and Neuroplastic Effects of Chronic Morphine and Morphine Withdrawal in (C57Bl/6) mice

response latency to blue light and the extent of locomotion during lethargus, we found that lite-2:2/nematodes were rendered even less responsive (p<0.01) and less mobile (p<0.01) than wild-type nematodes by pan-neuronal (Pesp-119-driven) KIN-2 rescue. However, (Plac-4-driven) KIN-2 rescue in TAC-4 expressing sensory neurons normalized lite-2:2/nematode lethargus responsiveness (p<0.01) to wild-type levels. We conclude that PKA may exert its wake-promoting effects in several neuron types, but physiologically, it seems, in TAC-4-expressing sensory neurons.
**Supervisor:** Dr. Irwin Lucki

Opioid administration has been linked to cognitive deficits such as impairments in learning and memory. Recent research suggests that such impairments may be related to a decrease in adult hippocampal neurogenesis. This study evaluated the effects of chronic morphine administration on cognitive performance and hippocampal neurogenesis in C57Bl/6J mice. We used an osmotic minipump approach in order to assess the effects of chronic morphine treatment as well as morphine withdrawal in hippocampal-dependent behaviors and hippocampal cell proliferation. We found that chronic morphine treatment produced a significant decrease in hippocampal-related behavior as well as a decrease in hippocampal cell proliferation, suggesting a potential mechanism behind the cognitive deficits produced by chronic morphine use. Conversely, we found that morphine withdrawal produced a significant increase in hippocampal-related behavior, as well as depressive-like behavior. The decrease in hippocampal cell proliferation seen during treatment persisted during withdrawal, demonstrating lasting effects of chronic morphine use on neuroplasticity. This study and future studies that further elucidate the effects of chronic morphine administration and morphine withdrawal on hippocampal-dependent behaviors and hippocampal neurogenesis will shed light on the effects of chronic morphine use in the treatment of patients with chronic pain.

**Sheila Shammugan**

**Title:** Psychostimulant Effects on PFC Activation and Function in Menopausal Women

**Supervisor:** Dr. C. Neil Epperson, Penn Center for Women's Behavioral Wellness

Many women report experiencing a cognitive decline concurrent with the menopause transition, specifically in areas involving executive functions. One mechanism leading to this decline in executive functions at menopause may be the loss of estrogen modulation of prefrontal dopamine systems. Vyvanse (lisdexamfetamine; LDX) may offset the effect of estrogen loss by enhancing dopamine function in the prefrontal cortex. This double-blind, placebo controlled, crossover study investigated the efficacy of LDX in improving cognitive symptoms, as determined by self-report and performance, as determined by neuropsychological tests, in healthy menopausal women who have experienced a midlife onset of executive functioning difficulties. Results suggest LDX may improve subjective symptoms of executive dysfunction in these women. Preliminary results from a neuroimaging study utilizing fMRI and fMRI suggest LDX decreases glutamate concentrations and increases BOLD signal in the DLPFC of healthy menopausal women with subjective executive difficulties.

**Leah Zuroff**

**Title:** The Effects of DHEA-S and Cortisol on Cognition in a Normal Elderly Population

**Supervisor:** Dr. Steven E. Arnold, Department of Psychiatry

The Effects of DHEA-S and Cortisol on Cognition in a Normal Elderly Population

Elevated levels of glucocorticoids as a result of a dysregulated stress response are known to negatively impact cognition. Similar effects are also seen in the normal aging process. Recent research has pointed to dehydroepiandrosterone (DHEA) and its sulfated form (DHEA-S) as a potential protective element against the effects of glucocorticoids. This study aims to elucidate the relationship between cognition, particularly cognitive decline, and the hormones cortisol and DHEA-S. We hypothesize that these two hormones have an antagonistic relationship, with cortisol promoting decreased cognitive functioning and DHEA-S providing resilience against these effects. The study cohort consists of 131 community-dwelling, cognitively normal elderly individuals who were followed longitudinally for three years. Participants were assessed on various measures including cognitive performance, levels of stress and neuropsychiatric symptoms, and personality traits. Hormone levels were measured in hair, saliva, and plasma. These findings could provide a better understanding of the mechanisms underlying stress and normal aging.

**Lauren Shook**

**Title:** Sleep Fragmentation and the Unfolded Protein Response in Drosophila Melanogaster

**Supervisor:** Dr. Nirmalini Naidoo

The endoplasmic reticulum (ER) is the site of secretory and integral protein folding and post-translational modification. Perturbation of ER homeostasis leads to protein misfolding, aggregation and accumulation. In response, the ER activates a quality control mechanism called the Unfolded Protein Response (UPR). The molecular chaperone Bip is the master regulator of the adaptive UPR and targets misfolded proteins for degradation or refolding. We hypothesize that sleep fragmentation will perturb ER homeostasis, induce the UPR and lead to an upregulation of Bip. We also hypothesize the reverse association: that the UPR will induce sleep fragmentation. To examine this, we mechanically fragmented sleep in Drosophila and used Western Blotting to determine Bip expression. To a second sample, we administered the drug Tunicamycin, a UPR inducer, and recorded the resulting sleep/wake behavior. Our results indicate that the UPR induces sleep fragmentation. Bip expression following sleep fragmentation is currently still being assessed.

**Daniel R. Greenberg**

**Title:** Center for Sleep and Circadian Neurobiology/Translational Research Laboratories

**Supervisor:** Dr. Sigrid C. Vassey

Adverse Behavioral and Neurological Effects of Long-Term Sleep Fragmentation

Sleep apnea causes chronic disruption of sleep and intermittent periods of hypoxia. Although the short-term cognitive effects are relatively well understood, the sustained neurological consequences are less defined. The aim of this study was to determine the behavioral and biological effects of long-term sleep fragmentation on dopaminergic neurons of the substantia nigra. To simulate the effects of sleep apnea, mice underwent a 14-week sleep fragmentation protocol. Rotated performance testing revealed a significant decrease in coordination and control of voluntary movement. Additionally, stereological analysis showed a significant depletion of dopaminergic neurons in the substantia nigra following long-term sleep fragmentation. These results may indicate a relationship between exposure to prolonged sleep fragmentation and the early behavioral and neurological symptoms associated with Parkinson's disease.
Mellisa Riegel
Title: Set-shifting deficits and increased estradiol and gonadotropin levels in female adolescents with anorexia nervosa and low-weight eating disorder not otherwise specified
Supervisor: Dr. Rebecca Peebles, Department of Pediatrics
Anorexia nervosa (AN) and low-weight eating disorder not otherwise specified (EDNOS) affect up to 5-6% of adolescents. These diseases are characterized by cognitive set-shifting deficits, characterized by difficulty shifting between different mental strategies or lacking mental flexibility. The biological basis of these deficits has not been studied. One goal of this project is to investigate associations between set-shifting deficits and luteinizing hormone (LH), follicle-stimulating hormone (FSH), and estradiol levels, which may be potential cognitive biomarkers that could have particular relevance during adolescence, a time of dynamic hormonal change. This project will allow me to better understand the mechanisms leading to set-shifting deficits in adolescents with restrictive eating disorders.

Bridget Ellsworth
Title: Epigenetic Regulation of Homer1
Supervisor: Ted Abel, Biology
Expression of memory related genes is impacted by epigenetic modification of chromatin. Mutating the epigenetic regulator Sin3a enhances memory and alters expression of memory-related genes. We found that Sin3a mutant mice have increased Homer1 mRNA after learning. We hypothesize that Sin3a affects memory by regulating histone modifications at the promoter of Homer1. We found that Sin3a does not affect global histone modifications, including acetylation of histone H3 and methylation of H3K4, suggesting that Sin3a affects histone modifications locally at specific promoters. To test this, we are currently measuring histone modifications at the Homer1 promoter using chromatin immunoprecipitation. Collectively, these studies suggest that the Sin3a complex targets Homer1, providing one possible molecular mechanism to explain the memory enhancements of Sin3a mutants.

Ashley Baldwin
Title: Studying Social Reward Pathways Using Multivariate Pattern Analysis
Supervisor: Dr. Robert Schultz
Social dysfunction is shown behaviorally in both Autism Spectrum Disorders (ASD) and Social Anxiety Disorders (SAD). Children with ASD often do not find satisfaction in social contact whereas children with SAD avoid social punishment but want social contact. These behavioral aspects of these disorders are hypothesized to be a dysfunction of the mesocorticolimbic reward pathway, but a difference in activation of this pathway between the two (avoidance of social punishment or social approval) has not previously been found. This study attempts to separate the pattern of activation of the two cognitive processes. Through analyses with a generalized linear model as well as techniques in Multivariate Pattern Analysis, we have investigated the patterns of activation for these cognitive processes. These findings could be helpful in further studies of children with ASD and/or SAD to be able to determine differences in aberrant activation in the two disorders.

Lauren Ambler
Title: Pharmacological modulation of negative endophenotypes of schizophrenia in Brattleboro rats
Supervisor: Dr. Steven Siegel, Psychiatry Department
Vasopressin levels in the brain are altered in schizophrenia patients, and administration of vasopressin has been shown to reverse some of the negative symptoms of the disease. The Brattleboro rat, a Long Evans rat with a naturally occurring mutation in the arginine vasopressin gene, was used as an animal model for schizophrenia. These studies focused specifically on a social interaction test with pharmacological modulation. Amphetamine (0.5mg/kg), a dopaminergic agonist, and m8801 (0.25mg/kg), a glutamatergic antagonist, were administered to the rats prior to the social interaction test, and video and ultrasonic vocalizations were recorded. The Brattleboro rats and wild type rats had differential responses to amphetamine, suggesting that vasopressin modulates dopamine signaling, but not glutamate signaling. These results help to elucidate the role of vasopressin in the CNS as well as suggest a novel drug target for schizophrenia.

Deborah Silber
Title: Examining Neuropsychological Deficits in Children and Young Adults with Chronic Kidney Disease Using the Computerized Neuropsychological Battery
Supervisor: Ruben Gur, PhD
Research Mentor: Tyler Moore, PhD MSE
Cognitive deficiencies in IQ, memory, and attention have been observed in children and young adults with Chronic Kidney Disease (CKD), but there is a lack of specificity and consistency in existing literature. The Computerized Neuropsychological Battery (CNB), uniquely constructed to differentiate and measure extremely specific cognitive domains, is therefore being used in this study to reassess and more precisely characterize such deficits. Multiple uncorrected t-tests of mean scores for both accuracy and speed on each of the fourteen tests of the battery were used to compare performance between thirty-six patients and twenty controls. Patients demonstrated significantly poorer accuracy (compared to controls) in the domains of facial memory, verbal memory, spatial memory, working memory and spatial processing. Counter-intuitively, patients outperformed controls on speed measures for attention and spatial memory, perhaps indicating a speed-accuracy trade-off. Additionally, logistic regression was used to assess the CNB’s diagnostic power. The CNB was able to predict patient status via the expected neurobehavioral function (Episodic Memory).

Drew Goldberg
Title: An Assessment of Cognitive Performance in Adolescents with Chronic Kidney Disease
Supervisor: Dr. Azuni Collier, Dr. Ruben Gur, Neuropsychiatry
Cognitive deficits have been noted in both adolescents and adults with Chronic Kidney Disease (CKD). We used the Penn Computerized Neurocognitive Battery (CNB) to analyze performance in four main domains of cognitive function (episodic memory, executive control, social cognition, and complex cognition) and to assess cognitive deficits in an adolescent population with CKD (n=39). Results demonstrated cognitive deficits in at least one task in each domain, with the most amount of difference in task performance in episodic memory. We then analyzed the level of impairment within the episodic memory by assessing performance on face and word memory tasks to identify differences in familiarity and recollection (FvR) recognition memory. The FvR paradigm differentiates recognition memory into a dual process model. Participants were more accurate at recollection based memory compared to familiarity based recognition memory in both tasks. Furthermore, as age increased, participants became more accurate at face memory. This study provided novel evidence about the cognitive impairments in general and in episodic memory in adolescents with CKD.